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PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY
JOHN NACHBAR, M.A., M.D.
UNDER THE DIRECTION OF
THE EDITORIAL COMMITTEE

VOLUME THE SIXTH
SESSION 1912-13

PART I

GENERAL REPORTS
SECTION OF ANÆSTHETICS BALNEOLOGICAL & CLIMATOLOGICAL SECTION
SECTION FOR THE STUDY OF DISEASE IN CHILDREN
CLINICAL SECTION DERMATOLOGICAL SECTION
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I

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VOLUME THE SIXTH

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SESSION 1912-13

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GENERAL REPORTS

LONDON
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1913

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The Royal Society of Medicine.

March 10, 1913.

Sir FRANCIS H. CHAMPNEYS, Bt., President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.

General Survey.

By W. HALE WHITE, M.D.

I AM much honoured by being asked to open this debate, but the subject is so difficult and so wide that it would be beyond me to give a proper survey of it even if I had more than half an hour; therefore I ask you to forgive the inadequacy of what I am about to say. The term "alimentary toxæmia" at once shows our ignorance, for it is unscientific to group cases of poisoning according to the point of entrance of the poison; they should be grouped according to the variety of the poison: it is a minor matter whether our murderer comes in at the window or the door. Further, the term is too wide. An illness due to any poison absorbed anywhere from the mouth to the anus is strictly an alimentary toxæmia, but the phrase would hardly cover a case of opium poisoning. The user of it probably wishes to suggest that an illness is due to poisons made by micro-organisms resident in the alimentary canal, although with our present knowledge he can never name the poisons, nor is he certain of the offending micro-organism: still, we are equally ignorant of the poison in uræmia and diabetic coma. But it is not clear that it may not be used when micro-organisms from the alimentary tract—e.g., *Bacillus coli*—pass into the blood and so cause illness, and it is somewhat confusing that if infected food causes poisoning we talk of food poisoning, not food toxæmia; on the other hand, when the food is not infected by pathogenic micro-organisms, but makes the person who eats it ill, he is by some said to be suffering from alimentary toxæmia—e.g., as in a person who has urticaria after eating strawberries.

The simplest alimentary toxæmia is that due to pyorrhœa alveolaris. This local trouble in the mouth leads to imperfect mastication, hence large pieces of food are swallowed which cannot be digested; and if they contain micro-organisms which, if the mass were properly masticated, would be set free in the stomach and destroyed there, they may indirectly lead to the production of poisons in the intestines. Further, the micro-organisms discharged in the pus from the gums may be swallowed, and it is possible that the gastric secretions, being abnormal or deficient, may not kill them, or that if the stomach is well filled with solid food and the micro-organisms are then washed down with a draught of water, they, suspended in the water, will be rapidly passed through the stomach on the top of the solid food, and hence reach the intestine and cause intestinal toxæmia. It may be that in some diseases micro-organisms found in the intestine have been excreted from the blood, and that such organisms may, when in the intestine, manufacture poisons. Micro-organisms taken into the blood from the diseased gums may perhaps cause intestinal toxæmia in this way. Lastly, the micro-organisms in the gums may pass into the blood and cause septicæmia varying from mild to severe. I have met with several fatal cases, and very many persons owe general ill-health or chronic indigestion to pyorrhœa. It is surprising how often this is overlooked; when seeing a patient for the first time, it is as important to examine the teeth as to feel the pulse. Although most common among poor, ill-educated people, pyorrhœa is often to be found in those who presumably clean their teeth properly, and frequently sufferers from it are unaware that they have anything amiss with their teeth. Its presence does not prevent the patient from having other illnesses; sometimes these are wrongly attributed to pyorrhœa. It is dangerous to remove too many affected teeth at once, for a rapid absorption of micro-organisms may take place by the damaged vessels. The value of vaccines will be discussed later by others. My own practice is to give them, for I have never known them do harm in this condition, and I think I have seen them do good.

When we consider bacterial poisons formed in the gastro-intestinal tract the difficulties are so great that we may almost despair of understanding the subject. External temperature is said to play a part, for although in temperate and tropical climates the lower intestines of animals which have been weaned swarm with micro-organisms—a human being excretes daily 128 billion, 99 per cent. of which are dead, in the fæces—yet the intestinal contents of Arctic animals are by some observers stated to be sterile or almost so, suggesting that it would be a good thing to establish at Spitzbergen a health resort for those thought to be suffering from alimentary toxæmia. Ill-understood alterations in the gastro-intestinal contents must influence their bacteriology, thus *Bacillus coli* is usually confined to the large intestine, but I know a woman suffering from severe dyspepsia, and in whom lavage always shows the stomach contents to be swarming with *Bacillus coli.* Then, the presence of one micro-organism affects others, for example, one of the chief functions of the *Bacillus coli* is to smother others either by making bodies poisonous to them or by withdrawing their nourishment; it thus kills typhoid bacilli, a fact of interest when we remember that *Bacillus coli* may often be found in the urine of those afflicted with typhoid fever. On the other hand, the *Bacillus coli* may itself be killed by invaders—e.g., Morgan's bacillus, *Bacillus suipestifer*, *Bacillus enteritidis.* Then, the same micro-organism may act differently in different hosts—e.g., one person's strain of *Bacillus coli communis* often differs from another's. Again, alterations at one part of the intestine will affect processes going on lower down. For instance, colon bacilli have no effect on proteins, but they decompose (putrefy) peptones. If, therefore, there is complete absorption in the small intestine, no peptones will reach the large, and so there will be no putrefaction, nor will there be if only unaltered proteins reach the large intestine, but any peptones will putrefy. We hope to hear from the bacteriologists which are the micro-organisms causing alimentary toxæmia. Herter taught us that there are probably three groups of cases: (*a*) The indolic, in which the probable fault was that the colon bacillus invaded the lower part of the small intestine, and the patient was unable to digest carbohydrates, and usually passed abundance of indican; (*b*) the saccharobutyric, in which the organism mostly concerned is the *Bacillus aerogenes capsulatus*—the abnormal changes here occur in the large intestines; (*c*) a group combined of (*a*) and (*b*). We must, when thinking of alimentary toxæmia, bear in mind that the culture medium itself is as important as the bacteria, and therefore alterations of diet may influence the production of intestinal poisons as completely as bacteria themselves. A good instance of this is the improvement that follows the withdrawal of carbohydrates in cases of carbohydrate dyspepsia.

Those who ascribe most to alimentary toxæmia are unable to tell us what the poisons are. Much work has been done on the excretion of indican and ethereal sulphates in the urine, and there is no doubt that an excess of indican is often associated with serious intestinal disturbance, but many persons pass large amounts of indican for many years and remain in good health: on the other hand, indican is not passed in many instances in which the patient might be thought to have alimentary toxæmia. It is generally allowed that the poison is not indol, indican, or ethereal sulphates. Indeed, just as we do not know what the poison is in uræmia or diabetic coma, so we do not know what it is in the illnesses ascribed to intestinal toxæmia, nor do we know for certain which are, in any case, the offending micro-organisms, nor do we understand the circumstances favouring their appearance and development, and, unfortunately, the circumstances cannot be exactly reproduced in the laboratory. Further, it would not necessarily follow that if, say by subcutaneous injection, certain symptoms were produced, the same would follow when the poison was absorbed from the intestines; mercury, when inhaled, causes tremors, but not when taken by the mouth. Surely, all this being so, statements should be made cautiously, yet one reads that indol-forming organisms must be harmful because they are prominent—and many equally foolish arguments. Nevertheless, some very suggestive work has recently been done by Mellanby, Twort, and others, who have isolated creatin-destroying micro-organisms from the alimentary canal, and their experiments show that in the intestines of animals another bacillus forms β-imidazolethylamine, which is a powerful poison, from histidine, but the process cannot take place in an acid medium. This poison is probably destroyed in the liver, and it is suggested that it may be a cause of cyclic vomiting. It would be very interesting to see if it is present in a piece of gut excised at an operation on the human subject. Bertrand and Berthelot have called the micro-organism *Bacillus aminophilus intestinalis*.

But even if we knew all about the formation of the toxin, and had identified it in the intestine, our difficulties would not be at an end, for before it reaches the general circulation it might be altered in its passage through the intestinal wall or in the liver. Indeed, it is possible that sometimes alimentary toxæmia may be a hepatic disease in which substances absorbed from the intestine in all human beings are so altered in the liver of some that they become poisonous, or abnormal bodies arriving at the liver may so alter it that it produces poisons; anyhow, there is strong evidence that an important function of the liver is to protect us from poisons absorbed from the intestines, and that its efficient performance of this function depends upon carbohydrates. The difficulty is well illustrated by cirrhosis of the liver, which was formerly always ascribed to alcohol, but now we know that it occurs in some who do not take alcohol; it does not occur in some who do, nor in animals poisoned by alcohol, and it is a part of some diseases in which alcohol plays no part. Clearly, some other substances than alcohol can set up the cirrhotic process, and probably, under circumstances of which we know nothing, alcohol can accelerate the formation of these substances, which may well come from the intestine, and, if so, cirrhosis of the liver may be a form of alimentary toxæmia.

I am afraid our knowledge is not sufficient for us to argue that because certain processes go on in the intestine there must be a clinical condition to which the term "intestinal toxæmia" may be applied, but it does not follow that there are not assemblages of symptoms of which an alimentary toxæmia is such a likely cause that we may with reason assign them to this cause until some other cause is shown to exist. The best example of such an assemblage is that known as carbohydrate dyspepsia. An infant afflicted with this has pyrexia; it loses flesh but its tissues gain in water, its abdomen is distended by the accumulation of gas in the bowel, it is pale, fretful, and sleeps badly, it is often sick and has diarrhœa, its stools contain green colouring matter and an excess of fatty acids, they irritate the skin round the anus, leading to dermatitis. These symptoms may be produced in a previously healthy child if an excess of carbohydrate is given, and will pass away if that excess is withheld. I would suggest that these are the cases of alimentary toxæmia that should be first studied, for in them we see the problem at its simplest, the illness being induced by a single simple article of food. As this is carbohydrate, and the intestines are distended with gas, there is every probability that excessive fermentation accompanies the formation of some poison which is capable of causing pyrexia, indeed, much older children often suffer from attacks of pyrexia, which may be prevented by abstention from carbohydrates. Enterogenous cyanosis is a form of intestinal toxæmia from which we might learn much if it were commoner, for we know that in the methæmoglobinæmia variety there is a nitrite in the blood probably derived from some abnormal intestinal process, and that in the sulphæmoglobinæmia variety hydrogen sulphide is concerned with the production of the disorder; and as the cyanosis has disappeared when the accompanying constipation has been overcome, there is every probability that the complaint is really an example of alimentary toxæmia. As nitrites and hydrogen sulphide are simple chemical bodies it is particularly disappointing that we can discover nothing about the abnormal intestinal processes at the bottom of enterogenous cyanosis, and this should make us particularly careful of dogmatizing when we do not even know what the poisons are. Certainly, sufferers from enterogenous cyanosis have not the symptoms which it is the fashion now to assign to alimentary toxæmia.

Lately it has been urged with much insistence that many suffer from ill-health due to absorption of poisons from the bowel which are formed there because the bowel is imperfectly emptied, or in the modern terminology, these patients are said to have intestinal toxæmia due to intestinal stasis. That such is the case is very likely. Most people feel miserable if their bowels are not open for some time. Advertisements of medicines designed to keep the bowels open assail us all over the world; many health resorts flourish exceedingly because their natural waters are aperient; colossal fortunes have been made out of the sale of purgatives; the Pharmacopœia contains more examples of the aperient than of any other class of drugs; and the improvement in health that follows keeping the bowels properly open is often so striking that, from the clinical point of view, there is strong evidence that we may suppose such an alimentary toxæmia secondary to intestinal stasis, although we are unable to indicate the poison, nor say how it is formed. But it must never be forgotten that individuals vary exceedingly about their bowels; some are perfectly well if the bowels are open only once a week; presumably, if intestinal stasis is an important factor in causing alimentary toxæmia these persons do not readily form poisons, or, if they do, they are immune to them. It has, however, been stated recently that the results of chronic alimentary toxæmia are much more numerous than was previously thought, and that the cause of the failure to empty the bowel is frequently mechanical and has been often overlooked.

To take the last point first. It is not necessary for the production of alimentary toxæmia that there should be any mechanical obstruction to the passage of the contents of the bowel. If the poisons are bacterial, the quantity of them produced will depend upon the number of micro-organisms and the suitability of the medium for their growth, which, for example, might be so favourable that toxæmia would result without any stasis ; therefore, toxæmia is not proof of stasis. Those who attach great importance to mechanical stasis differ much among themselves as to its cause. Some attribute it to excessively large valves of Houston, some to pericolitis, some to dropping of the colon, some to adhesions around the ascending colon, some to spur formations at the hepatic and splenic flexures, some to abnormal mobility of the cæcum—although others say that the mobility of the cæcum thought to be abnormal is normal—and some to the development of various bands and abnormal mesenteries that drag the bowel and kink it, but the reasons given for thinking that these bands are evolutionary are by no means convincing—most observers regard them as inflammatory. We have no sufficiently large number of cases proving that any of these conditions are more common in those supposed to be suffering from intestinal toxæmia than in those who are not, and it is strange that when the bowels are, by chronic peritonitis, matted into one mass, so that there must be many abnormal kinks and great interference to peristalsis must exist, yet the symptoms said to be indicative of alimentary toxæmia are not constantly present and even intestinal obstruction is rare. Although it may be that some of these mechanical causes lead to alimentary toxæmia the matter cannot be regarded as proved, and it must be remembered that the co-existence of two phenomena does not prove that they stand in the relation of cause and effect ; they may have a common cause, indeed, it is possible that the abnormal bands and the toxæmia are induced by the same cause.

Much investigation with X-rays has been performed upon those thought to be suffering from alimentary toxæmia, and there is no doubt that the X-rays have added and will add enormously to our knowledge of intestinal movements, both normal and abnormal, but we must bear in mind that although X-rays may show a kink they cannot show the cause of the kink ; that if large amounts of such a heavy substance as bismuth are used, the bowels can hardly be said to be acting normally ; that we ought to be provided with dozens of X-ray pictures of dozens of healthy people taking the same amount of bismuth ; that when we look upon a screen we only see a plane picture, and that all inferences from X-ray examinations as to whether the duodenum or any other part of the bowel is dilated must be received with caution, for the rays from the X-ray tube are divergent ; we are dealing with a plane picture, and usually the statement is made that the duodenum, for example, is dilated without stating its size or that of the normal duodenum.

We may now turn to the symptoms which these sufferers from alimentary toxæmia are said to show. There is no need to detail them all, for they have been recently described over and over again in the medical press. I am one of those who think it probable that they are due to alimentary toxæmia, but the poison has never been found, the symptoms have never been produced experimentally; few observers have up to the present even given an opinion on the matter, and in their extreme degree these symptoms are, in my experience, decidedly rare, yet we find it definitely stated time after time that these symptoms are due to alimentary toxæmia, but I submit that the evidence does not amount to proof, although it renders such a suggestion probable. If we are not careful we shall, before we know where we are, be invoking the aid of alimentary toxæmia in the same reprehensible, loose way as gout, uric acid diathesis, and congestion of the liver have been called in to explain various vague symptoms which we do not understand.

So many conditions which cannot be called symptoms have been ascribed to intestinal stasis and consequent alimentary toxæmia, that they cannot all be examined now, but every one of them needs more careful scrutiny than it has received. Cystic disease of the breast, cancer in many parts, and duodenal ulcer, for example, have been set down to intestinal stasis, but it has not been shown that, among a large number of sufferers from any one of these, intestinal stasis is more common than among those in whom these conditions are not present. Then, intestinal stasis is commoner in women, duodenal ulcer in men; yet the first is said to cause the second. This casts so much doubt on the correctness of this view that if there are special circumstances which can dispel the doubt they ought to be so convincingly stated that their adequacy is accepted by all. Nor have any of these conditions been induced in animals by artificially induced stasis. Then, again, we read that the "so-called Bright's disease is merely a product of intestinal stasis." There are many varieties of Bright's disease. Which is meant? That being settled, let it be shown that a considerable proportion of those suffering from that variety of Bright's disease have alimentary toxæmia. This has not yet been done. Again, I see it suggested that curing alimentary toxæmia cured a case of exophthalmic goitre. No such conclusion can be drawn from a single case, for exophthalmic goitre is a disease which tends to get well, especially if the patient rests, and this patient rested in bed during her operation for intestinal stasis. A woman suffering from exophthalmic goitre was admitted under me with typhoid fever; when she was discharged her exophthalmic goitre was cured. This does not show any relationship between typhoid fever and exophthalmic goitre, which was probably cured by the rest in bed.

If it is allowed that ill-health is due to alimentary toxæmia caused by bacterial changes, the best treatment would be to stop the ingestion of the offending bacteria, but as we do not know what they are we cannot do this; or to give intestinal antiseptics, which experience has shown to be of limited use, although I think their uselessness has been exaggerated, for occasionally cyllin seems to do good ; or to alter the medium in which the bacteria grow, which is successful when we cure children by diminishing their carbohydrate; or to give other micro-organisms which will render difficult the growth of the offending micro-organism—this is the principle of the sour milk cure, from which much was hoped, but which has been disappointing; or if there is delay in emptying the intestine, to overcome this.

The last is the commonest mode of treatment, and thousands of people keep themselves in good health by natural aperient waters or some other simple aperient, either food or drug. Abdominal exercises and massage will, if properly carried out, cure many a case of constipation which has resisted all other methods, and the same may be said of a visit to a hydropathic institution or a spa which lays itself out for the treatment of this disorder. Lately, some surgeons have been trying to limit the formation of poisons in the intestines by preventing delay, by performing an ileo-sigmoidostomy, either with or without excision of the colon. When these cases are reported we are always assured that all medical means had been adopted without benefit, but we are never told what the medical means were. It would add greatly to our knowledge if our surgical friends would give precise details of the medical treatment, the patient's statements being corroborated by the various doctors who treated her. A suspicion comes into one's mind sometimes that perhaps some surgeons do not know all the means the physician has at his command for the treatment of delayed action of the bowels. Some have opened the appendix and washed the bowel through from the appendix to the anus. If the stasis is in the small intestine, this operation will not be of much use, but the success which may follow it when performed for colitis is so brilliant, that when the stasis is in the large bowel much benefit might be expected. One thing is certain, namely, that if surgical interference is ever necessary the patient for whom it is necessary is a reproach to her doctors, if she has consulted any in the earlier period of her illness, for the condition never ought to be allowed to advance to the stage which requires surgery, and although no other operations may be feasible, yet short-circuiting and opening the appendix are only makeshifts, for neither removes the cause of the intestinal stasis.

It is too early to speak definitely as to the results of these short-circuiting operations. Occasionally the operation itself is fatal. I, like some others, have seen some women who are not in the least better for having had it done; on the other hand, many successes have been reported, but sometimes more than one operation has been required and sometimes aperients are still necessary after the operation. It would be a good thing if some of these cases were more fully reported, photographs before and a year after the operation would be useful, and the patients who had been operated on, whether successfully or not, might be shown before this Society; and it is desirable to have the after-history of every case that has ever undergone operation, for it may be that any improvement that ensues is not permanent. This is especially important, as many of the subjects of operation are neurotic women, and we all know that the immediate effects of operation on them may be extraordinary. Further, it is unfortunate that the most frequent symptom—namely, pain—is subjective.

In this brief survey I have tried to indicate the direction which future work should take. I hope I have not been too critical; if any think so, I would beg them to remember that friendly criticism can do no harm. Our interest in alimentary toxæmia has been awakened by the fascinating suggestions of Metchnikoff, and by the bold extension of them by Mr. Arbuthnot Lane, but it is an approved principle of science that the results of one worker cannot be accepted until confirmed by others. We ought, therefore, to withhold our verdict on many points until we see whether confirmation is forthcoming. In the year 600 A.D., Isidorus Hispalensis, Bishop of Seville, wrote: "For a medical man should know the ars rhetorica, that he may be able to support with sound arguments the matters which he deals with; and also the ars dialectica, so that by the exercise of reason he may investigate the causes of sickness for the purposes of cure." That is true now, as then.

The Bacteriology of the Alimentary Canal.

By F. W. ANDREWES, M.D.

THE interests of this discussion will, I think, best be served if I devote my allotted time first to a general review of the bacterial flora of the alimentary canal and then to a consideration of the part played by bacteria in the production of alimentary toxæmia. I must leave to others the more contentious details of this large subject.

THE CONDITIONS FOR BACTERIAL GROWTH OFFERED BY THE ALIMENTARY CANAL.

These conditions are for the most part ideal. Bacteria find there an equable temperature, and the moisture they require, while the supply of foodstuffs is ample and varied. Free oxygen is largely absent in the intestine, except perhaps in certain diarrhœal conditions; we therefore find that the habitual tenants of the gut are facultative anaerobes, and that even strict anaerobes can grow there freely. It is well known that anaerobic conditions favour profound chemical cleavages in the medium in which bacteria are growing.

While the reaction of the greater part of the alimentary canal is alkaline, and thus favourable for bacterial growth, the local acidity presented by the gastric contents is not merely unfavourable but actually destructive to the more sensitive bacteria ingested. The pneumococcus, common in the mouth, does not seem, as a rule, to reach the intestine, and it is believed that normal gastric acidity is a safeguard against cholera.

There is a lack of repose in the contents of the alimentary canal; its tenants are liable to daily expulsion. Too much stress must not be laid on this, for a bacterium can multiply a thousand-millionfold during the average time occupied by its transit from mouth to anus, and vast numbers must remain in the various recesses of the canal or adherent to its mucosa. Nevertheless, stasis is a fertile adjuvant to bacterial multiplication, and hence to alimentary toxæmia, whether in a dilated stomach or under the commoner form of constipation.

THE BIOLOGICAL CHARACTERS OF THE NORMAL FLORA OF THE CANAL.

If we knew as much about the protozoan inhabitants of the alimentary canal as we do about its bacteria, we should probably find much the same laws governing both, but we know little on this subject, and I must pass the fauna by.

Of the flora of the canal we can safely affirm that it is *sui generis*. Certain groups of bacteria have specifically adapted themselves to life under the conditions just outlined, and have practically abandoned other modes of existence. Such are the bacilli of the colon group, the majority of the streptococci, and certain anaerobes. So characteristic are these of the alimentary canal of man and animals that by the universal consent of bacteriologists they are taken as indices of the fæcal pollution of water, milk, earth, and other substances. They constitute the enormous majority of the normal tenants of the canal. Their biological properties have been moulded by their environment; they grow best at body temperature, they are potential anaerobes, and they show a high capacity for splitting up various foodstuffs, primarily for their own use, but incidentally to the occasional detriment of their host. Moreover, they can withstand considerable periods of desiccation, for, owing to their wholesale expulsion with the fæces, their chance of reaching a new host is thus increased.

THE NUMBERS OF BACTERIA IN THE ALIMENTARY CANAL AND THEIR VARIATION IN CHARACTER AT DIFFERENT LEVELS.

Our knowledge on this point is limited to the bacterial species which can grow on our ordinary culture media. It is likely that agar cultures give us an imperfect idea of the total flora, but we can control our observations by stained films, and in the case of the fæces it would appear that agar cultures, aerobic and anaerobic, yield a moderately true picture of the real state of affairs. In the crypts of the fauces and in dental cavities, on the other hand, there would seem to be many organisms which we cannot cultivate. In the following remarks I can only pretend to deal with the organisms which can readily be cultivated.

In the secretions of the healthy *buccal cavity* bacteria are present, according to Gordon, to the number of 10 to 100 million per cubic centimetre. The most striking feature of this buccal flora is the overwhelming preponderance of streptococci, which form at least nine-tenths of the total

In the *stomach* and *duodenum*, on the contrary, bacteria are extremely few. For in health, when the stomach empties itself properly after each meal, it is well scoured, and a large proportion of the bacteria swallowed perish during digestion.

In the *small intestine* bacterial multiplication recommences, but so long as the contents are fluid and pass rapidly along the gut, the numbers are not very high, though they increase in passing downwards. The influence of the bile, which is supposed to check intestinal putrefaction, is of a selective character, for certain of the most typical intestinal bacteria have so acclimatized themselves to it that their powers of growth are in no way impaired. Bacteriologists commonly add bile or sodium taurocholate to their culture media when studying intestinal bacteria, in order to take advantage of this selective action. We have thus learned that bacilli of the colon group can all grow well in presence of bile, and that certain of the hardier streptococci can do the same. On the contrary, the more delicate streptococci and the pneumococcus will not grow in its presence, while such organisms as *Bacillus proteus* are much restricted in growth.

In the *cæcum* and *colon* the conditions for bacterial multiplication become more favourable than anywhere in the alimentary tract. The intestinal contents are here mechanically delayed in order that the last remnants of nutriment may be absorbed, and the numbers of bacteria found in the fæces surpass even those of the buccal cavity. According to Houston, the number per gramme of normal fæces ranges between 100 and 1,000 million. Supremacy in numbers is divided between the streptococci and members of the *Bacillus coli* group, sometimes the one and sometimes the other predominating. I have usually found *Bacillus coli* the more numerous. In the colon, too, anaerobic bacilli begin, in lesser degree, to assume prominence, while *Bacillus proteus* and sometimes *Bacillus pyocyaneus* are not uncommon.

THE NAMED BACTERIAL SPECIES OF THE FLORA OF THE ALIMENTARY CANAL.

This subject is one of much difficulty. At any time one is beset with doubts as to the criteria to be employed in deciding specific differences amongst bacteria; it is possible that they scarcely exist in the sense to which we are accustomed amongst higher plants and animals. And nowhere is the difficulty so great as amongst the coliform bacilli and the streptococci, which present infinite gradations in their biological characters. I cannot attempt to deal with this subject here, but must content myself with a few general statements.

The members of the *Bacillus coli* group are roughly classified according to their fermentative reactions with certain sugars and alcohols, and in a more refined manner by their agglutination reactions. The commonest variety is that now recognized as the classical *Bacillus coli communis*, which ferments lactose and dulcite but not saccharose. Hardly less common is its saccharose-fermenting variety, whilst the various types known as *Bacillus lactis aerogenes*, *Bacillus acidi lactici*, &c., are frequently met with. Less common are *Bacillus fæcalis alkaligenes* and members of the Gaertner group.

As regards the *streptococci*, terminology is even more difficult. The attempts to assign provisional names to the more frequently occurring types, by Horder and myself amongst others, have met with a good deal of adverse criticism, because the sugar reactions, introduced by Gordon, upon which we chiefly relied, have been considered by others to be too variable for the purpose. This is not the place to discuss this very controversial subject, but it will be admitted that the common streptococci of the mouth and intestine are of a relatively harmless nature as compared with *Streptococcus pyogenes*. Personally, I believe that a legitimate distinction may be drawn between the streptococcus most abundant in the mouth and upper air passages and a form which is common in the fæces, though it must be admitted that intermediate forms occur, so that one is probably not entitled to regard them as species. These are the types which Horder and I described as *Streptococcus salivarius* and *Streptococcus fæcalis*. Even if all sugar reactions were discarded, it would still, I think, be possible to distinguish the much greater hardiness, vigour of growth, and metabolic activity of the fæcal form from the feebleness of the salivary one. I have never been able to find *Streptococcus fæcalis* in the buccal cavity, though the salivary type is common in the fæces.

Our knowledge of the *anaerobic bacilli* of the alimentary canal is very imperfect. The most abundant appears to be the organism which has been variously termed *Bacillus aerogenes capsulatus*, *Bacillus welchii*, and *Bacillus enteritidis sporogenes*, but, with this, the ordinary putrefying anaerobe, which Klein terms "*Bacillus cadaveris sporogenes*," but which has also been described under other names, appears frequently present. In the colon, at all events, free oxygen is so far absent that these organisms, and probably other less well known anaerobes, can flourish, and it is clear that their activity in breaking down proteins is of a very high order.

While members of the three foregoing groups of bacteria constitute the great mass of the intestinal flora, every plate culture shows that other organisms are also present in small numbers—staphylococci and sarcinæ, *Bacillus proteus*, in more than one form, and *Bacillus pyocyaneus*. The last-named organism is one with which I have repeatedly met in the intestine, and it is of interest as one which forms soluble metabolic products. As regards *Bacillus proteus*, it would appear that its powers of decomposing protein have been a good deal exaggerated.

THE EFFECTS UPON THE HEALTHY BODY OF THE NORMAL ALIMENTARY FLORA.

It is sometimes supposed that the intestinal flora performs certain "functions" in the economy of its host. Attempts have been made by more than one observer to rear mammals or birds under germ-free conditions from birth. In many cases the creatures thus treated have not survived for many weeks, but the experiments teach little, for it is at least as likely that premature decease was due to the wholly unnatural conditions under which it was needful to keep the animals as to the absence of bacteria from the alimentary canal. I do not know of any good evidence that it is of benefit to have our intestines swarming with bacteria, many of the products of which seem harmful to us. Bacteria are not actuated by any sentiments of altruism. They take advantage of the favourable conditions offered by the alimentary canal purely for their own good; and if they do us little harm, it is because the whole race of higher animals has been evolved under these necessary conditions, perpetually present from the beginning, so that there has been ample time for the evolution of such protective mechanisms as are needful to neutralize the habitual effects of bacterial commensalism. I refer not merely to defence against bacterial invasion, but to a neutralization of their toxic products. It is conjectured that such injurious substances, absorbed from the alimentary canal, as may escape the alchemy of the liver are neutralized by the secretions of certain of the ductless glands—for example, the thyroid. There can, however, be no doubt that the intestinal flora is not without influence upon the body, even in health, for the more actively metabolic bacteria carry out protein-splitting to a point beyond that which can be exercised by the ordinary digestive ferments, and of the soluble products some are known to be toxic.

THE INFLUENCE OF PATHOLOGICAL CONDITIONS UPON THE FLORA OF THE ALIMENTARY CANAL.

Owing to the extreme rapidity with which bacteria can multiply, rapid changes can readily occur in the flora of any part of the alimentary tract, as may be seen in infective conditions. Further, any part of the tract in which retention of the contents is brought about is apt to show an abnormal bacterial flora.

In the mouth and nasopharynx infective changes are extremely common, and an invading microbe, such as the pneumococcus, *Micrococcus catarrhalis*, or *Streptococcus pyogenes*, may speedily predominate in the local flora. In chronic conditions of oral sepsis, such as dental caries or pyorrhœa alveolaris, there may be an excessive abundance of streptococci of lesser virulence.

Gastric dilatation and incompetence afford an instance of a radical change in the normal flora. The natural acidity of the gastric contents is disturbed, and in the absence of proper emptying and scouring of the viscus, yeasts, sarcinæ, and other unnatural tenants come to multiply in the stagnant contents, and may form a conspicuous microscopic feature in the vomit.

Nor, in considering the bacteriology of the alimentary canal, must we forget the gall-bladder, which is not rarely the seat of chronic bacterial infection. Usually such infection is by some normal inhabitant of the canal, such as *Bacillus coli*, whereby no great change is brought about. But the infecting agent may be a foreign organism such as the typhoid bacillus, and when this condition is persistent, as in a typhoid carrier, a more or less permanent addition is made to the flora of the intestine.

The bacterial contents of the small intestine may be profoundly modified in disease. In the rapid peristalsis of acute diarrhœal conditions the normal inhabitants of the gut are quickly swept away and a new flora may be substituted. Thus, in Asiatic cholera, the contents of the ileum may yield almost a pure culture of the cholera vibrio.

In the colon, diarrhœal conditions may produce a similar effect, so that the liquid dejections may show a predominance of whatever microbe is at the root of the trouble, be it cholera, dysentery, or Gaertner poisoning. But from the point of view of intestinal toxæmia, constipation plays a much more important part than diarrhœa, and its effects must form an essential element in this discussion. Retention of the intestinal contents affords a fruitful field for the multiplication of the normal bacterial flora, and for the full exercise of their fermentative activities, and at the same time it gives opportunity for the absorption of whatever toxic products may be formed.

THE PART PLAYED BY BACTERIA IN THE CAUSATION OF ALIMENTARY TOXÆMIA.

Having thus shortly considered the nature and properties of the bacteria of the alimentary canal, I must address myself to the second and more difficult part of my task, that of attempting to assess the share which is borne by bacteria in the causation of poisoning from the intestines. If, in this attempt, I trespass in any way on the branches of the subject allotted to others, I must ask pardon.

In the first place, I think it necessary, if this discussion is to be of any value, that we should endeavour to reach clear conceptions as to what we mean by "alimentary toxæmia." There are few terms more loosely employed, because we have so little accurate knowledge on the subject. The very terms "toxin" and "toxæmia," though they drop so easily from our lips and soothe our minds with the feeling that at least we have a name for these things even if we know nothing about them, are in truth but confessions of ignorance. The word "toxin" is a convenient one for chemical poisons of which we do not know the composition, but it might be better for clear thinking and the advance of knowledge if we were less ready to cloak our ignorance by the too common use of the term. Our hope of salvation rests with the chemists. The word "toxæmia" is equally vague, and in danger of loose usage, but it is a legitimate word if we employ it to mean the circulation in the blood of chemical poisons, whereby they are enabled to attack the cells or tissues for which they have the requisite chemical affinity. By "alimentary toxæmia," therefore, I understand the absorption from the alimentary canal of chemical poisons, of known or unknown composition, in sufficient amount to cause clinical symptoms, the blood having served as the channel of distribution to the tissues which are poisoned.

This definition would exclude all those cases in which actual bacterial invasion of the blood occurs from the alimentary canal, though not those in which there is absorption of the toxic products of bacteria concerned in purely local infection of the canal. I would lay some stress on this, because I think it is hardly realized how commonly slight invasions of the blood-stream take place; evidence is gradually accumulating to show that the normal intestinal bacteria are constantly gaining access to the blood in trivial numbers, and as constantly destroyed by the various protective mechanisms which the body has been compelled to evolve in order to avert the danger. Only when these mechanisms are in default does serious trouble arise, but it is possible that some of the cases of local disease attributed to toxæmia are really of infective nature.

There are certain alimentary toxæmias which are clearly not of bacterial origin. We subsist on foreign proteins, hydrocarbons and carbohydrates, and of these there is abundant evidence that alien proteins may be toxic and sometimes highly so. The normal processes of digestion break down the foreign protein into somewhat simpler, though still complex, atom groups, which, after absorption, are carried to the liver, whose business it is to reconstitute the protein in a form adapted for human needs. One of the primary functions of the liver is to act as a shield against the toxicity of foreign proteins, and we have evidence that when, by the formation of an Eck's fistula, this function is eliminated, serious toxic results occur. It follows that grave hepatic disease may be the cause of a toxæmia, partly, at least, alimentary in origin, and instances of this, in acute yellow atrophy and some puerperal eclampsias, will at once occur to any mind.

The bacteria in the alimentary canal, in virtue of the high fermentative activity which many of them possess, carry out protein cleavage to a point beyond that which the body demands for its own nutritional needs, and certain of the products which thus arise are actually harmful to the host. But this has always been so, ever since bacteria took up their residence in the alimentary canal of animals, and it has been necessary for animals to evolve some means of chemical antagonism to neutralize the harmful effect. We are still in comparative ignorance of the precise chemical mechanism by which this permanent menace is met in health, but that it is so met is certain, for in health we remain untouched. It is conjectured that one of the functions of the thyroid gland is to deal with this matter. A number of observations by different workers have shown that the activity of this gland is excited by the presence of bacterial toxins, and it has been asserted by certain observers that thyroidectomized animals are abnormally susceptible to infective processes. It is possible that the antitoxic functions of the thyroid extend to chemical poisons other than bacterial toxins. Be this as it may, it is clear that the body in some way compensates the ordinary activities of bacteria in the intestinal tract, so that no harm results.

Only when such bacterial decompositions are excessive, and when fæcal retention affords unusual opportunities for the absorption of the products, are symptoms manifested.

Thanks to the labours of chemists, our knowledge of the protein decomposition products is fairly extensive, though our knowledge as to their toxicity is incomplete. Most of them appear to be of feeble toxic powers, but it must not too hastily be assumed from animal experiment that such substances as, for example, indol, are so harmless as they would appear. I am very far from agreeing with the recent hypotheses put forward by Metchnikoff as to the rôle of indol and allied substances in the causation of arterial degeneration, for the evidence put forward seems to be wholly insufficient. But we can, to some extent, measure the absorption of indol by the excretion of indican in the urine and correlate its excess with clinical phenomena. The excessive indicanuria in conditions of mental depression and melancholia—for a knowledge of which I have to thank Mr. Mackenzie Wallis—may be of great pathological significance, and may outweigh any negative evidence derived from animal experiment.

There is also a possibility—for the suggestion of which I again owe my thanks to Mr. Wallis — that excessive bacterial activity in the intestine may have its negative as well as its positive influence. Thus, still to take indol as an instance, it is known that this substance is derived from the tryptophane element in proteins. If tryptophane is an important element in tissue nutrition, it may be that excessive splitting up of this substance in the intestine by bacteria may lead to tissue starvation in an important detail. An element in a supposed alimentary toxæmia may possibly be due not so much to the positively toxic action of the derivative as to the absence of the needful substance from which it has been derived. I must leave such considerations to those who follow in this discussion.

Bacteria, however, may do more than provoke merely fermentative changes in the medium in which they are growing. They produce, in virtue of their own intrinsic metabolism, substances of unknown chemical composition which are deleterious to their hosts, and which we must be content to term "toxins" in the absence of more exact knowledge. It is established that a few—a very few—bacteria produce soluble toxins which pass out into the surrounding medium without any disintegration of the bacterial body. These are the pre-eminently "toxic" bacteria, of which the diphtheria and tetanus bacilli are the commonest examples, and they lie outside our present subject. Indeed, almost the only essentially toxic bacterium which could be dragged into the discussion is van Ermengem's *Bacillus botulinus*, present in certain ham poisonings, though *Bacillus pyocyaneus* might perhaps also be included. But the great majority of pathogenic bacteria, and practically all those which are normally saprophytic in the alimentary canal, form no soluble toxins; so far as they possess toxic properties, these reside in the bacterial protoplasm itself and are liberated only on its dissolution—so-called "endotoxins."

Bacteria are, of course, constantly undergoing dissolution in the alimentary canal, and one cannot dispute the possibility of harmful effects from such endotoxins on absorption. But we are in almost complete ignorance of the extent to which alimentary toxæmia rests on such a basis. So far as concerns the normal intestinal flora, I should be disposed to believe that poisoning by bacterial endotoxins plays a comparatively negligible part in the process. As soon as evidence is brought forward showing this to be the case I shall be ready to accept it, but so far there is none, and it is probable that the endotoxins of most of the normal inhabitants of the alimentary canal are of very feeble virulence, though those of the colon bacillus may perhaps prove of some importance. This does not, of course, apply to cases of infective enteritis, in which the absorption of bacterial toxins plays a large part in the production of the constitutional symptoms.

To sum up what I have said as to the part played by bacteria in alimentary toxæmia, it seems to me reasonable to lay down the following propositions:—

(1) In an important group of cases the poisoning is by foreign proteins as such, the defect lying in the liver, which ought to shield the body from their effects. With this bacteria have, as a rule, nothing to do.

(2) The main effect of bacterial activity in the production of alimentary toxæmia lies in their ability to carry out protein cleavages beyond the capacity of the ordinary digestive ferments, with the formation of products which, when in excess, the body is unable to neutralize.

(3) There is at present little evidence that true toxins, derived from the ordinary flora of the gut, soluble or intracellular, play much part in alimentary toxæmia.

The Toxins of the Alimentary Canal.

By Vaughan Harley, M.D.

Considerable difficulties occur in the study of the toxins of the alimentary canal, since pure toxin, like pure enzyme, has not yet been isolated, even from bacterial culture. Yet more difficult is the case when one deals with those toxins which may be formed in the alimentary tract, since an examination of the fæces would only yield the toxins which have not been absorbed. The more easily absorbed toxins, which may be deleterious to the organism, would not be recovered in the stools.

That toxins are formed in the alimentary canal there can be little doubt, and, when absorbed into the general circulation, they would be poisonous to the organism.

That erythema and urticaria are seen to occur in some digestive derangements, after taking food that can be regarded as in no way tainted, can only be explained by the formation of such toxins in the alimentary canal. Considerable erythema accompanied by urticaria may occur when lavage of the bowel with boracic acid solution had been employed [9]. I have occasionally noted urticaria with erythema after simple rectal lavage with normal saline solution, when there has been some considerable constipation. It would appear only reasonable to suppose that, in these cases, this erythema and urticaria are due to the fluid injected into the bowel liberating toxins which have, so to speak, been locked up in the retained scybala. Halliburton and McKendrick [7] endeavoured to recover the toxin in the retained stomach contents from a case of dilated stomach which suffered from tetany, but even here they were unable to isolate the toxin. So far as I am aware, these intestinal toxins have not been demonstrated, but it is no more correct to deny the existence of such toxins because they have not, up to the present, been isolated, than to say the ultra-microscopic bacteria, such as those of hydrophobia, do not exist because we are, up to the present, unable to demonstrate their presence.

The special toxins formed in the alimentary canal by different species of bacteria belong to the realms of the bacteriologist, and therefore do not come into my contribution to this discussion.

The products occurring in the alimentary canal, which may be more or less poisonous, are now to be mentioned. These substances can be derived from the protein, fat, or carbohydrates taken in the diet, or they may be derived from the secretions into, or excretions from, the alimentary tract, either in health or disease, during the process of ordinary digestion, or from putrefaction and fermentation in the alimentary canal.

That the various secretions into the alimentary canal, together with the excretions of the alimentary tract and the thrown-off epithelial cells, yield considerable residue has been shown by the observations on fasting individuals, in which fæces are found to be regularly formed. In the same way, isolated loops of the bowel have been observed rapidly to fill up with such accumulations. These secretions and the shed epithelial cells of the alimentary canal can also undergo a certain amount of putrefaction change during starvation, as demonstrated by the fact that in such fasting individuals the urine has been found to contain aromatic substances. Müller [15], in his experiments on Cetti, during a fast, found that indican decreased in the urine day by day, so that by the end of the third day it was practically absent, while phenol progressively increased in the urine, until by the eighth or ninth day it was three to seven times as much as in any ordinary man. In dogs, however, during a prolonged fast, the indican is greatly increased, while there is no increase in phenol.

In considering the possible poisons formed in the alimentary canal, it is as well to discuss first those substances which may be derived from the protein, as they are of greater importance, the substances derived from the carbohydrates or fat having little, if any, toxicity. Protein during the ordinary process of digestion, even without the action of the bacteria present in the intestine, may yield toxic products. The protein, while in the stomach, is rapidly acted on by the hydrochloric acid and pepsin, so that three-fifths of the protein is converted into peptone and about two-fifths of this is in the form of proteoses (Zunz), [3] before the chyme passes on through the duodenum. The protein itself, together with the proteoses, on passing through the pylorus, is acted on by the trypsin of the pancreatic juice, together with the erepsin from the intestinal walls, so that the protein and proteoses are still further converted into simpler products. The protein is now known not to be simply converted into proteoses and peptone, but the digestive juices act on it still further, so that the breaking down can lead to the formation of abiuret bodies in the formation of the polypeptides, and the comparatively simple mono-amino and di-amino acids (Cathcart).

The proteoses and simpler products, when injected directly into the circulation, can be rapidly eliminated by the kidneys, but are apparently toxic, since they tend to inhibit the coagulation of the blood, and have a lymphagogue effect. They also cause a fall in arterial pressure, and a febrile reaction, and large doses in animals may even cause death (Wells [22]).

The end-products of the breaking up of protein do not, however, in ordinary conditions reach the circulation as such. The epithelial tissues of the intestinal walls seem to have some power of converting these substances back into harmless protein. Hofmeister considers that these end-products are taken up by the leucocytes in the intestinal wall, and by them conveyed to the tissues. It has been demonstrated that in health neither the proteoses nor end-products are found in the blood of the general circulation, nor even in that of the portal vein.

In cases of ulceration or other lesions of the alimentary canal, it is conceivable that the proteoses and end-products of protein digestion may reach the general circulation, and, if the quantity absorbed is larger than the liver can deal with, their presence may explain many of the toxic symptoms which one periodically observes.

Albumosuria is known to occur when any considerable amount of tissue or extensive exudate is being autolysed and absorbed. The products thus formed, reaching the general circulation, are readily eliminated in the urine.

In children and in old age the epithelial cells of the alimentary canal are apparently thin, and here also may be the explanation why in such people toxic attacks are more frequently observed. After a too copious protein diet probably no abnormal products have been formed in the intestines, the wall of the bowel having become permeable to the normal end-products of protein digestion, and they reach the circulation in a greater amount than can be grappled with by the organism. The albumosuria occurring in children and old age may be due in some cases to these proteoses having reached the general circulation from the bowel itself.

The healthy digestive processes are accompanied by an enormous amount of bacterial action. Strasburger has shown that at least one-third of the total fæces in man consists of bacteria, while in diarrhœa this quantity is even greatly increased. The decomposition products of protein putrefaction, proteoses, peptone, polypeptides and amino-acids are practically the same whether formed by the action of bacteria or by the action of enzymes in ordinary digestion, so that it has been considered that the bacteria normally present in the intestine may be useful to the organism, provided they are not in excessive quantity. The action of the bacteria is certainly of service in the breaking down of some substances taken in the diet, such as cellulose.

Indol, skatol, phenol and cresol are aromatic substances which are formed by the breaking down of protein by various bacteria in the alimentary tract. These aromatic substances have been shown by Herter and Wakeman [13] to be fixed in the living cells, so that they form some loose chemical combinations, which hinders them from being able to be recovered by simple distillation, the liver cells being most active, then the kidney, muscles, blood and brain in descending order. Indol is more easily detected in the faeces and indican in the urine, and it has consequently been more studied than skatol, phenol and cresol. That the indican in the urine is derived from the indol from the alimentary canal, and not by autolysis of the protein in the general metabolism, is now an established fact. It would appear as if indol or other aromatic substances, when absorbed from the bowel, are conveyed by the portal vessels to the liver, where they are oxidized into indoxyl, skatoxyl, &c., and are combined with the sulphuric acid so as to form aromatic sulphates, in which form they are eliminated in the urine. When large quantities of these aromatic substances are absorbed, as may occur in phenol poisoning, they may use up all the available sulphuric acid, then they are combined with the glycuronic acid, and eliminated in this form in the urine.

In consequence of diminished motility in the small intestine, or of excessive quantities of protein reaching the bowel, increased intestinal putrefaction may occur, leading to an increased formation of indol in the alimentary canal, causing an increased quantity of indican in the urine. Jaffé found that ligature of the small intestine, but not of the large intestine, was followed by a large increase of indican in the urine. Ellinger and Prutz [4] did some interesting experiments, in which they reversed a loop of the small intestine, so that the lower end was joined to the upper part of the bowel, and the upper end was joined to the lower part of the bowel, causing a reversed peristalsis, which hindered, although it did not stop, the passage of the intestinal contents along the bowel, and this was followed by an increase of indican in the urine. The same operation on the large intestine was accompanied by no increase of indican in the urine.

In all diseased conditions of the bowel which lead to increased intestinal putrefaction, one usually finds an increase of indican, skatol and phenol, sometimes one and sometimes the other being dominant. The indican found in the urine is undoubtedly due to the protein putrefaction occurring in the bowel, and not, as already stated, to simple autolysis in the tissues. In cases of absorption of empyema or in purulent accumulations in the organism, it is possible for some of the indican in the urine to have this source of origin, but, under normal conditions, where no bacterial action is present in the tissues, no indican is formed in general metabolism.

The indican would appear to be derived in the bowels from the protein product, tryptophan, as Underhill [20] found that, when gelatine, which yields no tryptophan, was given instead of ordinary protein, no indican was found in the urine, and this fact has been made use of in the treatment of cases of increased indol formation. In adults, even in health, the bacteria of the bowel would appear to produce indol, as well as skatol, phenol and cresol, and these are the principal sources of the fæcal smell, while in children, especially when breast-fed, these substances are not formed, and hence the difference in smell of the stools.

As is already seen, when there is delay in the intestines, there tends to be a marked increase of aromatic substances formed in the bowel, which are eliminated as aromatic sulphates, and hence the importance of estimating the aromatic sulphates in the urine as an indication of increased intestinal putrefaction. It would appear, however, that the quantity of aromatic sulphates found in the urine does not always indicate the true condition occurring in the bowel, since in cases of diarrhœa and enteritis the stools may be found to be loaded with indol, skatol, &c., while little or no increase of indican may be found in the urine. The quantity of fluid present in the alimentary canal also influences the putrefaction, so that putrid substances are formed in larger quantities where there is any decrease in the absorption of water from the bowel contents.

In constipation one generally finds no increase of indican in the urine, owing to the dry condition of the stools hindering a marked increased intestinal putrefaction, and we know how dry stools have not the repulsive smell of semisolid or liquid ones. In some cases, however, it would appear as if there was no increase of indican in the urine, even when there is increase of indol in the scybala, as large quantities of indol may be found on distillation of these scybala in the laboratory. Such cases may be explained, as demonstrated by X-ray examination, from the constipation being due to the retention of the scybala in the sigmoid and descending colon, and the indol apparently is not readily absorbed by the lower part of the colon. In support of this view, we have the observations of Jaffé, &c., that indicanuria is especially marked the higher up the alimentary canal the obstruction occurs. In cases of atony or dilatation of the cæcum, where one finds splashing, indicating a fluid accumulation in this part, very marked increase of indican in the urine is generally observed.

One has to realize that absence of any increase of indican in the urine is certainly not conclusive that there is no increased putrefaction of the bowel, even when the diet contains a sufficiency of protein. Moraczewski [14], in a very interesting paper, which I unfortunately have been unable to read in full, as it is published in Polish, has carried out some very careful quantitative analyses of indican in the urine and of indol in the fæces. He finds on adding an increased quantity of fat to an ordinary mixed diet there is a slight decrease of indol in the stools, and a very marked increase of indican in the urine. On a mixed diet, if large quantities of sugar are added (250 grm.), there is a decrease of indican in the urine but an increase of indol in the stools. On a protein diet, when the nitrogen excretion was as much as 29·5 grm. *per diem*, there was a greater quantity of indol in the stools, with an absolute increase of indican in the urine, but, in relation to the quantity of nitrogen, the indican was really decreased.

The condition of the stomach itself has apparently an influence on the intestinal putrefaction. The digestion process of the normal stomach has been shown to alter the protein so that it is more rapidly acted on by the trypsin and erepsin in the small intestine. In cases of decreased acidity due to gastric catarrh or neurosis, even when unaccompanied by any delayed gastric motility, I have frequently found an increase of aromatic substances in the urine. It would appear as if in these cases the protein had not been sufficiently digested by the secretions in the small intestine, so that an increased quantity of protein reached the large intestine, where it had formed an increased quantity of indol, skatol, phenol, and cresol. When the gastric condition is accompanied by an enteritis, the intestinal putrefaction may commence in the small intestine, and with the increased quantity of protein in the bowel, if there is no diarrhœa present, in these cases the aromatic sulphates may be very greatly increased in the urine.

In cases of delayed motility of the stomach, especially if there is a decrease in the hydrochloric acid secretion, the protein putrefaction may commence in the stomach itself, and is therefore accompanied by an increased formation of the aromatic substances in the alimentary canal. In the most advanced forms we have fæcal vomiting, as occurs in volvulus. In cases of volvulus it has long been recognized that lavage of the stomach can ward off the fatal collapse, due no doubt to the rapid absorption of these putrid products. Under ordinary conditions, it would appear, therefore, that little or no protein putrefaction takes place in the stomach unless there is delayed motility or obstruction at the pylorus, but in nearly all disorders of the stomach there tends to be an increased intestinal putrefaction.

Anæmia accompanies all those cases in which one finds increase of indican in the urine, and of indol in the stools. The increase of indican and aromatic substances formed in the bowel might be due to the simple anæmia lowering the general condition of the individual, so that the motor power of the alimentary tract was decreased, together with the secretory power in the alimentary canal, the anæmia being in those cases the cause of the condition. In many cases it would appear that the anæmia in reality is due to the increased intestinal putrefaction itself, the excessive formation of the aromatic products formed in the alimentary canal being absorbed, and leading to hæmolysis, this destruction of red corpuscles producing the condition of anæmia.

The stinking stools which occur in biliary obstruction were formerly thought due to the want of antiseptic action of the bile in the bowel. It was shown that dogs fed on fat and meat, after ligature of the common bile-duct, passed very foul-smelling stools, lost weight, and became ravenous. The increased quantity of undigested fat accumulating in the alimentary canal led to a decreased digestion of protein, and this increase of the protein in the intestine caused increased putrefaction in the bowel, not the loss of the antiseptic action of the bile. The animals' hunger and loss in weight were due to a real starvation in spite of the large quantity of food given. On being put on to a carbohydrate and meat diet, these dogs no longer passed foul-smelling stools, since the protein was normally digested, and there was no longer a loss of weight, since the carbohydrates took the place of the fat.

In cases of pancreatic obstruction, marked increased intestinal putrefaction is found to occur, leading to very foul-smelling stools, the increased quantity of protein remaining in the bowel being due to the decreased fat absorption, and to the absence of trypsin, so that the protein is not properly digested in the small intestine, and leaves a large residue to be acted on by the bacteria in the large bowel. The estimation of the indican or even the aromatic sulphates in the urine would never give a true insight as to the amount of intestinal putrefaction occurring, unless we knew the cause of its absorption from the bowel. The quantitative estimation of indol in the stools is far too complicated to have any clinical significance, and really to estimate the amount of putrefaction occurring in the intestine one ought to have a quantitative estimation of the indican in the urine and of the indol in the fæces, and for comparison one would have to have the case on a fixed protein diet.

Skatol, which in all probability is principally responsible for the fæcal smell, accompanies indol in the stool in a varying amount, sometimes one and sometimes the other predominating, and the reasons for this are, at present, unknown. It appears that skatol is not so easily absorbed from the bowel as indol, but when absorbed it is acted on in the liver, and eliminated from the organism as an aromatic sulphate. The recognition of skatol is more difficult, however, than of indol, unless one agrees with Herter that the Ehrlich colour reaction with dimethylamidobenzaldehyde is due to the skatol in the urine, and not, as some people consider, to the presence of urobilinogen.

Phenol and cresol are also formed in small amount in the bowel by the breaking down of protein by putrefaction. They have been found greatly increased in the case of ileus, diffuse peritonitis, and simple atony of the bowel, as well as in tubercular enteritis, in all of which cases there is apparently increased putrefaction in the stagnant contents of the bowel, while no increase of phenol or cresol have been noted in simple constipation.

Indol, skatol, phenol and cresol may all be considered more or less toxic substances. Herter has shown that in healthy man frontal headache, irritability and insomnia can be produced by feeding with indol, and Lee [11] has shown that indol, skatol and methylmercaptan cause the muscles to react to stimuli like fatigued muscles. Rovighi [18] found that both indol and skatol were fatal to rabbits. The general conclusion would appear that these aromatic substances may be the cause of many of the symptoms of neurasthenia which we see accompanying cases of increased intestinal putrefaction.

Herter quotes some interesting experiments by Richards and Howland [11] which seem to show that reduced oxidization in the tissues may explain the more marked toxicity of indol in some individuals. The subcutaneous injection of potassium cyanide, which lowers the power of the animal cells to take up oxygen from the blood, caused in dogs the subcutaneous or peritoneal injection of indol or phenol to produce convulsions and twitchings of greater intensity and longer duration than occurred in control animals.

The quantity of indol absorbed from the intestines, even in a case of marked indicanuria, would probably have very little effect unless the toxic symptoms are due to the continual absorption day after day, and, if there is at the same time a diminished oxidation of the tissues, the substances of this aromatic group may be more toxic. This may provide an explanation of the clinical experience how fresh air and outdoor life improve the condition of such individuals.

The toxic effects due to indol, skatol, phenol and cresol that I have noted may be now briefly considered. The persistence of an increased quantity of indican and aromatic sulphates in the urine is certainly of significance, and is apt to occur frequently in middle-aged people who are in the habit of taking too much protein in their diet, or those in whom there is a diminished normal digestion of protein, leading to a too large quantity of protein reaching the large intestine to be acted on by the normal bacteria of the bowel. This condition is more marked if the alimentary canal is diseased, so as to allow an increase of the normal number of bacteria present. In these cases, sooner or later, the individual shows certain toxic symptoms which I think one is justified in considering to be due to these aromatic substances acting on the organism, since the toxic symptoms are found to disappear with the decrease of these aromatic substances.

In children, increased protein putrefaction has a greater significance than in adults or old age, and one not infrequently notes very marked retardation in growth, accompanied by mental irritability, together with anæmia and muscular fatigue, when indican is found increased in the urine, indicating increased protein putrefaction.

In adults, in cases of increased formation of the aromatic substances, indol, skatol, phenol and cresol, one finds anæmia always present, and in some cases this anæmia may be so pronounced that one may have a difficulty in deciding whether one has not a true case of pernicious anæmia. A peculiar muddy complexion is commonly seen in cases of increased indican in the urine, and is out of all proportion to the amount of anæmia as found by making a blood count. Lassitude and headache are frequently complained of, and in some cases the nervous system seems especially affected by these aromatic poisons, causing lumbago-like pains or sciatica or general pains, which, for want of a better name, are called "rheumatic." In some cases arthritic pains, in other muscular pains, are seen where one gets increased formation of aromatic substances in the bowel. The various symptoms recognized as neurasthenia are commonly present, and I believe a true neurasthenia can often be caused by continual absorption of indol, skatol, phenol, and cresol from the bowel.

The fact that Lee demonstrated that these substances had a toxic effect on muscles resembling fatigue is of great interest in this connexion. I frequently find marked indican in the urine in people who complain principally of muscular and mental fatigue, with little or no symptoms of what they please to call indigestion, although they may consider themselves "livery," as they wake in the morning with a headache and a bad taste in the mouth. In such persons, when one has an opportunity of examining their metabolism on a fixed diet, such as Schmidt's, one finds evidence of increased intestinal putrefaction, with an excess of indican, skatol, and urobilin, as well as, in most cases, an increase of indican and aromatic sulphates in the urine, although, as I have already shown, we may get increase of the aromatic substances in the fæces without necessarily getting increase in the urine.

Premature senile decay with early arterio-sclerosis is apt to occur in people who have an increase of indol and urobilin in their stools. In such thin subjects, even when the diet contains such a number of calories that an ordinary individual would be putting on weight, these will gain no weight. On metabolism analysis I have found the protein and fat to be fairly well absorbed, in some cases even as well as in normal individuals on a similar diet, so one has to consider that in these cases of increased protein putrefaction there is a waste of the food materials in the tissues themselves. I believe in these cases the aromatic substances, indol, skatol, &c., must be regarded as the toxic substances which are causing this tissue waste.

Irritability of the skin, often accompanied by erythema, or even urticaria, I frequently noted in similar cases, but it is possible that these symptoms may be the result of other toxins being formed in the alimentary canal together with aromatic substances.

Benzoic acid combined with glycocoll may be excreted in the urine as hippuric acid, and is derived from intestinal putrefaction (Magnus-

Levy). One has at present, however, no evidence as to the amount of toxicity of this substance in the organism.

The diamins, putrescin (tetramethylendiamin) and cadaverin (pentamethylendiamin) have been found in the urine and fæces in cases of cystinuria by Baumann. These diamins are the products of protein putrefaction in the alimentary canal. The diamins were at one time considered to be the cause of the cystinuria, but the experiments of Garrod, Hurtley, and others have shown that in cases of cystinuria the diamins are not constantly present, so that one can no longer hold that they are the cause of cystinuria. The diamins, putrescin and cadaverin have also been found in the stools of cholera patients. Dombrowski has found cadaverin even in the fæces of a healthy man. These diamins, putrescin and cadaverin, when formed in the alimentary canal, would be easily absorbed, but, as we now know, they are only to a small extent toxic, and, as the quantities formed in the bowel are not very considerable, it would hardly appear that a sufficient quantity can be absorbed to cause any serious toxæmia.

Homogentisic acid is one of the products of the splitting up of the protein molecule, tyrosin, or phenylalanin, in the intestines. In a normal condition this substance is, however, broken down after being absorbed in the general metabolism. In the condition of alkaptonuria, when the urine on standing becomes dark, it was found that this coloration is due to the presence of homogentisic acid in the urine, and it appears that in certain conditions of normal metabolism the individual is unable to split up the benzine ring. Garrod has shown that this chemical abnormality appears to have an hereditary origin, but, as it is not due to any abnormal formations in the intestines, the subject need not be considered here.

The sulphur of the protein can readily form in the alimentary canal sulphuretted hydrogen, when there is any putrefaction going on. Its easy detection leads to its ready recognition, and it is found that even where there is simple stagnation of food in the stomach the protein may yield sulphuretted hydrogen in sufficient quantities to be eructated. In the normal bowel, when there is no abnormal increased intestinal putrefaction, especially in elderly people, sulphuretted hydrogen can often be recognized by chemical tests in the stools, if any considerable quantity of protein has been taken in the diet. Under pathological conditions, such as catarrh of the intestines, or when large quantities of protein have been taken in the diet, large amounts may be formed in the intestines. Sulphuretted hydrogen, when absorbed into the blood, has been held responsible for various symptoms, especially disturbances of the nervous system, which vary from simple headache to absolute collapse. Herter [12] has demonstrated the toxic effect of sulphuretted hydrogen when given to dogs *per rectum*. Cyanosis has been described by Stockvis and Talma, and is considered to be due to the presence of methæmoglobin in the blood, while Van der Bergh [21] has apparently demonstrated the presence of sulphæmoglobin. It is difficult to determine how much one may consider sulphuretted hydrogen responsible for the symptoms occurring when this substance is demonstrated as present in the alimentary canal, as I have generally found it accompanied with some increase of indican in the urine, and in those cases in which I have had an opportunity of making a more complete metabolism analysis, indol, skatol, phenol, and cresol are also increased in the stools. I have found that when sulphuretted hydrogen is very markedly increased, or even more if the sulphuretted hydrogen is persistently found on analysis of the fæces, the headaches often complained of by these people seem to be more persistent, and accompanied with greater depression. The depression in some of these cases is so severe that it can be described as melancholia. In these cases there is also generally considerable anæmia, and a dirty colour of the complexion is common. I have, unfortunately, never examined the blood for methæmoglobin with a spectroscope, although I have noted in some such cases a distinct blueness of the lips.

Methylmercaptan was described by Neucki as one of the gases formed by protein putrefaction in the intestine. Herter studied the formation of methylmercaptan in various culture preparations mixed with fæces, but even in a case of increased intestinal putrefaction he was unable to demonstrate its presence in fresh stools, and considered that the observers who have found it had not been working with sufficiently fresh fæces. The gas itself apparently has little toxic properties, and has only been able to produce vomiting when very large doses are injected in the bowel of dogs. Methylmercaptan as a toxic factor in disorders of the bowel would apparently play a small rôle.

The principal substances which can be isolated and are derived from the protein by putrefaction in the alimentary canal having been considered as possibly responsible for auto-intoxication, it is now incumbent on us to discuss the decomposition products of the fats and the carbohydrates. In contradistinction to putrefaction it is as well to use the term "fermentation" for the breaking down of the fats and carbohydrates. In some cases one finds, especially in children, an increased fermentation in the bowel unaccompanied by any increased putrefaction. As age advances, increased putrefaction in the bowel generally predominates, and in all probability one never gets a pure case of increased fermentation, unaccompanied by putrefaction, in a man after middle life. The fats taken in the food are broken down from the neutral fats to various fatty acids, which partly form soaps during the process of normal digestion, without the action of any bacteria, and the process of splitting up of fats commences even in the stomach. The presence of bacteria, however, increases the breaking up of the fats, so that we may get acetic acid, lactic acid, butyric acid, caproic acid, or caprylic acid in catarrhal conditions of the intestinal tract (Borkai) [2].

The ether extract of the stools has been shown by several observers to have a hæmolytic action on the blood corpuscles. In diarrhœa it is generally found to be the case, and may be due to the hæmolysin which is normally present in the pancreatic secretion (Cohnheim) being not broken down in the bowel, owing to its rapid transit, so that it appears in the stools. Grafe and Röhmer [6] have demonstrated that the hæmolytic action on the ether extract in the stools is due to the fat acids, and principally to oleic acid.

The products of decomposition of fats would be better considered when we discuss the carbohydrate fermentation. The lecithin, however, deserves to be here mentioned. As we know, in the process of digestion, although some may be absorbed as lecithin itself, the greater part is broken down in the alimentary canal into glycerophosphoric acid, fatty acids and cholin. The cholin is further broken down in the alimentary canal into the highly poisonous substance muscarine, which yields the almost equally poisonous compound, neurin. Nesbitt [17] demonstrated in dogs, who had been copiously fed on eggs, that in the obstructed loops of the bowel cholin was present, and in all probability neurin. The idiosyncrasy of some people in the eating of eggs may therefore be explained by this formation of cholin and neurin leading to toxic symptoms.

The various carbohydrates taken in the diet by the fermentation in the alimentary canal can be broken down into various acids, such as acetic, lactic and butyric acid, as well as the volatile fatty acids. A certain amount of carbonic acid and alcohol are also formed during this fermentation. The carbonic acid formed in fermentation, beyond giving a certain amount of discomfort to the patient by the distension of the intestines, cannot be considered as toxic, and the alcohol in all cases is probably so small in amount that it can hardly have any effect on the organism. Marsh gas has also been demonstrated in the gases of the alimentary tract, but little is known of its toxic effects in the quantities which could be thus formed.

The acid products of the carbohydrate and the fat fermentation have a certain amount of significance, since the acid reaction of the fæces, when it occurs in disease, is due to these products of fermentation, the normal stools being alkaline. The stools of children, even when not pathological, may be neutral or tend to acidity owing to the presence of the various acids above mentioned. Irritation of the stomach, leading to vomiting, or of the bowels, causing diarrhœa, may be often due to the simple excessive formation of the various acids. Butyric acid seems to be specially irritant, and the cause of even distinct pain in the gastric region when formed in excessive quantities.

Apparently all these acids are easily absorbed into the general circulation, and equally rapidly oxidized under ordinary conditions. In cases of excessive formation of these acids, or where there is an excessive absorption from the alimentary tract, it may be that the quantities in the circulation are too great to be rapidly broken up, and in such cases are found in the urine, and they may, in these circumstances, have some toxic significance. Volatile fatty solids are found in small quantities in normal urine (Hammarsten) [8].

The condition of increased intestinal fermentation of fat and carbohydrates is most commonly present in children, and frequently accompanied by vomiting or diarrhœa, which undoubtedly is due to the fatty acids acting as distinct irritants. In children where this condition persists one finds anæmia, which one can consider due to the oleic acid causing increased destruction of red blood corpuscles. In children with persistent fermentation one finds marked irritability together with considerable loss of weight. The loss of weight which occurs in these children, however, may not be entirely due to the toxic effects of the fermentation products, but due to the loss of foodstuffs from the alimentary canal.

The acids, when not broken down in the blood-stream, may contribute to acidosis by fixing some of the alkali of the blood, and in children it is possible that this acidosis, from simple excessive fermentation in the bowel, may be a serious factor.

In adults, as we have already said, increased fermentation is generally accompanied by increased putrefaction, so that it is difficult to say how much of the auto-intoxication may be in these cases due to the acid products of fermentation, or due to protein putrefaction, or both these may be accompanied by the formation of true toxin not yet recognized.

The oxalates found in the stools or urine are apparently derived from the diet taken. In diabetes a parallelism has been noted between the increase of oxalic acid and indican in the urine, and it has been considered that the carbohydrates play some part in this condition of oxaluria. Baldwin [1] demonstrated that oxaluria can be produced in dogs who have been given for a considerable time large quantities of sugar, with a meat diet which would not yield any oxalic acid. The excess of sugar apparently caused a gastritis, which was accompanied by an excess of mucus and absence of hydrochloric acid in the stomach, and dogs in this condition pass considerable quantities of oxalates in their urine. The oxaluria, as we all know, is generally accompanied by a faulty digestion, and it is as well to remember that the fermentative changes of the bowel may be partly responsible for the condition.

The urobilin found in the urine is formed in the bowel, and the quantity is due to the amount of bile reaching the intestine. It has been demonstrated by Gerhardt [5] in a case of obstruction of the bile-duct that no urobilin is present, either in fæces or urine, but on the administration of bile by the mouth urobilin appeared in the stools and in the urine. The increased quantity of urobilin that is found in the urine to occur during the absorption of extensive blood extravasations, and in the infective fevers, where blood destruction is going on in the organism, appears to be due to the fact that the blood pigment is converted in the liver into the bile pigment, so that there is an increased quantity of bile pigment in the bile thrown into the bowel. The increased quantity of bile pigment present in the alimentary canal is in its turn converted by the action of the bacteria normally present into urobilin, some of which is absorbed and eliminated in the urine, as urobilin itself or its chromogen urobilinogen. The easy detection of urobilin in the stools by the rose-red colour, which occurs on the addition of a concentrated solution of perchloride of mercury (Schmidt) [19], allows us to recognize very small quantities of the substances in the fæces, while, if the bile pigment present is still in the form of bilirubin, the perchloride of mercury gives a bright green colour by its conversion into biliverdin. The rose-coloured solution, when examined by the spectroscope, is found to give the urobilin band between F and b.

The examination of the contents of the alimentary canal, either in animals or the human subject [10], shows that, under ordinary conditions, the bile pigment can be traced in the ileum as far as the ileo-cæcal valve, while the contents of the cæcum and ascending colon will be found by the perchloride of mercury method to contain most of the bile pigment converted into urobilin. In cases where there is increased putrefaction, this may extend into the small intestine, and in such cases one finds the bright rose-pink colour in the ileum, or even high up the small intestine.

In very young children, where there is no putrefaction present in the bowel, I have found the same as Müller [16] that bilirubin may be present in the alimentary canal with no urobilin. Urobilin is also found absent from the stools of newborn children. The presence of an increase of urobilin in the urine, however, in cases of intestinal putrefaction may be due partly to the bilirubin present in the alimentary canal being converted into urobilin by the increased quantity of bacteria, and thus yielding an increase of urobilin in the urine, and partly to the intestinal toxins yielding an increased formation of bile pigment, by causing an increased destruction of red blood corpuscles. So far as I am aware, we have no evidence that urobilin itself causes any toxic symptoms, although its presence in increased quantity in the circulation may lead to "urobilin icterus," as described by Gerhardt. In such cases one finds a dirty yellow colour somewhat resembling jaundice, but the urine gives no bile pigment reaction with Gmelin's test, but shows a marked increase of urobilin.

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The Consequences and Treatment of Alimentary Toxæmia from a Medical Point of View.

By Robert Saundby, M.D.

The doctrine of intestinal auto-intoxication was originated by Bouchard, and has formed the foundation for far-reaching speculations. There is little evidence that the normal products of intestinal fermentation and putrefaction are capable of producing disease, but if acetic and butyric acids, indol, skatol, and the like are harmful their effects must not be exaggerated. Sulphuretted hydrogen is only present normally in traces and much larger quantities may be injected into the rectum without hurt, although admittedly this gas is more harmful when formed in the small intestine or the stomach. Ammonia is converted into harmless urea, while volatile fatty acids and acetic and butyric acids only produce local stimulation even when present in quantity. Acetone, diacetic acid and beta-oxybutyric acid are present in the urine together with various symptoms of disturbed digestion, but this does not prove that the symptoms are due to them. The first two are found in the urine of starving animals with no digestive disturbances, and merely indicate the breaking down of tissue protein. Ptomaines may be present in excess in intestinal contents without any toxæmic results, as, for example, in cystinuria. On the other hand, there is a widespread belief, not without evidence in its favour, that many diseases depend upon the absorption of poisons from the intestine, either introduced with food or formed from it by the fermentative or putrefactive processes in the alimentary tract.

Food Poisons.

In former days food poisoning referred to the accidents that result from the ingestion of substances rendered harmful by the poisons contained in them, but modern research has added to these the effects of microbes and the toxins due to their activity. These last are exclusively produced from animal food; the danger of vegetable food depends solely upon its natural poisons or its accidental contamination by microbes in the water with which it has been washed, or in the dust lying on its surface.

Vegetable Food Poisons.

Among the best known poisons from vegetables are those contained in many kinds of mushrooms, and the poisonous effect varies not only with each kind but with season and place, some being poisonous at one time of the year and not at others, some dangerous in warm countries but harmless in colder climates; some of these poisons are destroyed by boiling, the water in which the mushrooms are cooked becoming highly poisonous.

Phallin, the poison of *Amanita phalloides*, determines destruction of the blood cells and causes choleriform symptoms with vertigo, delirium and somnolence that may pass into coma, convulsions, and death; or final fatal icterus may occur after a remission of the symptoms has taken place; while should the patient recover, convalescence is slow and the myocardium and kidneys may remain damaged.

Muscarine, the poison of *Amanita muscaria*, causes nervous symptoms resembling alcoholic intoxication, with cramps, convulsions, contracted pupils, rapid small pulse, and death in collapse, or recovery may follow a prolonged sleep.

Flour is sometimes poisonous by the accidental admixture of the seeds of tares, cow-wheat, smut, ergotized rye or vetch with the meal of wheat, maize, or rye. Adulteration from tares and cow-wheat gives rise to nausea, vomiting, colic, disturbed vision, vertigo, somnolence and lassitude, which disappear in a few hours. Cow-wheat may also produce convulsions. Smut contains a poison called githagin, which may determine intense colic with diarrhœa and hæmorrhage from the bowels. Ergotized rye causes nausea, vomiting, lassitude, with constipation or diarrhœa, and subsequently local symptoms such as cramps of certain muscles or gangrene of the fingers and toes. Vetch poisoning induces spasmodic paraplegia with disorders of sensation and tremors.

Potatoes sometimes produce poisonous symptoms from the presence of solanine causing slight vertigo, headache, vomiting, dilated pupils, and rapid action of the heart.

ANIMAL FOOD POISONS.

The putrefaction of animal food leads to the production of (1) ptomaines and (2) leucomaines. The first are formed by putrefaction, the second by the excessive destruction of protein. Certain poisons are developed spontaneously and apart from microbial infection — e.g., oxybetaine from meat and putrefying bones, choline from herring pickle, muscarine from decayed fish, methylgadinine from putrid horse-flesh, and ptomatropine from salted sturgeon. On the other hand, the *Bacillus botulinus* and other microbes of the type Aertryck and the type Gaertner cause a poison, botulin, in putrid ham. Other poisons are formed by the paratyphoid bacillus, by enterococcus and proteus, while the *Bacillus coli* may under certain circumstances enter the blood and make the meat poisonous, as, for example, in hunted animals. Meat preserved in various ways, especially in cold storage, seems to be liable to undergo poisonous decomposition. This is also true of gelatine, cream and Rouennaise ducks; these last are killed without loss of blood by puncturing the medulla, and are then beaten so as to effect the diffusion through their tissues of anything absorbed from the intestine. Certain fish, the herring and the conger, and certain roes, are at times poisonous; mussels contain a poison which has been called mytilotoxin (Virchow and Brieger) and mytilocongestine (Richet), while oysters sometimes contain similar poisons. It is not known how these poisons are produced; by some (Thesen) it is thought they are formed in sea water. Young veal is highly poisonous when the cow has suffered from septic fever or puerperal metritis. The meat of animals suffering from septic diseases, metritis, pneumonia and typhus, is undoubtedly poisonous. Hunted game is sometimes also poisonous from the intestinal microbes entering the blood. Preserved meat, potted meat, sausages and the like may be infected before being packed or during preparation, and in hot weather the microbes develop and produce poisons. Eggs may be contaminated in the oviduct, and when used in confectionery, especially when mixed with such favourable media for the cultivation of microbes as sugar, milk and gelatine, may develop poisons. Cheese is also occasionally the seat of poisonous putrefaction.

There is little doubt that food may become poisonous from the absence of certain principles normally present but which have been removed or disappear when the food has been kept too long. Of these beriberi, although only known to Western medicine in recent years, is the example in which this relation has been most clearly established. The belief that it depended upon some dietetic defect was maintained as long ago as 1878 by Wernich, supported later by Van Leent, and the practical value of the doctrine was demonstrated by Takaki, who by the reform of the diet of the Japanese navy wholly eradicated the disease from that service. But it was Braddon who was the first to show the connexion between the consumption of white rice and the prevalence of the disease, and the conclusive proof has been worked out by Fletcher, Stanton, and Fraser, who have shown that rice completely denuded of its pericarp, if used as an exclusive diet, causes beriberi, while the disease may be prevented by mixing a proportion of the "cleanings" with the diet; and conversely that the disease does not arise when imperfectly husked rice is consumed.

Although the position of pellagra as a food disease is disputed, the belief is general in the United States, where its incidence has extended widely during the last few years, that this spread is due to the fact that maize, which was formerly consumed where it was grown, is now transported long distances and stored for use. The cases that have been observed in Scotland are also thought by some to be due to an analogous change in oatmeal.

Scurvy or scorbutus has been long recognized as related to insufficient diet, and should be included among diseases due to dietetic error or defect, for the suggestion that it is caused by a poison formed in tainted meat receives small support from the experience of those who have seen most of the disease. This view impressed itself upon the mind of Captain Robert Scott, as in the second year of his former Antarctic expedition, when he had plenty of fresh seal meat, the disease disappeared; but fresh meat contains the necessary principle. There is strong evidence of the value of varied diet, of fresh or preserved vegetables, and lime-juice; the use of the last article coincided with the practical disappearance of the disease from the British Navy.

Barlow's disease is another result of dietetic defect, as, like scurvy, it depends upon the want of some principle usually present in fresh milk, but absent in patent and preserved foods; cases have been alleged to occur in children who were fed on breast milk, but the breast milk may have been defective owing to the impaired health of the mother.

Speaking generally, the results of food poisons may be classified into slight, medium and severe. (1) In slight cases there may be only some digestive trouble, nausea, vomiting, colic, headache, pain in the back, with recovery in two or three days, or there may be urticaria without digestive trouble, or simply diarrhœa. (2) In medium cases, in addition to the symptoms already described there may be a persistently dirty tongue, slow convalescence, a disposition to relapse accompanied by constipation and enfeebled digestion lasting for weeks. (3) Severe cases may present (*a*) the typhoid state with high temperature, epistaxis, diarrhœa, gurgling in the right iliac fossa with enlarged spleen, such cases often giving rise to considerable difficulty in diagnosis; (*b*) the choleriform type with violent vomiting and purging, pains, cramps, dyspnœa, thready pulse, sweating, purpuric and scarlatiniform eruptions, low temperature, collapse, and perhaps death, or a reaction with return of diarrhœa and vomiting, followed by gradual improvement, slow convalescence and prolonged weakness; (*c*) the icteric type characterized by jaundice, which may be malignant in form and results, especially if the kidneys are diseased. Many doubtful cases of disease are due to these toxic infections.

Botulism is a special form in which after two or three days of colicky pain, vomiting and diarrhœa set in with dryness of the skin and mucous membrane. There may be constipation. External and internal ophthalmoplegia may occur. There is no fever, but there is difficulty in deglutition owing to the dryness of the pharynx. These symptoms may gradually disappear and slow convalescence follow, or death may result from cachexia, broncho-pneumonia, or asphyxia from food sticking in the pharynx.

In Captain Ejnar Mikkelsen's book, "Lost in the Arctic," which describes his adventures and hardships during his expedition to Greenland, he tells how when hard pressed by hunger he and his companion were driven to eat freshly killed dogs' liver and suffered in consequence. Their symptoms were peculiar, as these consisted of an uncontrollable desire to sleep, their sleep lasting ten or twelve hours, and when they awoke they suffered from a splitting headache; some days later their skins peeled off, and Mikkelsen says his companion Iversen had "big sores all over his body." In an earlier part of the book a dog was made ill by eating dog's liver, but somnolence was not a symptom. The livers when eaten by Mikkelsen and his companion were cut in slices and "fried in water," so that the meat was imperfectly boiled. The poison was probably toxic albumose, which was all the more active as the meat was freshly killed and only partially cooked.

Sir Lauder Brunton, Dr. A. C. Farquharson, and others, have drawn attention to the symptoms produced by food poisons, which in many cases resemble the action of a paralysing narcotic, but Farquharson attributes this effect not to a direct soporific poison but to dilatation of the gastro-intestinal blood-vessels, with consequent anæmia of the brain.

Many skin eruptions are attributed to the absorption of intestinal poisons—for example, erythematous rashes, urticaria, pemphigus or pemphigoid eruptions and pruritus. The effects of fish, shell-fish and certain fruits in causing erythematous eruptions are well known. Coffee may cause pruritus and sweating of the palms of the hands and soles of the feet. A hospital patient had many recurrent attacks of herpes iris with large bullæ in the mouth and pharynx and over the elbows and knees, which invariably followed a bout of beer-drinking.

Cyclic or periodic vomiting or food fever is a condition which occurs in childhood from the second or third year onward up to puberty, and is attributed by most writers who have concerned themselves about it to disturbances of digestion, especially the digestion of carbohydrates. It does not always cease at puberty, or at the age at which puberty generally occurs. A case of this kind under my care a few years ago was that of a lad, aged 18, as undeveloped as a boy aged 12; he weighed only 4 st. 9 lb.; his attacks of vomiting occurred every few weeks and were accompanied by severe headaches; he was quite unable to earn his living. At the urgent request of his father his abdomen was opened and gastro-enterostomy was performed without any definite condition being discovered or any improvement resulting. Acetonuria has been observed in this affection, but there is little likelihood that the cause is acetone poisoning.

It is doubtful whether there is any sound foundation for Nothnagel's view that certain cases of infantile marasmus, cachexia and pernicious anæmia result from atrophy of the intestinal mucous membrane. It is possible that pernicious anæmia may result from the absorption of poisons from some part of the alimentary tract which destroy the blood, but the atrophy of the stomach or intestinal mucous membrane is probably associated or secondary (Knud Faber). The relation of pernicious anæmia to intestinal intoxication, although possible, has by no means been proved, and Schmidt says he has not met with sufficiently severe fermentation or putrefaction to support this theory. Chlorosis and leukæmia have also been attributed to intestinal intoxication, but without any adequate proof, and there are now few persons who would support Sir Andrew Clark's view that chlorosis is due to intestinal poisons and may be cured more efficiently by purgatives than by iron.

Certain affections of muscles may result from severe digestive disorder, as in the cases of polymyositis acuta described by Senator and Albu.

Oxaluria or oxalæmia may be fairly reckoned to be of alimentary origin, for oxalic acid is introduced preformed in the food or is the product of carbohydrate fermentation in the stomach which may occur when the hydrochloric acid is deficient; hence the old Edinburgh treatment by nitro-hydrochloric acid is well founded.

Arterio-sclerosis is one of the conditions attributed to auto-intoxication, but the argument seems to be that as only a small percentage of cases give a Wassermann reaction they are not all due to syphilis, and if not to syphilis they must be due to auto-intoxication (Aufrecht), which only shows how comprehensive this doctrine may become. Indeed, Combe makes it play the same universal part that others assign to uric acid, while Tissier in an equally wide generalization maintains that the decomposition of animal protein in the intestine prepares a favourable soil for the growth of all pathogenic bacteria, and is therefore the source of most diseases.

It is sufficient comment on these theories to say that, however plausible, they do not rest upon a solid foundation.

POISONING BY FÆCAL STASIS.

It is tolerably certain that there is little evidence in favour of the popular notion that mere fæcal retention favours the production in the bowel of fermentative or putrefactive changes giving rise to poisons which are absorbed into the blood, and cause various pathological symptoms.

Simple constipation does not lead to any increase of decomposition in the fæces, and infrequent evacuation of the bowels is consistent with perfectly good health and longevity. But it is a not uncommon cause of chronic intestinal catarrh, which may go on to ulceration and even perforation, and it is to these inflammatory consequences that must be attributed the varied symptoms which are associated with constipation. Fæcal retention is not inconsistent with a daily action of the bowels, as in a case reported by Mannaberg of a public official who asserted most positively that he had always made a point of getting daily evacuation, but who died of perforative peritonitis caused by a large number of enormous fæcal masses nearly as hard as stones. While, therefore, we may be reasonably sceptical about "copræmia," "fæcal fever," and other alleged results of fæcal fermentation or putrefaction, nothing is more certain than that many symptoms may result from constipation where alterations in the wall of the bowel have taken place. Nor is it inconsistent with this view that thorough emptying of the bowel should be followed by temporary relief to these symptoms, but at the same time we should expect that the simple evacuation of the pelvic colon would not effect a cure.

Without inflammatory changes the natural protective agencies existing in the alimentary canal can keep us safely from such toxins as are formed in the bowel; so long as the several organs concerned are structurally healthy, the intestinal contents are practically outside the body, and are powerless to injure us.

PROTECTIVE AGENCIES.

Against the long catalogue of supposed evil influences of intestinal microbes and toxins must be set the very real protective agencies by which, under physiological conditions, their entrance into the circulation is prevented. The general harmlessness of pathogenic microbes and their toxins in the digestive tract is supported by strong evidence. The healthy mucous membrane presents such a barrier by its relative impermeability that their diffusion takes place slowly, and time is allowed for the action of the digestive secretions which destroy the microbes and neutralize the toxins. This action is effected most efficiently in the stomach and small intestine, less so in the large intestine and rectum, but dogs and rabbits can withstand the rectal injection of cultures of diphtheria and tetanus, of which a fractional dose would be fatal if injected under the skin or into the circulation. Mere quantitative variations in the amount of the digestive secretions do not appear to interfere with this protective action, but in *achylia* infective microbes and toxins are not destroyed in the stomach, and pass rapidly into the duodenum. Even here they have to run the gauntlet of the pancreatic and intestinal digestion, which possesses almost equal destructive and neutralizing properties.

The bile has a much weaker action, its antitoxic effect being apparently due to its saponifying power, as an artificial solution of palmitate of soda possesses the same antitoxic co-efficient. Roger attributes the antitoxic action of the bile to its power of modifying pathogenic bacteria, so that they no longer secrete toxins; but he maintains that so far from neutralizing toxins already formed, it actually increases their toxicity. But should these poisons enter the portal circulation in spite of the destructive neutralizing or attenuating effects of the digestive secretions and the barrier of the mucous membrane, the liver itself possesses the power of arresting poisons or of converting them into harmless or less poisonous substances. Further, the intestinal mucus which coats the lining of the bowel delays absorption, and prolonged contact with the mucous membrane neutralizes toxins. Finally, the ordinary intestinal microbes are able to neutralize the toxins secreted by allied species, and can inhibit the putrefactive power of others, while they can also neutralize toxins, although to a much less extent than the digestive juices.

Inflammation of the mucous membrane of the bowel favours the absorption of toxins, and its permeability seems to be normally greater in infants and young animals as shown by experiment. It is claimed that diphtheria antitoxin may be administered successfully to infants by rectal injection (Chantemesse), and that other antitoxins and vaccines may be so employed. Further, it is found that various vegetable toxalbumins are not completely destroyed by the digestive juices, but diffuse through the intestinal wall into the blood, and it is said that tuberculin differs in this respect from the other microbial toxins, behaving like these toxalbumins.

TREATMENT.

The treatment of these conditions cannot be discussed in detail, but the principles may be laid down.

In the first place, it is essential that food should be wholesome, that articles of doubtful nature should be avoided, that preserved foods, especially animal foods, should not be eaten when fresh can be obtained, and that as far as possible all food should be prepared by submitting it to cooking at a temperature of not less than that of boiling water. The eating of all uncooked food is attended by a certain amount of risk, and although we all run this risk at times, it is unwise to eat such food when we know little about its origin or handling. For example, many English travellers suffer from eating fruit and salads in hotter countries, and even at home oysters are justly shunned unless we know their source.

In the second place, all food should be eaten slowly, properly masticated, and mixed with saliva. Defective teeth should be repaired or replaced, and the mouth kept in a healthy condition.

Provided these conditions are all fulfilled, the stomach, if healthy, may be trusted to perform its share, but no doubt Trousseau's traveller was right, who concluded each meal in strange places by taking a few drops of hydrochloric acid to reinforce the action of the gastric juice.

By all means let the diet be a mixed one of carbohydrates, protein and fat, and consist of such things as are suited to the individual, avoiding excess, and remembering that while every stomach can digest small quantities of most things, no stomach can digest large quantities of everything. In youth amounts of food can be assimilated with advantage that in middle age tend to obesity and gout, and in later life to disordered digestion and many evil consequences. Moreover, mode of life must be allowed to regulate the quantity of our food, and as we become less active we should eat less.

Alcohol should be regarded with doubt and suspicion, for while capable of conferring a certain amount of pleasure and at times of promoting appetite and even digestion, its general effect is to slow the digestive process, and in large quantities to check it, and to inhibit those protective agencies upon which our safety depends.

While due time should be given to meals and resting after assists digestion, daily exercise favours absorption, maintains the circulation of the blood and lymph, prevents constipation, and promotes elimination by the lungs and skin.

When alimentary toxins have been absorbed and passed into the circulation the most rational treatment would be the administration of an antidote if we possessed one. Failing that, we should try to prevent the further formation of poisons and to favour their elimination. Nature endeavours to get rid of the poison by setting up diarrhœa, vomiting and sweating, more rarely by diuresis, all of which may be imitated, guided, or controlled. The means by which we may endeavour to provoke the prevention of the further formation of poisons and the destruction and elimination of those already present may be by: (*a*) cutting off the supply of material; (*b*) reinforcing the digestive juices; (*c*) bacterial action; (*d*) drugs; (*e*) hydrotherapeutics.

(*a*) We seek to cut off the supply of further material for the formation of poisons by simplifying the diet. In acute conditions, after washing out the stomach, we may give nothing but hot water or the same with the addition of a little milk or milk diluted with lime-water, soda-water or barley-water; in subacute and chronic conditions it is best to rely on lactofarinaceous diet. As a general rule, eggs, milk and cheese are allowed, but some forbid even these and give only vegetable protein, carbohydrates and fat (Tissier). In all cases it is better that the food should be varied, well cooked, and in moderate quantity. Fasting is a perfectly sound method of diminishing toxins if combined with copious draughts of water or mineral water (Guelpa). Fruit "cures," such as the strawberry cure that enabled Linnæus to get rid of his gout, the grape cure and the like, probably act partly as fasting cures, partly by supplying a non-toxic and moderately purgative diet. The advice of Albu to take the juice of two or three fresh lemons three times a day and to abstain from milk and fatty food, is based on similar principles. The so-called Salisbury diet, which consists of three small meals of finely minced lean meat, each followed at an interval of two hours by half a pint to a pint of hot water, is a starvation diet and acts probably very much like fasting, while the hot water promotes elimination. Schroth's dry diet is also a cure by inanition, but the rational basis of the plan in all its details is not apparent, as after four or five days' abstinence from all drink and all food, except five or six small dry rolls, the patient is encouraged to take a litre or a litre and a half of light wine, which, as might be expected, often causes intoxication; but the results of the Lindewiese cure, as reported by a recent observer (F. Sandoz), are on the whole not unfavourable.

(*b*) Physiology teaches us how to stimulate the secretion of the various digestive juices, as the whole series depends for its efficient working upon the proper initial start. Careful mastication not only divides the food mechanically and mixes it with the saliva, but stimulates the salivary glands to secrete, and the saliva in turn provokes the flow of gastric juice and this the other secretions. Therefore, the habit of hasty eating and imperfect mastication from any cause may impair the whole process of digestion, while conversely due attention at this stage is calculated to ensure the complete carrying out of the entire function. As we have seen that these digestive agencies are the most powerful means we possess for destroying or neutralizing alimentary toxins, the importance of this point can hardly be exaggerated, although to chew food until it is tasteless and remove the residue from the mouth, as recommended by Mr. Fletcher, goes beyond what is reasonably necessary.

(*c*) We have seen that the ordinary intestinal bacteria afford a protective agency, which, although weaker than the digestive juices, checks the action of putrefactive and pathogenic bacteria and neutralizes the toxins formed by them. Of the ordinary bacteria which play this useful part the best known is the lactic acid bacillus, a normal inhabitant of the intestine which has been made famous by Metchnikoff, but the value of buttermilk in remedying the results of a debauch has long been known in the North of Ireland. Bulgarian sour milk or Yoghourt, when properly prepared, is a pleasant article of food which promotes the normal action of the bowel, and the use of this or of buttermilk should be encouraged.

(*d*) As the stomach is the place where the food first rests and remains longest, it possesses the most efficient antitoxic properties. The healthy stomach is sterile, and all food during its stay within its cavity is sterilized as a result of peptic digestion, and this is carried out in the presence of sufficient hydrochloric acid and pepsin. Pepsin is deficient in only the rarest instances, but the supply of hydrochloric acid may be defective or altogether absent. Under these circumstances the administration of hydrochloric or nitro-hydrochloric acid is indicated, but should be in larger doses than those commonly administered. Ten or fifteen drops of hydrochloric acid (B.P.) may be added to half a pint of sweetened water, and this quantity used as a drink during or after each meal. Mercurials, especially blue pill or calomel, act as antiseptics in the stomach and duodenum, but most of the so-called intestinal antiseptics have not justified the claims made for them, and it is doubtful whether any antiseptic drug in practicable doses can preserve its bactericidal capacity after passing a short distance down the intestine. Purgatives unquestionably eliminate toxins and are among the most powerful means we possess, but many act only on the pelvic colon and rectum, and do not clear the whole length of the large intestine. Senna possesses this valuable property, and one of the best aperients for chronic faecal stasis is an infusion of senna pods taken every night at bedtime.

(*c*) The practice of drinking large quantities of water, especially of the milder mineral waters, has much in its favour. Unmineralized water is apt to stay in the stomach and so to cause discomfort, but this does not seem to be true of hot water, which many people take on rising and again at bedtime. Small quantities of salines, especially magnesium chloride or sodium phosphate or sulphate, may be added to the water with advantage. This helps elimination by preventing the drying of the faeces in the colon and stimulating peristalsis. To ensure complete evacuation of the large bowel, irrigations or ascending douches are among the most efficient means, but there is a risk of inducing atony of the bowel when their quantity is excessive or their use is continued too long. The "entero-cleaner" of Anton Brosch is the most elaborate of ascending douches, the chief advantage claimed for it being that as it is administered under water the external pressure supports the bowel and prevents over-distension. When the above described methods have had a fair trial, and there is reason to believe in serious pathological changes in the colon permitting the continued absorption of toxins, its exclusion or removal should be considered.

The conclusions which I desire to submit are:—

(1) That under physiological conditions the natural protective agencies in the alimentary system are sufficient to shield the body from the dangers of poisons formed therein or of those introduced with the food, provided that these are not in overwhelming amount.

(2) That infrequent or incomplete evacuation of the colon is not in itself a cause of disease.

(3) That such morbid symptoms as arise result from a breakdown in the protective machinery in consequence of functional defects of pathological changes in the organs concerned.

(4) That wholesome food in reasonable quantity may consist of animal and vegetable protein, carbohydrates and fat in due proportion, and that the evidence does not justify the entire exclusion of animal protein as being directly or indirectly a cause of toxaemia.

(5) That when toxaemia is present treatment should be based on the principles of eliminating the poison already present, preventing any further introduction and reinforcing the natural protective agencies.

(6) Where there is extensive disease of the wall of the colon, the exclusion or removal of this organ is a justifiable proceeding after a reasonable trial has been given to medical methods.

The Consequences and Treatment of Alimentary Toxæmia from a Surgical Point of View.

By W. Arbuthnot Lane, M.S.

In order to consider the subject of chronic intestinal stasis in any detailed manner, it will be best to deal with it from its very beginning, and I shall commence by reminding you of several most important general laws which I formulated many years ago with regard to the skeleton and its articulations. I shall then proceed to demonstrate that precisely the same laws govern the soft parts and modify their structures, and that a knowledge of the several mechanical conditions which bring about chronic intestinal stasis is largely dependent on a recognition of these fundamental principles.

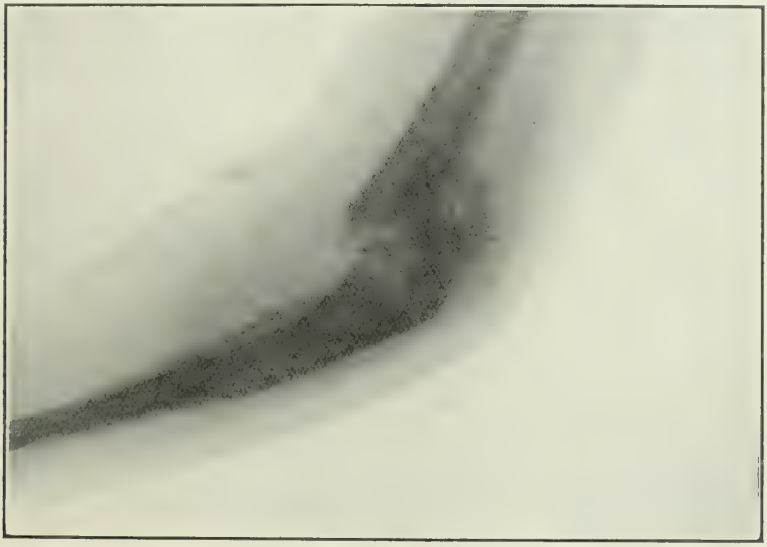

Fig. 1.

The skeleton represents the crystallization of lines of force which when exerted in a single direction are laid down as compact tissue; when in varying directions as cancellous. In young life any alteration in the length of a long bone following on a fracture in which the fragments have not been replaced in accurate apposition results in the formation of a new shaft and the absorption of the old one in a degree proportionate to the alteration in the form of the bone. This process takes place in a degree inversely proportionate to the age of the child. Later in life a lesser, but similar, change may be brought about by the artificial engorgement of the part with blood and much of the deformity and shortening which would otherwise occur can thus be obviated.

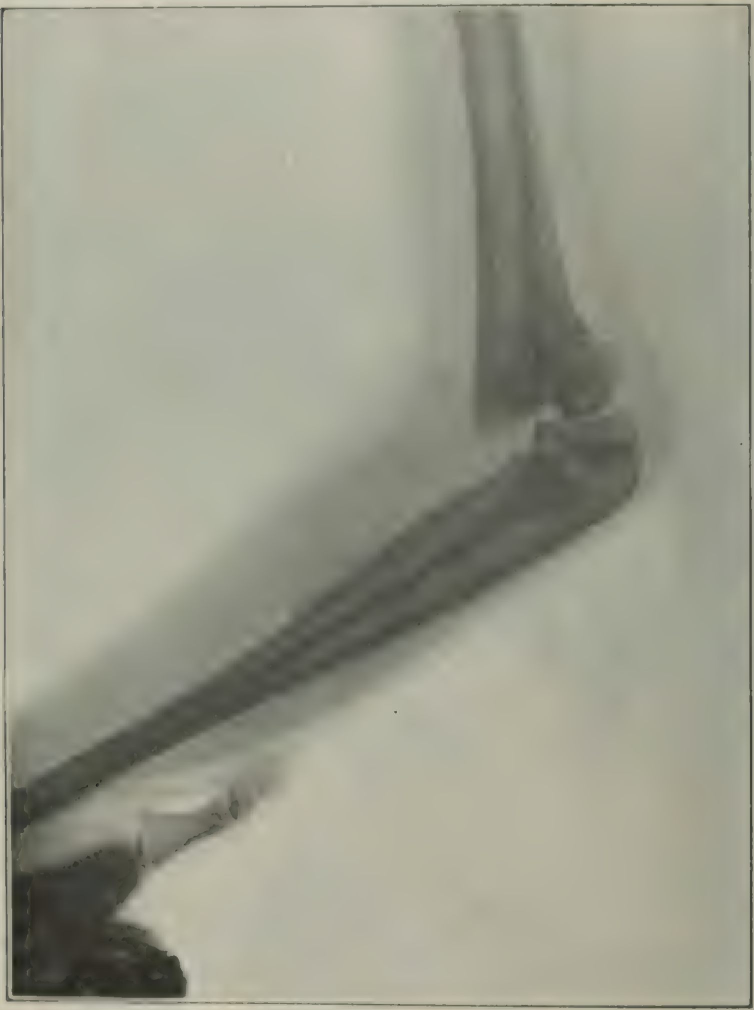

Fig. 2

It follows as a complement to this latter law that "the rates of bone formation in the several portions of a growing line vary inversely as the pressure transmitted through them," and, incidentally, it is by the exercise of these mechanical principles that Nature reduces to a minimum the harm done by the incapacity of the surgeon in the treatment of fractures in young life. The accompanying skiagrams illustrate the action of these laws:—

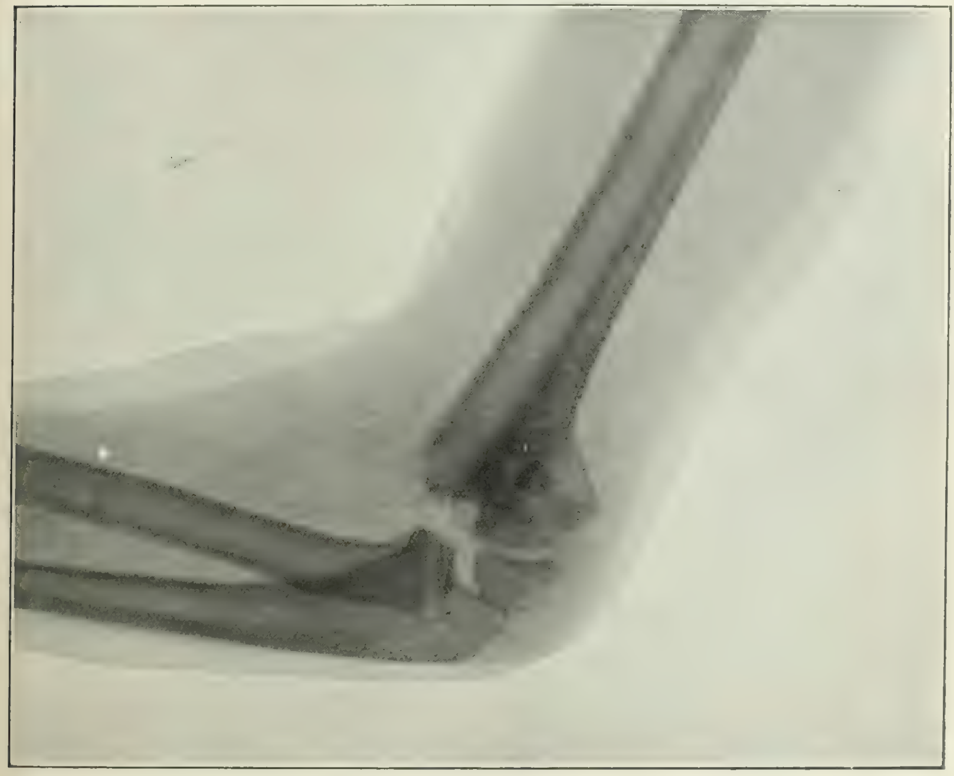

FIG. 3.

Fig. 1 represents a fracture about the lower epiphysis of the humerus with backward displacement of the fragment. Six weeks had elapsed since the injury. A dark zone extending vertically upwards from the epiphysis behind the shaft indicates the commencement of the crystallization of the lines of force.

Fig. 2 represents the condition three months after the injury. The perceptible layer of callus extends a greater distance up the back of the shaft. The shadow formed by it is much darker, equalling that produced by the original shaft.

Fig. 3 shows the part six months after the injury. The range of flexion has increased partly by the absorption of the end of the shaft and partly by that of the coronoid process. The layer of new material, which is extending much farther up the shaft, is denser, while the shaft is relatively lighter.

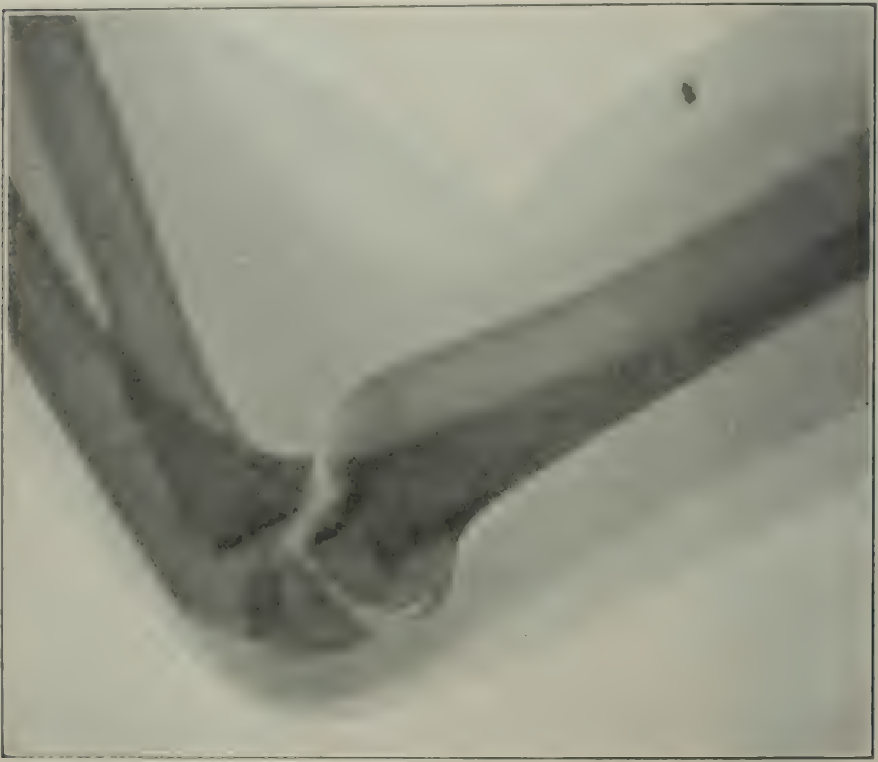

Fig. 4.

Fig. 4, taken sixteen months after the fracture, shows that flexion is still further increased by progressive absorption, while the changes already noted in the original shaft and in the new formation are much more marked.

Fig. 5, taken three years after the injury, shows that a new shaft connecting the extremities of the bones has been laid down, while the

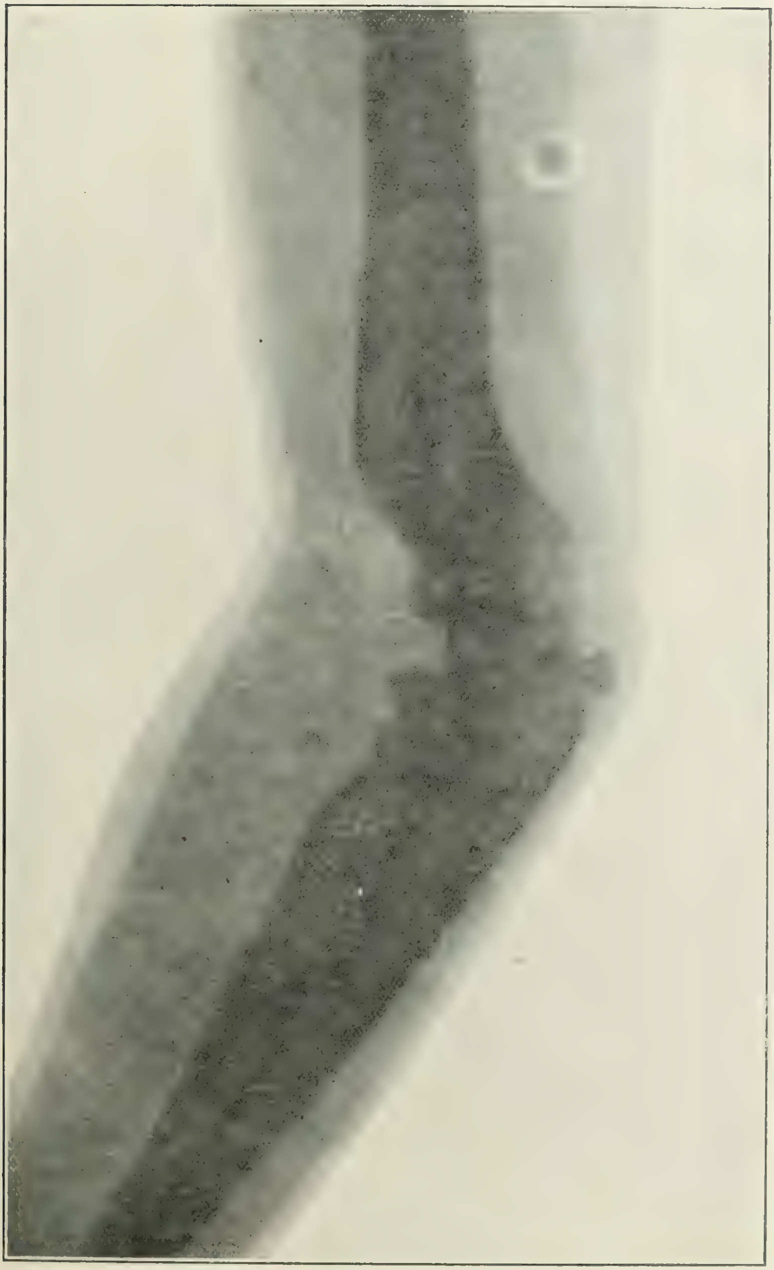

Fig. 5.

original one has almost completely disappeared. The range of flexion is still further increased.

On general principles we see that the necessity or the importance of effecting a complete restoration of the form of the fractured bone varies directly with the age of the individual.

Again, by altering the mechanical relationship of the individual to his surroundings, a skeleton is produced which differs from the normal often to an extent greater than the normal does from that of the higher ape. A study of the development of such changes in the skeleton which clearly occur during the lifetime of the individual shows them to be governed by the three following laws:—

(1) That pressure produces changes in the structure and form of the bones and in the form and function of existing joints, while it determines the formation of new joints.

(2) That strain produces change in the form of the bones, and in the form and function of existing joints, and also produces new joints.

(3) That when, apart from the exercise of pressure or strain, it is important, from the altered mechanical relationship of the individual to his surroundings, that a mechanism should be modified or an entirely new one developed, such a change takes place.

The normal form of the skeleton and of the soft parts depends on a normal combination of attitudes of activity with attitudes of rest. If attitudes of rest are habitually affected these attitudes become fixed and then exaggerated. If, on the other hand, certain attitudes of activity are habitually affected these also become fixed and later exaggerated. In other words, while an attitude of rest or of activity is being assumed on a single occasion there exist certain tendencies to change. If these attitudes are assumed habitually then these changes become progressive actualities, the result of which is to produce such very definite alterations in the form of the skeleton that the life-history of the individual can be readily determined not only from the examination of the whole skeleton but frequently from that of a single bone.[1]

The space at my disposal renders it impossible for me to consider at any length the work on this subject which I did many years ago in the dissecting room, and which has served as a perfectly secure groundwork on which has been based what little surgical work I have since been able to do. I would, however, remind you of the anatomy of certain labourers which afford excellent instances of the fixation and exaggeration of the tendencies to change which exist when a certain attitude is assumed habitually, and also of the fact that the same hold true of resting postures as illustrated by flat-foot, knock-knee, dorsal excurvation, lateral curvature, &c. I have much hesitation in inflicting these details upon you, but I am driven to do it as there exists still the same tendency on the part of many surgeons to consider any deviation from the normal as being congenital or inflammatory in origin. Just as pathologists used to regard all the evolutionary bone and joint changes of the labourer as being the result of a disease called osteo- or rheumatoid arthritis, so they now consider the acquired bands and mesenteries about the intestines as being congenital or inflammatory in origin, and employ such terms as pericolitis, mesosigmoiditis, &c., to indicate their causation. I fear it would be useless for me to attempt to alter the creeds of those who have possessed them for a long time, but I hope to induce the younger members of our profession to interest themselves in the mode of development of these evolutionary processes in the skeleton before arriving at a definite conclusion on the subject.

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[1] "The Causation, Pathology and Physiology of several of the Deformities which develop during Young Life," *Guy's Hosp. Reports* (Lond.), 1887, xliv, pp. 241-323; and "The Causation and Pathology of the so-called Disease, Rheumatoid Arthritis, and of Senile Changes," *Trans. Path. Soc. Lond.*, 1886, xxxvii, pp. 387-447.

To show the changes that arise in the skeleton which are obviously evolutionary in the lifetime of the individual by the habitual assumption of an attitude or sequence of attitudes of activity, I am employing the following illustrations which have all been fully described in early papers. Here I must necessarily describe them very briefly. I do so with the distinct object of illustrating the three laws I have already referred to.

Fig. 6 represents portions of the spine and thorax of a brewer's drayman, and shows the fixation and exaggeration of the attitude of activity which is assumed when a heavy barrel is supported on the right shoulder. The alteration in form is occasioned by a change in the shape of the ribs, by the destruction of the fibro-cartilages and the bone at the seat of greatest pressure, and by the formation of thick osseous lips at the margins of the articular surfaces to increase the security and strength of the spine. In this manner an almost rigid shelf is formed on which the barrel can be supported and through which the weight can be transmitted with a minimum expenditure of muscular energy.

Figs. 7 and 8 represent the spine of a coal-heaver. They show much destruction of fibro-cartilages, and the fixation of the margins of the vertebræ one to another by dense lips of bone which practically render the lumbar spine immobile. In fig. 9 the last lumbar vertebra is displaced forwards, and is ankylosed to the sacrum, forming a variety of spondylolisthesis. Contrast the conditions present here with those in fig. 10, where an arthrodial joint has been developed. This specimen

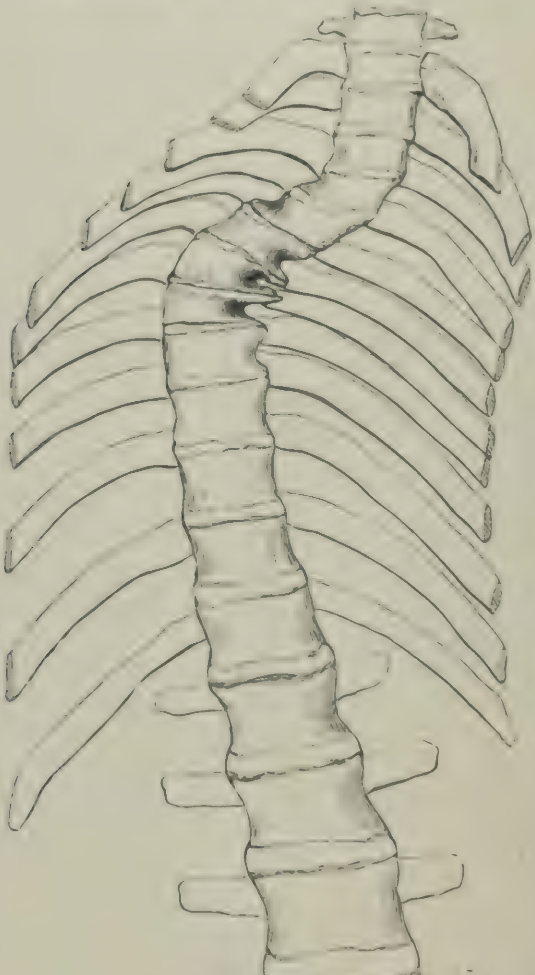

Fig. 6.

Spine and ribs of a brewer's drayman.

was obtained from the body of a labourer whose business it was to carry loads, lifting them from the ground and replacing them.

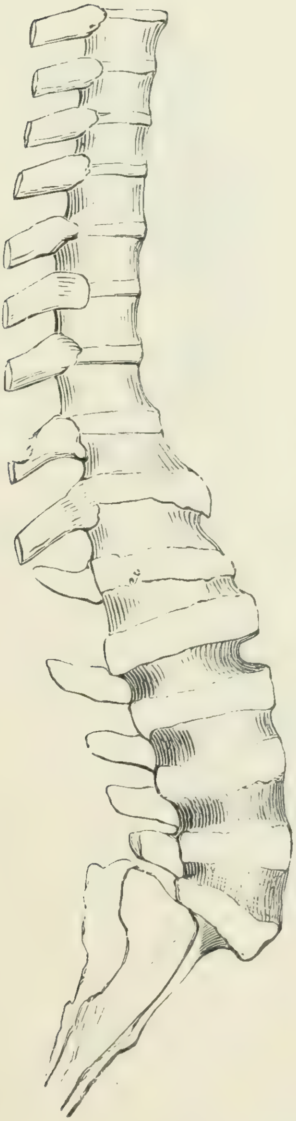

FIG. 7.

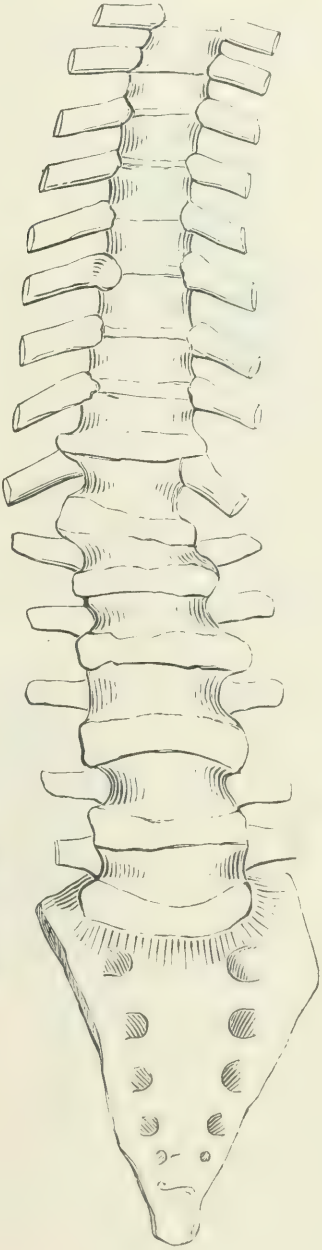

FIG. 8.

Fig. 7 shows a lateral view of a coal-heaver's spine, and fig. 8 an antero-posterior one.

In contrast with figs. 9 and 10, fig. 11 shows the condition of the lower part of the spinal column in a labourer who was habitually engaged in carrying loads in front of him. The constant over-extension of the spine resulted in a backward displacement of the fifth lumbar vertebra

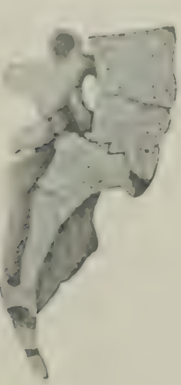

FIG. 9.

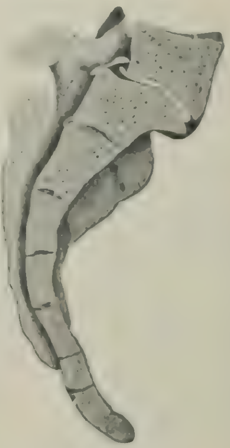

FIG. 10.

FIG. 9.—Fourth and fifth lumbar vertebræ and sacrum of coal-heaver.
FIG. 10.—Fifth lumbar vertebra and sacrum of deal porter.

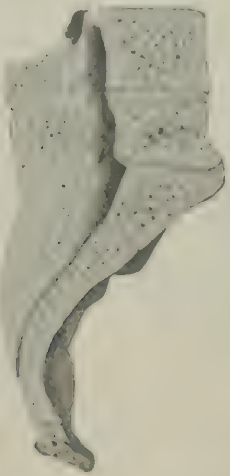

FIG. 11.

Fourth and fifth lumbar vertebræ and sacrum of labourer who carried loads in front of him.

on the sacrum, and an exaggerated development of the spines of the lumbar vertebræ and sacrum, which articulated with one another, transmitting a considerable proportion of the superjacent weight. This subject is dealt with in a paper published in the *Transactions of the Obstetrical Society*, 1887, xxiv, "The Mechanical Factors which determine the Form of the Pelvis in the two Sexes." The destruction of fibro-cartilage occurs as in all other laborious occupations.

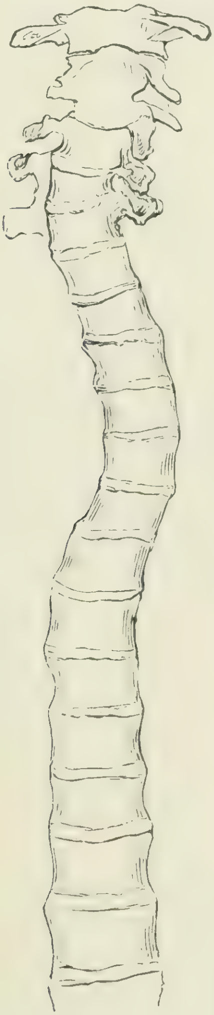

Fig. 12.

Spine of labourer who carried loads on his head.

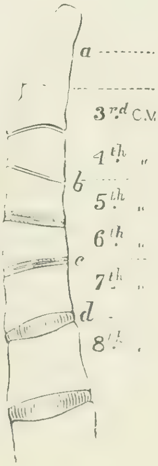

Fig. 13.

Figs. 12 and 13 show the changes which result from habitually carrying heavy loads on the head. Fig. 13 is a vertical antero-posterior section of the same specimen. The lateral curves should be noted, serving presumably to render the column less rigid. The destruction of fibro-cartilage, the formation of arthrodial joints in the upper part of the cervical spine, and the ankylosis of the second and third cervical vertebræ, are also indicated.

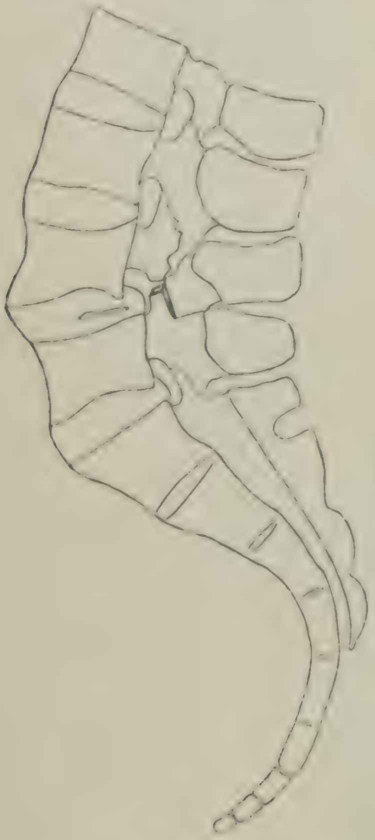

FIG. 14.

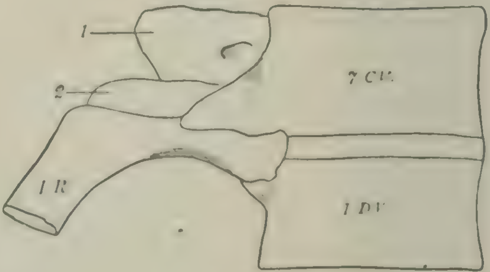

FIG. 15.

FIG. 14.—Lumbar vertebræ and sacrum of coal-trimmer.
FIG. 15.—Seventh cervical and first dorsal vertebræ of coal-trimmer.

Fig. 14 is a vertical median section through the lower part of the spinal column of a coal-trimmer.[1] It shows the formation of an arthrodial joint in the fibro-cartilage between the fourth and fifth lumbar vertebræ, and the fourth arch divided at two points. This division is seen more clearly in fig. 16, and has resulted from the forcible rotation of the spine on a vertical axis which takes place when coal is thrown with great force to a considerable distance behind this labourer when engaged at his work. Fig. 15 represents the seventh cervical and first dorsal vertebræ with the first rib, and the arrangement by means of which the head of the first rib is secured so as to obtain a firm and powerful hinge-joint.

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[1] "A Remarkable Example of the manner in which Pressure Changes in the Skeleton may reveal the Labour History of the Individual," *Journ. of Anat. and Physiol.*, 1886-87, xxi, pp. 385-406.

Figs. 17 and 18 illustrate the changes which develop in the right femur of the coal-trimmer in consequence of the special functions it performs: the area of impact of the neck of the femur locking and limiting the range of movement of the joint being shown very clearly.

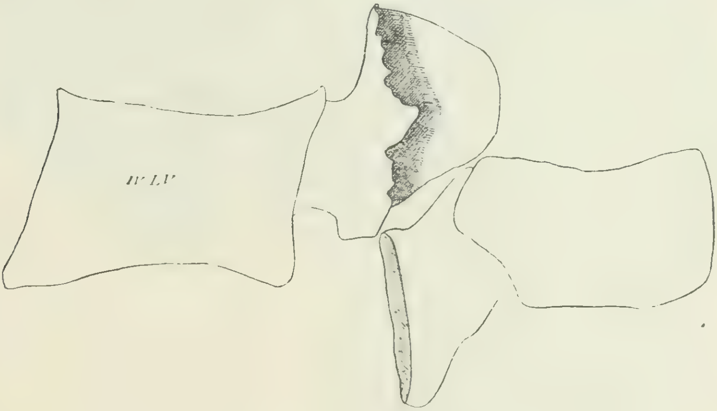

Fig. 16.
Fourth lumbar vertebra of coal-trimmer.

Fig. 19 shows the conversion of the first costal cartilage directly into bone under the influence of strain with the development of a new joint in the ossified cartilage; also the formation of a new joint between the clavicle and first costal arch, and of a similar arthrodial joint between the coracoid process and clavicle. All these evolutionary changes take place in the fully developed skeleton.

In fig. 20 the joint, probably arthrodial originally, has become amphi-arthrodial with very limited movement, while in fig. 21 the joint has been obliterated.

The second general law — namely, the influence of strain on the skeleton—is very well illustrated by figs. 22 and 23. They represent, respectively, the scapula of a shoemaker and of a deal-porter, the latter having carried his load habitually on the right shoulder. Besides the pressure changes as shown by the character of the acromio-clavicular and shoulder-joints, fig. 22 shows the result of strain exerted chiefly on the trapezius and levator anguli scapulæ, and fig. 23 the very great breadth of the acromion process, which develops in consequence of the strain on the back of the deltoid, and the eversion of the margins of the supraspinous fossa from that sustained by the supraspinatus muscle. The strain on the infraspinatus and teres major muscles, and upon the rhomboids during the process of pulling the threads by the shoemaker, has left a very definite impress upon the bone.[1]

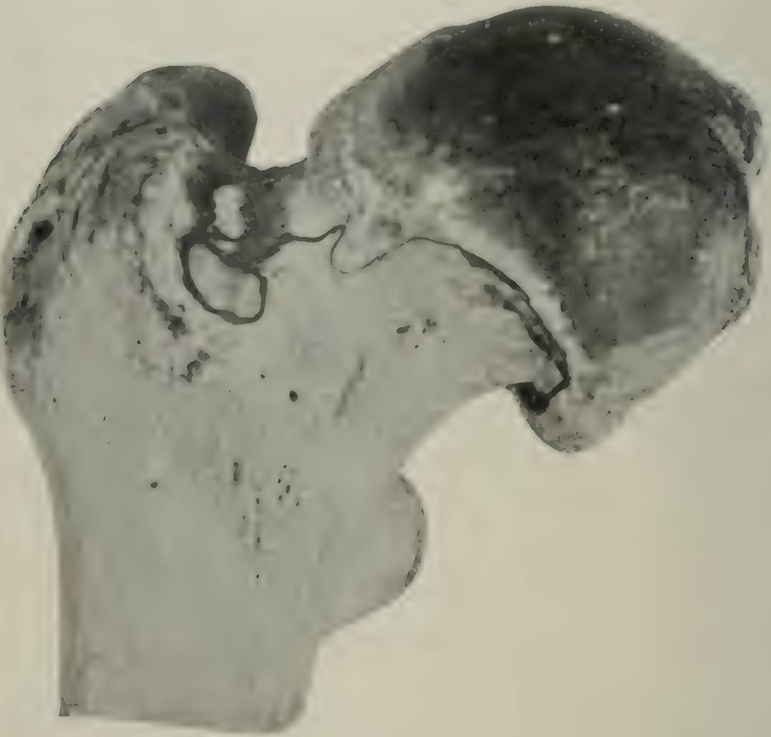

FIG. 17.

Anterior aspect of upper extremity of right femur of coal-trimmer.

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[1] "The Result produced upon the Muscles, Bones, and Ligaments by the Habitual Exercise of Excessive Strain," *Brit. Med. Journ.*, 1888, ii, p. 1205.

The third law, viz., "that a pre-existing mechanism may be modified or an entirely new one formed apart from the influence of pressure or strain," is well illustrated by a new joint which develops in the shoemaker. This structure is not present in the normal skeleton. During the jerk upon his thread, the head, which is held somewhat obliquely as regards an antero-posterior axis upon the spine, is rendered more secure by the formation of a buttress of bone, which extends upwards from the lateral mass of the atlas on the one side and articulates, by means of an arthrodial joint, with the jugular process of the occipital bone. This is shown in fig. 24, which represents the under surface of the occipital bone, C being the acquired facet.

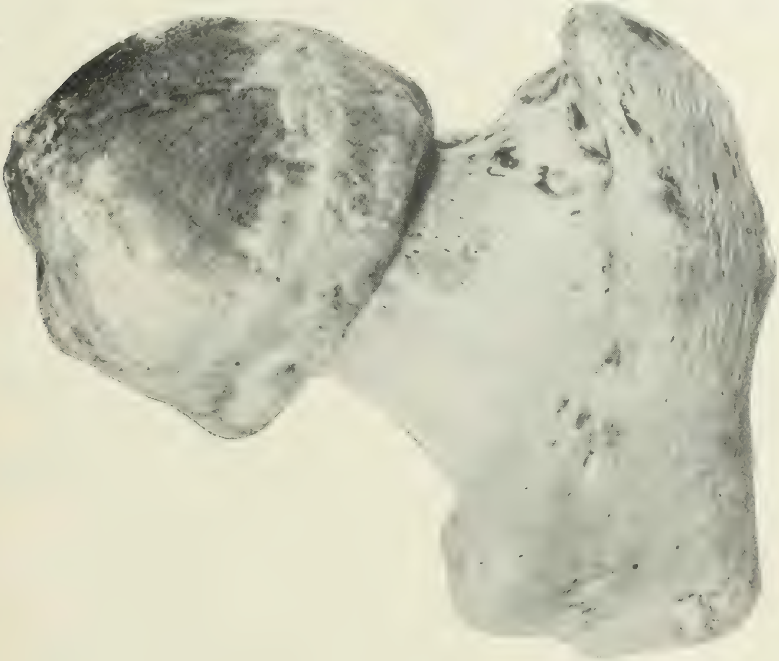

FIG. 18.

Posterior view of upper extremity of right femur of coal-trimmer.

Fig. 25 represents the anterior surface of the atlas with a prolongation upwards from its anterior arch, also the large quadrilateral column of bone which stands upon the upper surface of the left lateral mass and articulates with the jugular process of the occipital bone.

Fig. 26 represents the axis and third cervical vertebra of the shoemaker, the prolongation upwards of the odontoid process, with its articular facet, the formation of a layer of bone connecting the arches of the axis and third cervical vertebra, and the position of the ankylosed articulation of the articular process. The cause of the destruction of the intervening soft parts, and the union of the vertebræ to one another in this as in other laborious pursuits, is fully explained in the original paper in the *Journal of Anatomy and Physiology*, July, 1888, "The Anatomy and Physiology of the Shoemaker."[1] I have frequently seen these conditions exhibited as cured tubercular or other disease of the spine by surgeons, and congenital union of bone by anatomists. Besides showing very clearly the great changes which result from the habitual exercise of very considerable pressure, the elbow-joint of a coal-trimmer illustrates the same law, since both the olecranon and coronoid depressions have been filled up by dense bone-joint sufficiently to limit the range movement in the elbow to the extent requisite for the perfect performance of the occupation of a coal-trimmer. Such a change cannot result from pressure or from strain, since these cavities are not exposed to the

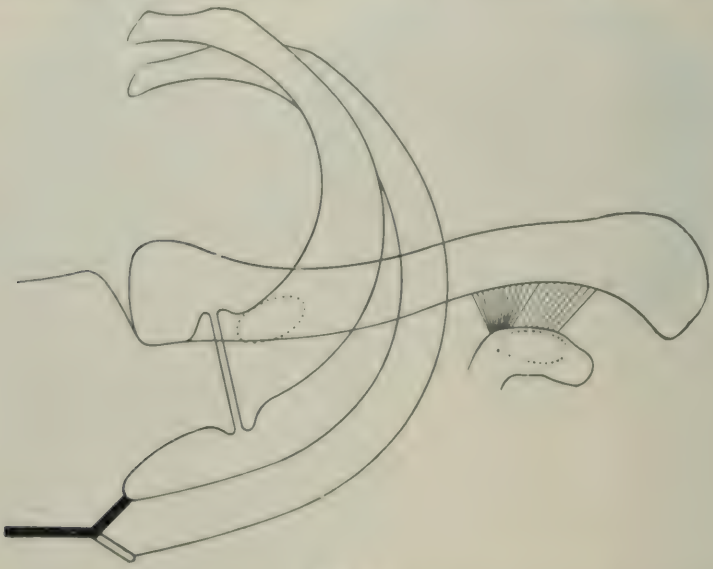

FIG. 19.

Represents the left first and second costal arches, with the manubrium, clavicle, and coracoid process, of a labourer.

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[1] *Journ. Anat. and Physiol.*, 1888, xxii, pp. 593-628.

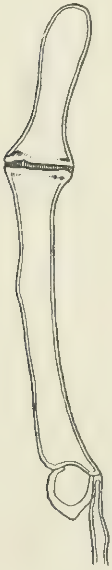

Fig. 20.

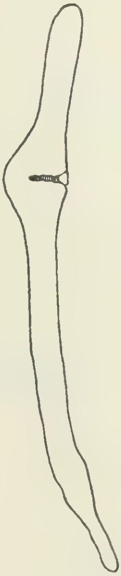

Fig. 21.

Figs. 20 and 21 show the changes that take place in the manubrio-gladiolar joint in consequence of the transmission through it of great pressure. These specimens were obtained from the bodies of labourers who had been engaged in heavy porterage work at the docks.

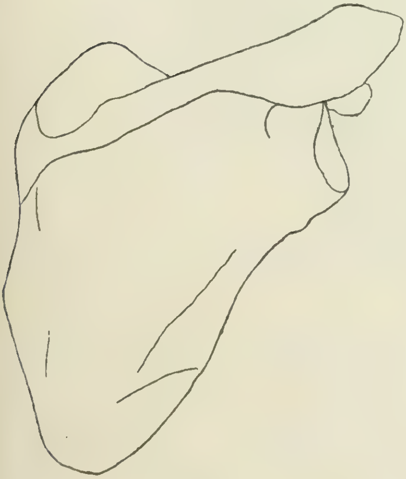

Fig. 22.

Scapula of shoemaker.

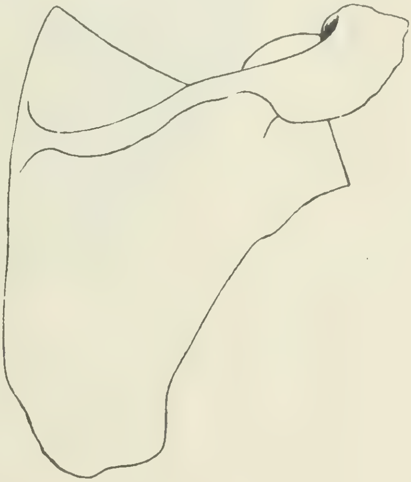

Fig. 23.

Scapula of deal-porter.

action of force in this manner, but it arises because it is advantageous and economical to the individual in his peculiar relationship to his surroundings, or, in other words, in his occupation.

In fig. 27 the joint is shown at its limit of flexion. The manner in which the humero-ulnar segment is strengthened by buttresses of bone, which increase the area of the articular surfaces and render the fit more accurate, is noteworthy. The range of flexion and extension is much limited, for reasons of economy, by the formation of bone in the coronoid and olecranon fossæ. These are seen in fig. 28, where the coronoid fossa and the depression for the head of the radius are rendered shallow by the deposit of bone on their floor.

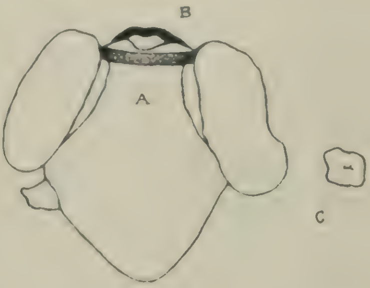

Fig. 24.

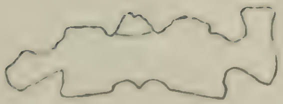

Fig. 25.

Atlas of shoemaker.

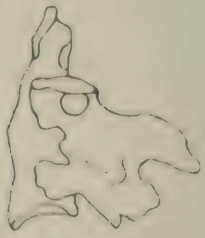

Fig. 26.

Axis and third cervical vertebra of shoemaker.

Again, an attitude of rest if assumed habitually in the child becomes fixed and exaggerated. Here the presence of the growing line assists in the production of an alteration in the form of the bones; this factor being usually absent in the labourer who does not commence to do heavy work till his skeleton is fully developed. The attitude of rest in advanced age can best be studied in the skeleton of the feeble old subject.

Many of the changes in feeble old age are illustrated in the following diagrams:—

Figs. 30 and 31 represent vertical median sections through the bodies of feeble old subjects. Note the resting position of complete flexion of the several parts of the spine. In the lower part of the spinal column the habitual flexion of the lumbo-sacral joint has resulted in an abrupt bend of the sacrum in the one case and in the other of a more general yielding, producing in both a considerable

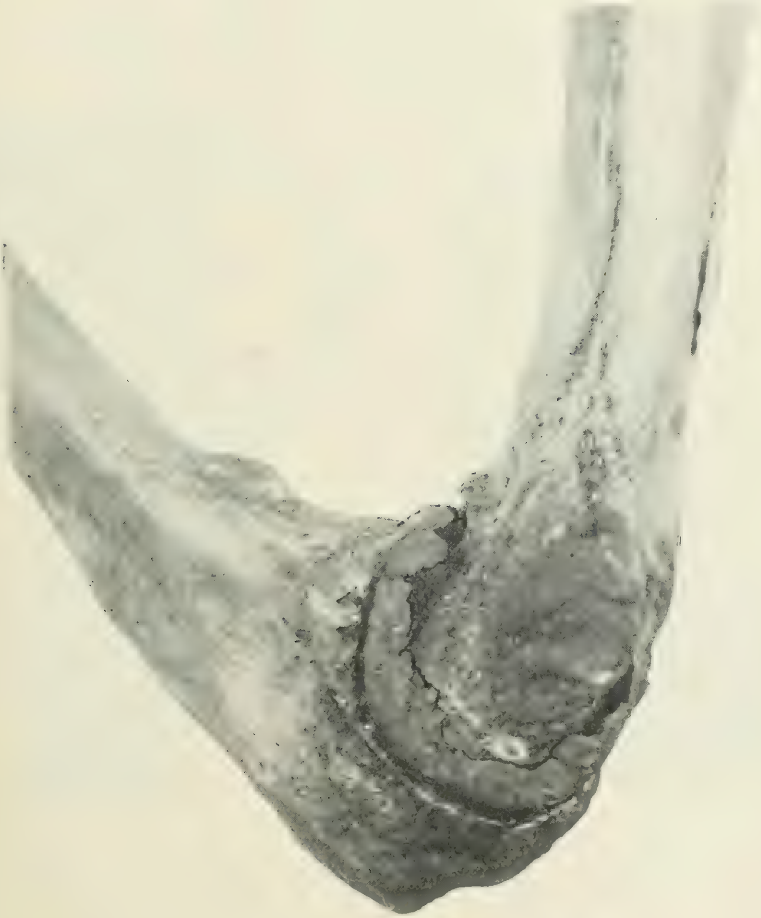

FIG. 27.
Right elbow-joint of coal-trimmer.

diminution of the conjugate diameter of the pelvic brim. The intervertebral disks disappear at the points of greatest pressure, their total bulk being proportionately very much less than in vigorous life. The shading represents alteration in the structure of the bones, loss of function of a part rendering the cancelli less conspicuous while the transmission of an excessive pressure exaggerates them.

So far I have confined my remarks and illustrations entirely to the consideration of the bony skeleton and its articulations; what I wish now to do is to point out that the same mechanical principles govern the soft parts and modify their structure. In addition to this we have the fact that the several viscera, and much more especially the brain, exert a varying influence on the bone-forming capacity of the individual.[1] All these evolutionary processes are useful to the individual in his special

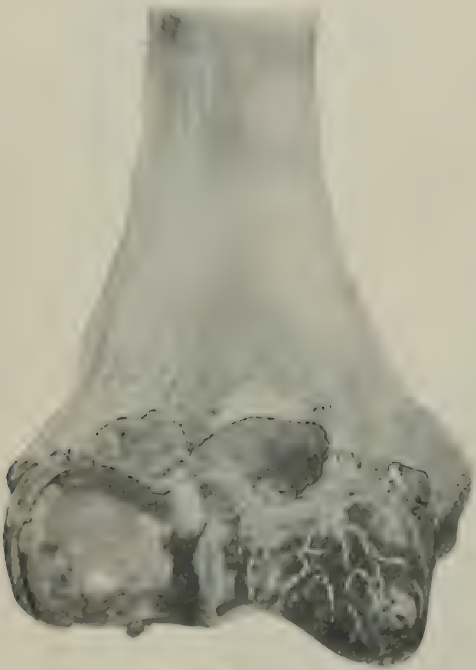

FIG. 28.

Lower end of right humerus of coal-trimmer.

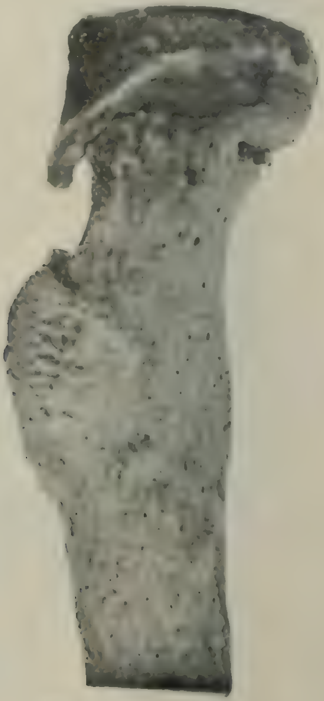

FIG. 29.

Upper end of right radius of coal-trimmer.

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[1] "The Factors which determine the Hypertrophy of the Skull in Mollities Ossium, Osteitis Deformans, Rickets, and Hereditary Syphilis," *Lancet*, 1888, i, p. 815.

mechanical relationship to his surroundings, since they economize expenditure of energy. One must, however, recognize that while these processes are advantageous in the pursuit of his particular occupation,

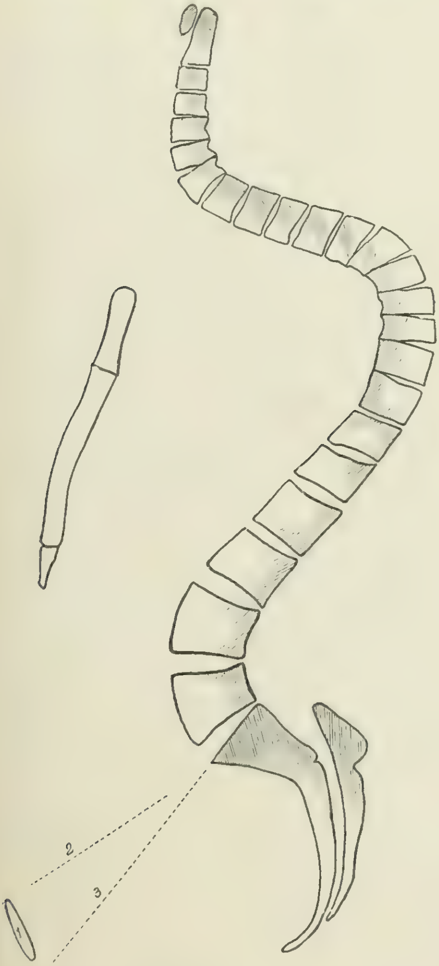

FIG. 30.

Spinal column of old woman.

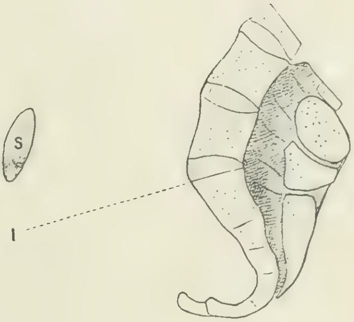

FIG. 31.

Lower part of spine of feeble old subject.

they are subsequently all more or less instrumental in tending to shorten his life.

We are quite familiar with the relatively short life of the labourer, the duration of whose existence may be said to vary inversely as the severity of his occupation as regards manual work. How far these mechanical agencies act as evolutionary factors in determining changes in the offspring I have considered in an earlier paper.[1]

In civilization the trunk is retained in a vertical position during the entire daytime, the reclining posture being only assumed at night. Even then the horizontal posture is modified in character from that normally assumed in savage life. The resting posture of the trunk is the prone position, which is that which is naturally assumed in sleep upon the ground. While in the erect position of the trunk all the viscera tend to displace downwards towards or into the true pelvis, in the prone position the tendency is for them to fall upwards and forwards out of or in a direction away from it. If these attitudes were assumed in a normal association the structures in the abdomen would retain their normal relationship to the abdominal wall and to one another. If, however, the attitude of activity is not compensated for sufficiently by the corresponding resting posture changes will certainly take place, varying in degree with the failure of compensation. It is now our business to study in detail the changes which arise in the several abdominal structures in consequence of this. The portion which is affected in the first instance is our drainage scheme, or, as it is commonly described, the gastro-intestinal tract.

The large intestine forms the cesspool of this tract. As it retains its contents for a comparatively long time, and as most of these contents are of solid consistence, it is natural that by its weight and situation it should tend to become displaced earlier in the lifetime of the individual.

In the erect posture the cæcum and ascending colon become filled with more or less fluid contents, and by exerting a hydraulic pressure on the cæcum tend to tire out its muscular wall and dilate its cavity. By the same means there is a tendency for the dilated cæcum to be displaced downwards into the true pelvis, where its presence is detrimental to the true comfort and functioning of the viscera, to which that space normally belongs.

This tendency to displacement in the cæcum in a downward and inward direction may be regarded as acting along the resultant of a parallelogram of forces, and to oppose this tendency resistances develop which correspond in position and action to the sides of the parallelogram. These lines of resistance are crystallized first as bands, and later as distinct membranes, which, as the outer limbs of the parallelogram, connect the peritoneum lining the abdominal wall to the outer surface of the colon in its immediate vicinity. A larger area of abdominal peritoneum and of colon is gradually involved, and later still, as is the case in all resistances which are crystallized in peritoneum, the bands are replaced by a layer of peritoneum which clings and tends to support the colon from the outside. As these bands and membranes develop, blood-vessels form in them which later may become sufficiently large to require to be ligatured if divided. This particular resistance, when fully developed into a membrane, has recently been termed "Jackson's membrane," and the manner in which it should be dealt with has afforded surgeons an excuse for doing many operations whose object is not very apparent, and which suggest ignorance of the factors determining its development as well as of its function. These crystallized resistances of peritoneum are not limited to the cæcum, but may extend up along the outer aspect of the ascending colon, even to the hepatic flexure.

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[1] "Can the Existence of a Tendency to Change in the Form of the Skeleton of the Parent result in the Actuality of that Change in the Offspring?" *Journ. Anat. and Physiol.*, 1888, xxii, pp. 215–24.

At this flexure a number of new bands are formed overlying the normal peritoneum and attaching the flexure. These bands as they develop drag the flexure upwards, and in some cases produce a distinct obstruction. As they are fixed in close relationship with the right kidney, they not infrequently produce symptoms which are regarded as being renal in origin. In consequence of this the right kidney has been frequently exposed in an ineffectual attempt to find a stone, and the surgeon has subsequently been agreeably surprised to find that his patient has been relieved of his pain merely by the exposure of the kidney. The relief, usually temporary, really results from division of many of these bands and the freeing of the obstruction at the hepatic flexure. The bands or membranes are liable to constrict the lumen of the ascending colon usually at the level of the crest of the ileum.

The inner limb of the parallelogram of forces is represented at an early period by opaque streaks on the under surface of the mesentery, attaching the last few inches of the ileum, and commencing at its base at a point most distant from the bowel. The reason of this development is obvious. Besides retaining the end of the ileum in position, the mesentery assists through the medium of the termination of the small intestine in holding up the cæcum and in tending to oppose its downward displacement.

The thickening in the under surface of the mesentery becomes more distinct, and later develops into a membrane which extends to, and secures, the under surface of the circumference of the ileum, and gradually creeps around it till it reaches that portion of the circumference immediately opposite to the attachment of the mesentery. As it extends around the ileum, it also contracts, with the result that it twists the ileum on itself along its longitudinal axis. In its earliest development, as is the case with most conditions which evolve during the lifetime of an individual, because of a variation from the normal in the relationship to his environment, the effect of this acquired ligament or mesentery is useful and physiological, but later, when it kinks and obstructs the lumen of the ileum, it exerts a progressively deleterious effect on the well-being of the individual. I have already called your attention to the harm done by structures which, when first evolved, perform a useful function, but later produce effects which are progressively deleterious. I have indicated the mechanics of these crystallized resistances in figs. 32 and 33.

Fig. 32 represents the distended loaded bowel dragging upon the resistances evolved to oppose its downward displacement into the true pelvis. The resistances external to the cæcum are indicated as arrows showing the general direction of the strain exerted by bands, adhesions, and omental structures. The lowest of these secures the appendix, which in these circumstances is kinked. The effect exerted upon the ileum at its point of fixation by the weight of the loaded cæcum on the one side of it, and by that of the obstructed loop of the ileum on its proximal aspect, is figured in the diagram.

Fig. 33 represents the anchored portion of ileum with its mesentery in an antero-posterior plane. The contracting bands which develop on the under surface of the mesentery, shortening it and attaching progressively a varying portion of the circumference of the ileum in the vicinity of the mesentery with the consequent alteration in the form of the bowel, are shown by the arrow. The compensatory shortening of the entire mesentery in this plane is not indicated in the diagram.

It is clear that in this condition the inner limb of the parallelogram of forces is represented by the portion of the ileum distal to the kink, by the contracting acquired membrane which creeps around the circumference of the bowel, and by the posterior layer of the mesentery to which the membrane is attached. The upper layer of the mesentery presents no change, as abnormal strain is not exerted along it but through the under layer only. In these circumstances the portion of ileum between the cæcum and the attachment of the acquired ligament is made to perform the function of a ligament very much more than is the case in the normal arrangement.

In figs. 34 and 35 are excellent clinical illustrations of this condition. They are contained in a paper by Dr. Charles H. Mayo in *Surgery, Gynæcology and Obstetrics*, entitled "Intestinal Obstruction due to Kinks and Adhesions of the Terminal Ileum,"[1] in which that distinguished surgeon describes his views on the causation of the condition.

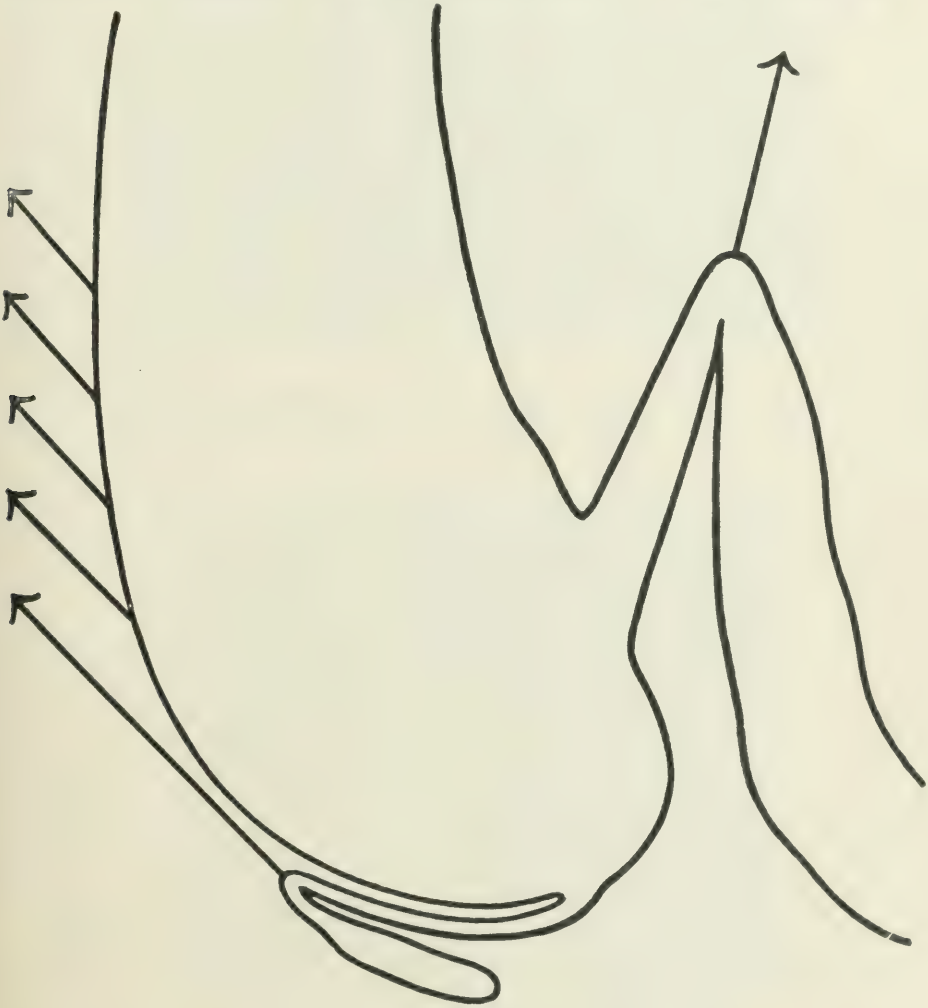

Fig. 32.

You will note that in the case here illustrated the appendix is fixed by the bands forming part of the outer limb of the paralellogram of forces, and that it has no anatomical relation whatever with the acquired evolutionary band forming the ileal kink, nor can it by any possibility have had any share in its causation. This may seem an unnecessary observation, but it is called for, since certain surgeons labour under the delusion that the band is produced by an inflammation that started spontaneously in the appendix. Against the supposition that these acquired bands or mesenteries are inflammatory in origin is the fact that they commence at the reflexion of the peritoneum at a point most distant from the intestine, and then gradually approach and secure the bowel. Also, they only exist on the surface of peritoneum on which strain is exerted, and they correspond accurately to the lines of force.

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[1] *Surg., Gyn. and Obstet.*, Chicago, 1911, xii, p. 227.

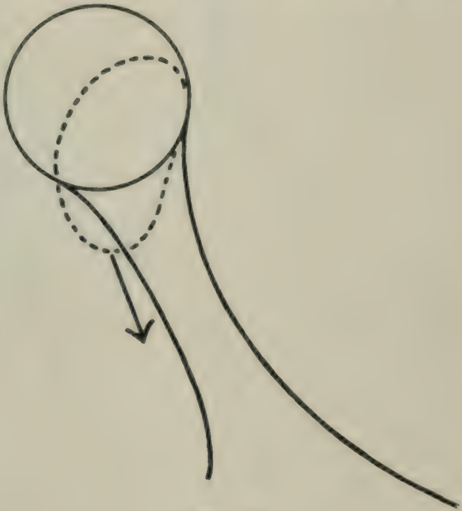

Fig. 33.

The gradual growth of these membranes can be studied in every degree of development in the mesentery of the iliac colon. A very little consideration shows that the inflammatory origin is absurd.

I would also refer to excellent work on the same subject, as well as on stasis generally, by Dr. Franklin H. Martin[1] and by Dr. Coffey,[2] which has appeared in *Surgery, Gynæcology and Obstetrics*, and with all of which you are doubtless quite familiar.

From an examination of the several illustrations, it is apparent that any accumulation in the ileum proximal to the kink aggravates the obstruction more or less effectually, and that in the supine, and especially in the prone, posture the reduction of the strain exerted in

[1] *Surg., Gyn. and Obstet.*, Chicago, 1911, xii, pp. 34-40.
[2] *Ibid.*, 1912, xv, pp. 365-429

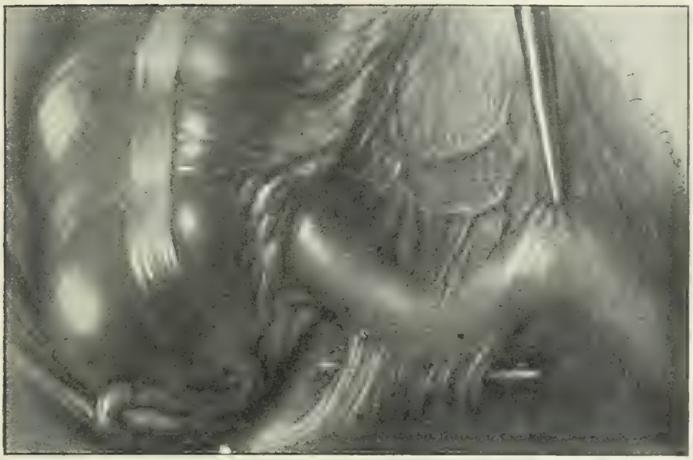

Fig. 34.

Shows the ileal kink with the band of peritoneum which produces it.

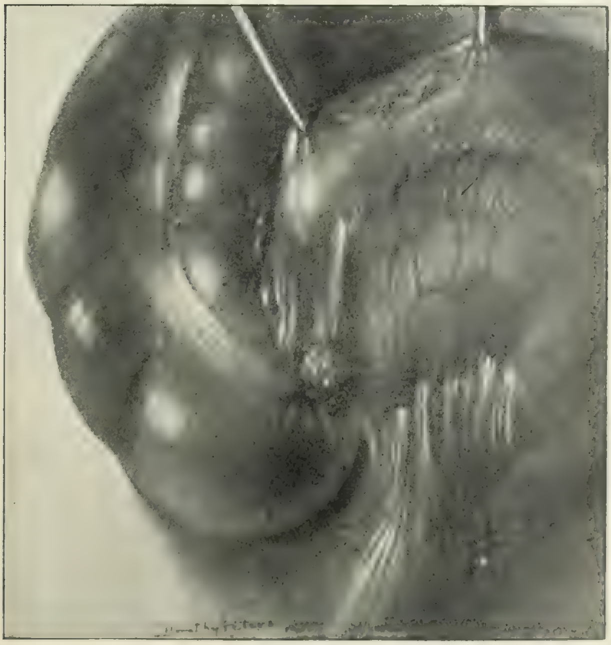

Fig. 35.

Represents the condition after division of the controlling membrane.

the kinked bowel is probably sufficient in most cases to re-establish the effluent through it more or less satisfactorily. This is the explanation of the improvement which results in conditions presenting a marked ileal kink when the recumbent posture is assumed.

A factor of more immediate urgency in the development of resistances to the descent of the cæcum is the share taken by the appendix. This structure being a firm one, and being securely fixed to the summit of the cæcum, offers an irresistible attraction to the acquired or evolutionary bands. It may be gripped by one of them forming the outer limb of the parallelogram as in figs. 32 and 34, when the portion of its length, which intervenes between the point of attachment by the band and the cæcum, is made to perform the function of a ligament. Should the grip of the acquired band secure the appendix at a point in its length, it is liable to kink the appendix when the blind part distal to the secured point readily becomes obstructed. The worst that such an appendix can do is to become inflamed, producing the condition called appendicitis. Should the appendix, on the other hand, form part of the inner limb of the parallelogram of forces, it is caught up at a point in its length, and is secured by an acquired band or ligament to the under surface of the mesentery. To reach this attachment it has to pass almost directly upwards behind the termination of the ileum. In these circumstances the inner limb of the parallelogram is formed by a portion of the proximal appendix stretched to its utmost and continued in the lower layer of the mesentery into a thick, dense, fibrous and peritoneal band, which has developed in order to secure it in this position. If the appendix is strong and the grip afforded by the acquired ligament or membrane in the under surface of the mesentery is secure and efficient, the acquired band which develops for the purpose of utilizing the termination of the ileum does not form. If, on the other hand, the hold of the appendix in the mesentery is not effective in restraining the cæcum, in that proportion does an acquired mesentery and ileal kink result. When they co-exist, the band fixing the appendix may become continuous with the band kinking the ileum. This has led those observers who consider that the fixation of the appendix is secondary to a primary inflammation of that organ to imagine that the membrane producing the ileal kink is also formed by an inflammation of the appendix. A careful examination of the condition at once disposes of such a supposition.

The appendix, fixed to the mesentery, exerts a pressure upon the end of the ileum, when the cæcum falls into the pelvis and the ileum becomes dilated and distended behind the control, which is exerted by the unyielding and practically rigid band formed by the proximal portion of the appendix and the ligament which secures it. As the appendix is usually gripped in such a manner that only a portion of its length is held by the acquired ligament, the portion distal to this, as already pointed out, is liable to become obstructed beyond its kink, and an inflammation of its distal extremity, or in other words an appendicitis, may arise. Therefore, such an appendix, by acting mechanically, may not only produce all the consequences associated with the damming back and fouling of the contents of the small intestine, but its end becoming inflamed, it adds to these mechanical symptoms those of appendicitis.

It has been observed by certain operators that the removal of the appendix may occasionally bring about a cure of such conditions as duodenal ulcer, &c., and it has been suggested that duodenal ulcer and allied conditions are produced by an infection by organisms which grow in the appendix, or it is assumed that they may be produced by other infections—e.g., pyorrhœa alveolaris, &c.

While the fact is correct that the removal of the appendix is occasionally followed by the cure of duodenal ulcer and allied conditions, the explanation of the phenomenon is incorrect. The ulcer and other allied conditions get well because the appendix, which was removed, had controlled the effluent in the ileum, and the freeing of this ileal effluent has of necessity relieved the results of its obstruction, of which the duodenal ulcer was one and only one.

The problem of ileal obstruction, either by an ileal kink or by the pressure of an appendix secured to the back of the mesentery, or by both sharing in the production of obstruction of the ileal effluent, is one of the greatest importance and frequency. If I might so describe it, it represents the failure of our present condition of civilization.

I have endeavoured to indicate diagrammatically, in fig. 36, the normal condition of the stomach, small intestine, and cæcum, and in fig. 37 the several changes which result from ileal obstruction.

I have endeavoured in fig. 38 to indicate the mechanism of the appendical tie or resistance when it forms the inner limb of the parallelogram of forces. The cæcum and ileal loop are both distended with fæcal contents and have fallen into the true pelvis. The end of the ileum is hung over, and its lumen is diminished by, the fixed portion of the appendix secured by its acquired membrane or crystallized resistance, which is represented by the arrow. It is apparent that the greater the drop of the cæcum as it pivots on the fixed appendix, and the greater the dropping and distension of the ileum, the more complete does the obstruction by the appendical tie become.

I should further like to point out that while the obstruction to the passage of the contents of the ileum into the cæcum usually results from a kink in the ileum, a suspending appendix, or from both together, yet the cæcum and ileum may bear such a mechanical relationship to one another, either because of the fixation of the cæcum in the iliac fossa by the bands of resistances forming the outer limb of the parallelogram of forces, or because of a distension of a large cæcum with fæcal contents occupying the true pelvis, that the ileum is unable to transmit its contents at a normal rate into the cæcum, so that it becomes obstructed and distended, and its contents infected by organisms to which it is unaccustomed. Associated with the obstruction at the end of the small intestine, the lowest portion of the ileum presents a condition of dilatation and hypertrophy varying in degree with the character of obstruction

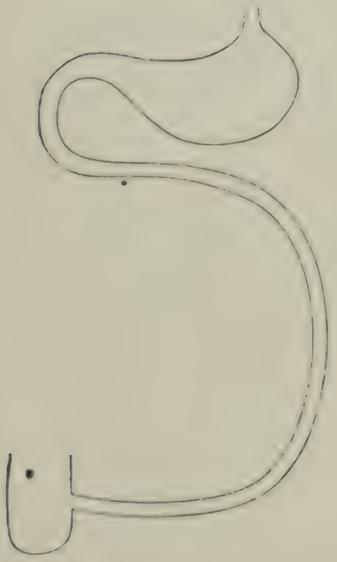

FIG. 36.

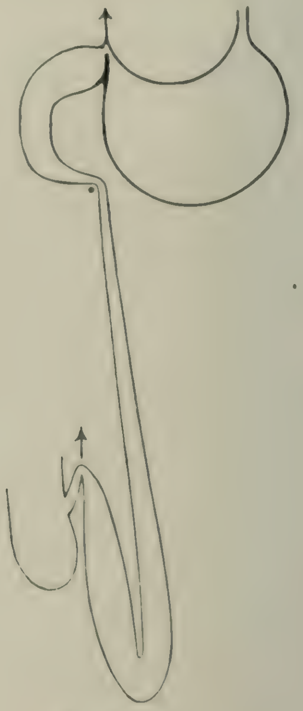

FIG. 37.

and the vitality of the individual. If the subject is still fairly vigorous the bowel is very distinctly hypertrophic, but if the condition is very advanced the intestine is dilated, or even bluish, as it would appear on the post-mortem table. The pull exerted by the prolapsed small intestines, rendered heavy with their accumulated contents, exerts through the medium of the jejunum a strain on the termination of the duodenum. The duodenum usually terminates vertically at the root of the transverse mesocolon, where it is continued into the jejunum as a gentle curve. This I have endeavoured to indicate in fig. 39. The strain exerted by the jejunum produces a kinking of the duodeno-jejunal junction, together with a twisting of the commencement of the jejunum. This produces an obstruction to the effluent from the duodenum. To oppose this drag upon the jejunum and the obstruction of the duodenal outlet consequent on it, resistances are laid down as peritoneal bands. These run upwards and outwards from the commencement of the outer aspect of the jejunum to the peritoneum lining the adjacent abdominal wall. At first

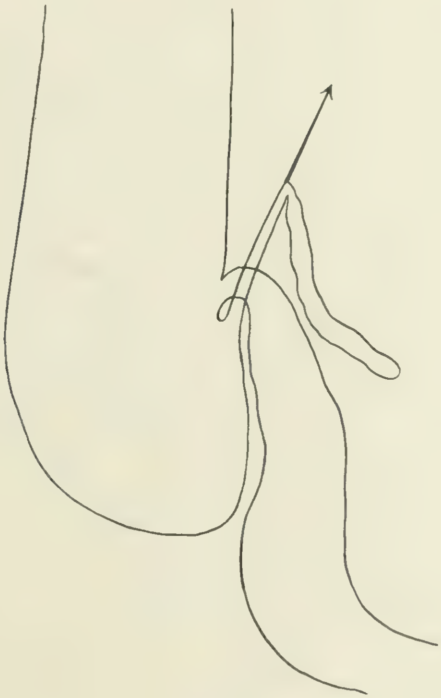

FIG. 38.

these acquired or evolutionary bands or ligaments serve a useful and physiological purpose, but after a time, as they contract they secure the bowel in this vertical position, and no longer permit it to return to the normal curve in any position of the body, so that the duodenal outflow is permanently obstructed, this obstruction becoming exaggerated at times when the patient is exhausted. I have attempted to represent the kinking and twisting of the bowel in fig. 40, and also to show the situation and direction of the crystallization of the lines of resistance in peritoneal ligaments or membranes.

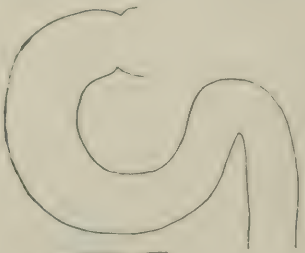

Fig. 39.

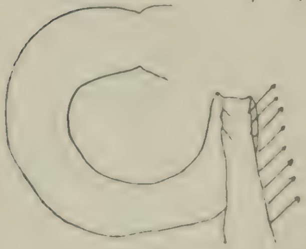

Fig. 40.

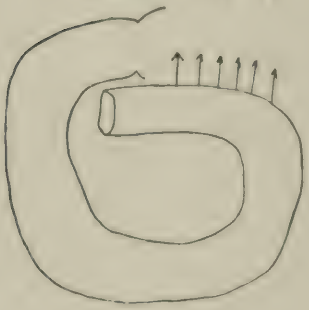

Fig. 41

It happens not uncommonly that Nature, recognizing the importance of avoiding annulation at and strain upon the duodeno-jejunal junction, develops resistances crystallized in membrane which fix the jejunal loop so as to avoid kinking. The jejunum becomes secured from left to right to the under surface of the transverse mesocolon by peritoneal bands or membrane. This I have indicated in fig. 41. By this means Nature obtains the same result that is arrived at by the surgeon who performs an anterior or posterior gastro-enterostomy for duodenal ulcer which does not mechanically obstruct the lumen of the duodenum. He simply secures the commencement of the jejunum, so that it cannot fall vertically and continue to obstruct the duodenum. Should the stomach be much dilated he may not even succeed in doing this owing to its continued dropping. In no case does the relief of the duodenal obstruction relieve the obstruction at the end of the ileum, with the consequent stasis and infection of the small intestine, and with the auto-intoxication which results from it.

The obstruction at the duodeno-jejunal junction produces a distension of the duodenum with dilatation of its lumen and hypertrophy of its wall. This is most marked in the first portion which is covered by peritoneum and has not the same support from the adjacent structures that the rest of the duodenum possesses. In consequence of the distension of the first piece of the duodenum and because of the infection of its contents by organisms to which it is unaccustomed, and of the depreciation of its vitality by the auto-intoxication, changes take place in its mucous membrane which are at first of the nature of vascular engorgement, and later of inflammation of varying degree, till an ulcer may form which may eat its way through the muscular and peritoneal coats. It may heal, cicatrize, and stenose this portion of the bowel.

The infection of the duodenal contents results in the fouling of the ducts which open into the duodenum—namely, the pancreatic and common bile-ducts. The extension of organisms up the duct of the pancreas, assisted by the depreciation in the vitality of this organ from the general auto-intoxication of the tissues, and possibly from the presence of deleterious organisms in the circulation, produces changes in the pancreas which vary from a mere vascular engorgement to the several stages of inflammation, and lastly may end in cancer of the organ itself.

The infection of the gall-bladder produces gall-stones in the gall-bladder, and on rare occasions in the hepatic ducts. Changes ensue in the wall of the gall-bladder consequent on the presence of stones in it or in the cystic duct. These changes are of an inflammatory or cancerous nature. Similar changes may occur in the liver and in its ducts secondary to stone in the common bile-duct, pancreatic inflammation, &c.

Later, the cicatrization of the duodenal ulcer produces more or less definite occlusion of the lumen of this portion of the bowel, with consequent changes in the stomach. Associated with the distension of the duodenum the pylorus develops a varying amount of spasm, obviously to prevent the return of the duodenal contents into the stomach. This produces a distension and dilatation of the stomach with a varying degree of hypertrophy of its coats. This accumulation of material in the stomach throws a strain upon the lesser omentum at its right limit, and resistances to the downward displacement of the pylorus crystallize as bands or adhesions which connect the pylorus progressively to the under surface of the liver and gall-bladder. An acquired band may extend from the under surface of the liver to the pylorus, which it thus fixes, and by passing on into the transverse colon will tend to secure that structure. Thus by transmitting its strain directly to the liver it will free the stomach from a portion of the weight of this part of the bowel. The distended, loaded stomach sustains the greatest strain along its lesser curvature. The strain is greatest near the pylorus because of the limited area of resistance and gradually diminishes as it approaches the œsophagus. This latter structure may be regarded as the chief supporting ligament of the stomach in the erect posture. Any descent of the liver brings the strain in the lesser curvature nearer the œsophagus and farther away from the pylorus. The strain exerted upon the lesser curvature of the stomach by the contents accumulated in it is increased by those portions of the strain which are transmitted through the greater omentum to the convexity of the stomach by a loaded transverse colon. In consequence of this strain, assisted by the bacteriological and chemical changes in the gastric contents and by the lowered vitality undergone by the tissues of the stomach and of the body generally by auto-intoxication, changes take place in the mucous membrane which vary from an increased vascularity to a cancerous infection (*see* fig. 42).

To bring about ulceration of the stomach, it is apparent that the mechanism which is responsible for the production of ulceration of the duodenum has superadded to it a distension of the stomach, with probably much stasis in the transverse colon. Therefore, while in most cases recumbency relieves the ileal stasis and consequently the duodenal distension and so permits the ulceration to get well, in the case of the ulceration in the stomach the recumbent position does not cure the stasis nor does it relieve the pyloric spasm, nor remove the stasis in the transverse colon. This explanation of the causation of the gastric ulcer is also confirmed by the benefit which frequently results from an efficient gastro-enterostomy. By efficient I mean one which allows the free passage of gastric contents in the jejunum.

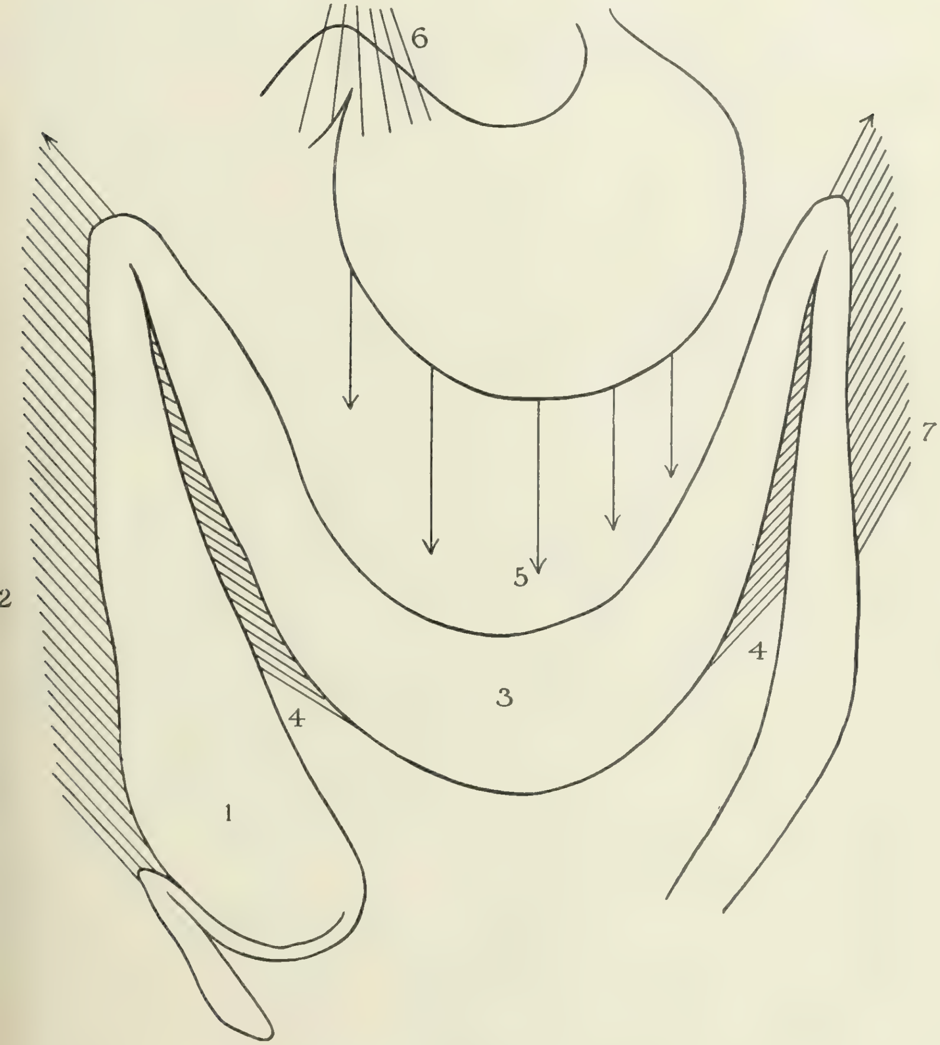

Fig. 42.

1 represents the prolapsed cæcum; 2 and 7, the crystallized resistances which tend to oppose the downward displacement of the large bowel and sustain some of the weight of the transverse colon transmitted through the crystallized resistances 4; 3, the transverse colon; 5, portion of the weight of the transverse colon transmitted through the great omentum to the convexity of the stomach; and 6, the acquired ligament that secures the duodenum and pylorus to the under surface of the liver and gall-bladder.

The rapidity with which duodenal ulcer can usually be temporarily cured by position, explains the great rarity of infection of such an ulcer by cancerous germs, while the difficulty of curing gastric ulcers renders them chronic and liable to such an infection.

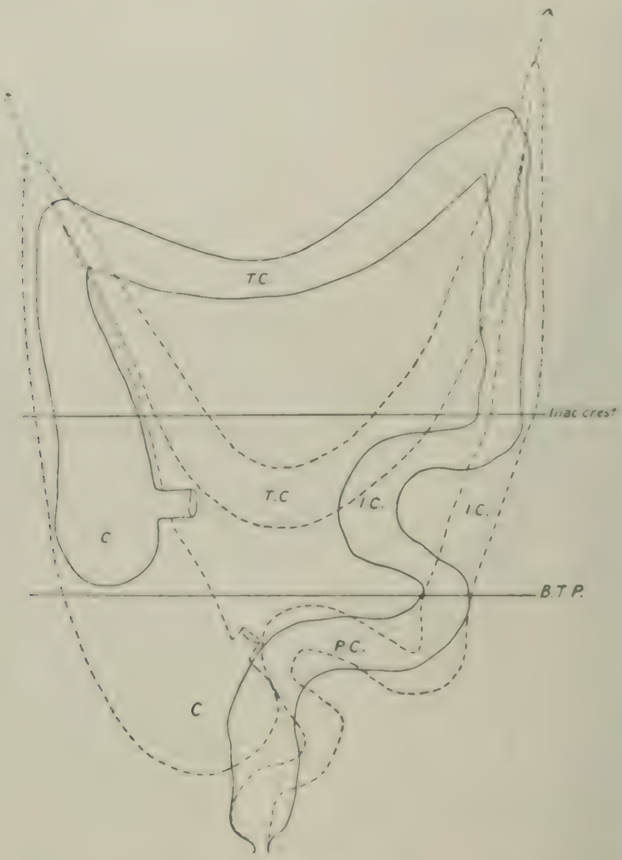

Fig. 43.

Fig. 43 represents the several variations from the normal which the large bowel may undergo. The normal condition is shown as a firm outline, and the altered condition as a dotted outline. Note the prolapse of the cæcum and transverse colon, the telescoping of the iliac colon and the elongation of the pelvic colon. *B.T.P.* indicates the brim of the true pelvis. The hepatic and splenic flexures are drawn up and kinked by the development of acquired resistances shown as arrows.

Returning to the large bowel, the transverse colon tends to drop. Part of its weight is transmitted to the convexity of the stomach through the great omentum and part through an acquired mesentery, which develops between the convexity and the ascending and descending colon. This acquired membrane is the crystallization of resistances to downward displacement of the transverse colon and is in the first instance useful since it serves to relieve the stomach of strain. Later by its contraction it renders the hepatic and splenic flexures more acute and affects deleteriously the passage of fæces through them. As I pointed out, this is the usual sequence in the case of changes which evolve during the lifetime of an individual. Much of the weight of the transverse colon is transmitted through the posterior layer of the transverse mesocolon, which shows distinct acquired thickening and sometimes independent membranes along the lines of greatest strain. The splenic flexure is, normally, much higher than the hepatic, and in cases of stasis it is still further elevated by the formation of membranes or acquired resistances, which by their progressive contraction accentuate the angulation and disability of this flexure. It is at the three flexures, and especially at the splenic flexure, that inflammatory and, later, cancerous changes are liable to develop in consequence of the constant irritation due to the increasing obstruction. The descending colon is fixed in a manner similar to the ascending colon by acquired crystallized resistances, which serve partly to secure the colon and partly to transmit to the abdominal wall the weight of the transverse colon.

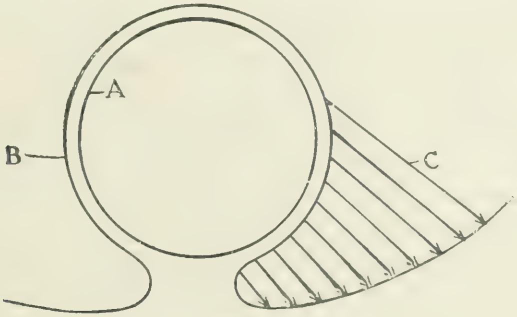

FIG. 41.

Transverse section seen from above. A, muscular and mucous coats; B, peritoneum; C, direction of adhesions.

Fig. 41 represents diagrammatically the development of these bands. They are liable in time, by their construction, to exert an excessive strain on some portion of the colon, and to produce a constriction in the lumen of the bowel with the consequences which follow upon it. Such an obstruction exists occasionally in the large bowel on the right side at the level of the iliac crest, where the abrupt change from a firm, bony relationship to that of a flaccid muscular wall renders the production of a kink more easy. The flexure or loop formed by the iliac colon is dealt with in the same manner as I have indicated in fig. 43.

Fig. 45 represents a transverse section through the sigmoid and mesosigmoid and the peritoneum covering the iliac fossa external to the mesosigmoid. As in the last diagram, the line B indicates the peritoneum and the line A the bowel. Owing to the possession of a mesentery of some considerable length, the bowel is at a much greater distance from the abdominal wall than the cæcum and ascending colon, which are immediately adjacent to it, and the mode of fixation differs somewhat in detail from the last described. The outer surface of the mesentery is gradually brought down to its base by the formation of adhesions, till the wall of the iliac colon itself becomes secured to the floor of the fossa. The mode and direction of the formation of these adhesions are represented by arrows, C.

Associated with the progressive fixation of this loop there is a diminution of its lumen and of its length, so that the bowel, which normally forms a loop of considerable size, moving freely at the end of a long mesentery, finally becomes telescoped as a short, straight, constricted tube, attached by a considerable area of its circumference to the abdominal wall quite devoid of any mesentery. In proportion as the bowel becomes fixed, its muscular coat wastes. The state of fixation and shortening of the sigmoid flexure, wasting of its muscular coats, and the general and occasionally also a very considerable local reduction of its lumen, and the obliteration of much of its peritoneal covering, are of great importance surgically, since they interfere very materially with the passage of material through the sigmoid and render any operation on this portion of the bowel abnormally difficult. In consequence of the irritation of the passage of firm fæcal contents through this fixed bowel, inflammation and later cancerous changes develop in it. The former condition—namely, the inflammatory one—has been dealt with in his usual masterly manner by Dr. W. J. Mayo in several papers under the title of "diverticulitis." The fixation of the sigmoid by the acquired adhesions may be of such a nature as to narrow its lumen in one or more places to such an extent that symptoms of obstruction may arise. This results from a failure of many of the bands of adhesion to secure a continuous grip upon the whole length of the mesentery of the loop. In consequence, the part, or parts, of the sigmoid which have become effectually anchored form kinks or obstructions, while the rest of the loop is distended. I have endeavoured to indicate this diagrammatically in fig. 46. In this the end of the descending colon, DC, is shown with the loop of the iliac colon and the upper part of the pelvic colon. The radiating lines represent the acquired adhesions which have secured the centre of the sigmoid loop, but have failed to grip the portions of the bowel above and below. Consequently the lumen of the intestine is reduced in two places, chiefly at the point where the centre of the loop is fixed, and to a lesser extent at the junction of the distal loop with the rectum.

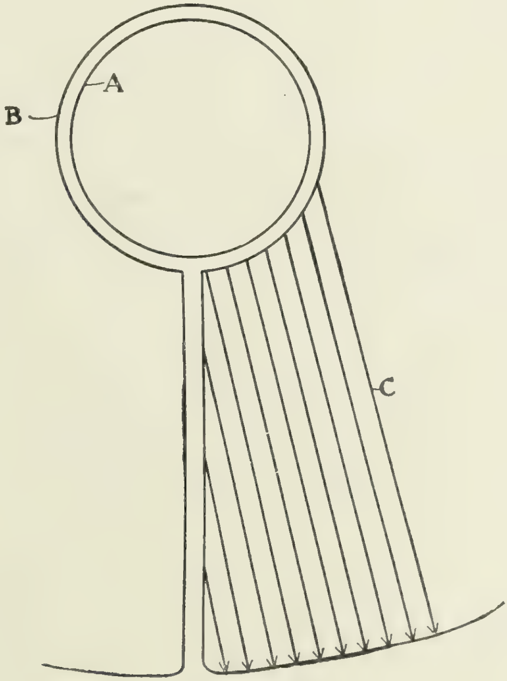

Fig. 45.

A, muscular and mucous coats ; B, peritoneum ; C, direction of adhesions.

Owing to the bowel having escaped beyond the control of adhesions, except at its commencement and termination, the sigmoid, instead of becoming shortened, atrophied, constricted, straightened, and fixed, may become very much more elongated, dilated, and mobile than normal, its muscular coat being correspondingly hypertrophied. At the same time the extremities of the loop held in the grip of adhesions are closely approximated, while the lumen of each is constricted, especially when the loop is distended with fæcal matter. This approximation of the extremities of the loop greatly facilitates its rotation.

Fig. 47 is intended to represent the mode of production of a volvulus of the iliac colon. DC, S, and R indicate respectively the lower limit of the descending colon, the sigmoid loop, and the upper part of the rectum. The radiating lines show the acquired adhesions which have secured and approximated the extremities of the sigmoid, but have failed to obtain a uniform grip upon the mesosigmoid generally. The mode of obstruction to the passage of fæces from the sigmoid into the rectum is depicted. This obstruction may become more complete because of a rotation upon the loop upon the base formed by its approximated extremities, or because of an excessive sagging of the lower part of the loop exerting a drag on its lower limit sufficient to occlude its lumen. A certain amount of obstruction to the passage of fæces from the sigmoid into the rectum is always present. It is in these cases that complete intestinal obstruction may occur, owing to the twist or rotation of the overloaded and elongated loop becoming sufficient to prevent the passage of any material from the sigmoid loop into the bowel beyond. It will thus be seen that the so-called volvulus of the sigmoid (occurring spontaneously and not as an after-effect of an abdominal operation) is a phase of a condition of partial obstruction of long standing, the loop being habitually over-distended with intestinal contents, and the passage of fæces from it being chronically interfered with. I do not think that acute volvulus of a perfectly normal sigmoid can occur spontaneously. The same remarks apply equally to volvulus of the cæcum.

I wish now to call attention particularly to the great importance of the last kink or obstruction which is developed in our drainage scheme and which affects the large bowel where it crosses the brim of the true pelvis on the left side. The fuller recognition of the function of this kink, the lowest in position, has helped me to understand the physiology of the large bowel both in the savage and civilized communities. It has also enabled me to deal surgically with the conditions of defective drainage of the intestine more effectually and at less risk to the life of the individual than I was previously able to do. This kink develops very early in life; indeed, I believe the bands which form on the outer surface of the mesentery of this portion of the large bowel, and which, like all other acquired mesenteries and adhesions, are the crystallization of lines of resistance to downward displacement, are the earliest to form in the body. They appear before the erect posture is assumed. I have

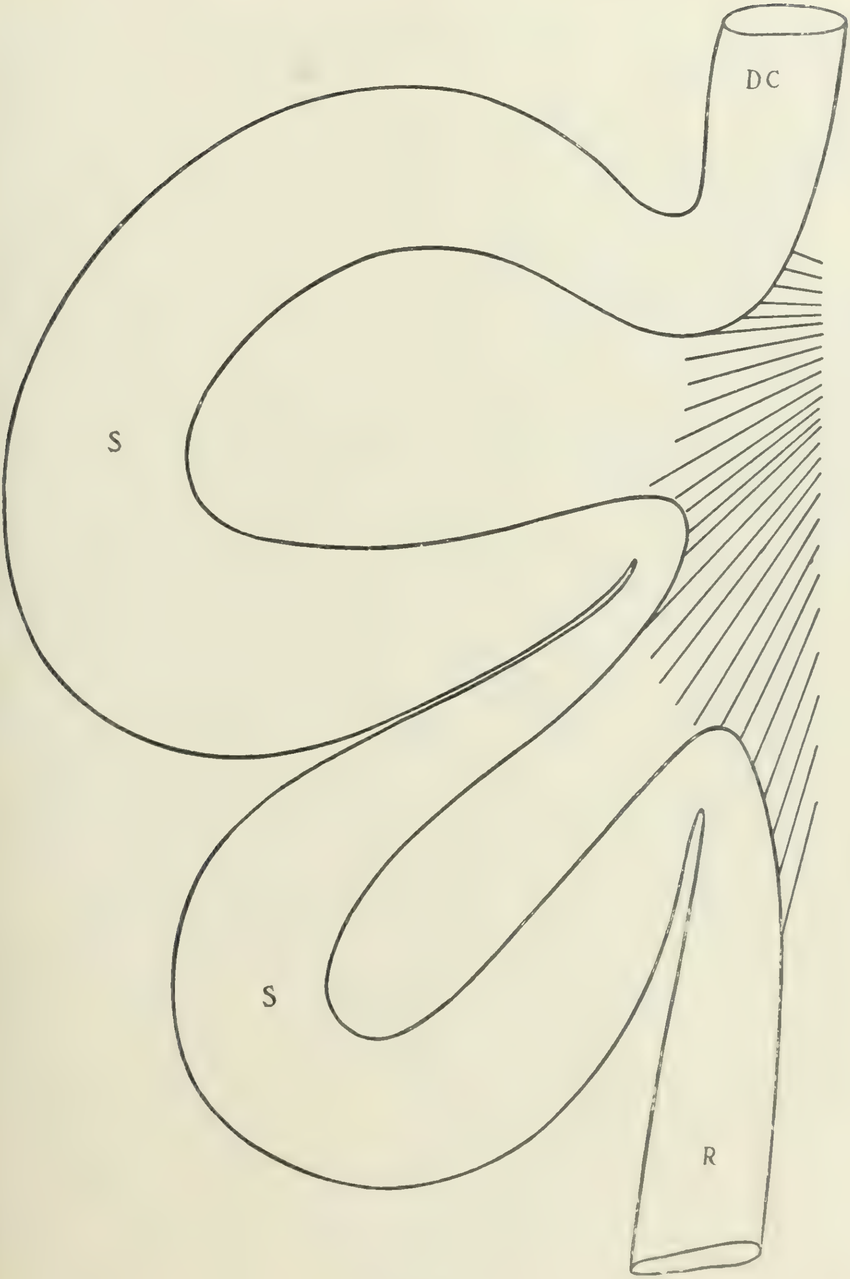

FIG. 46.

found them excellently developed in a child 2 years old, whom I short-circuited for extensive tuberculous disease of the hip-joint. This band obviously develops in order to resist the tendency to the downward displacement of the large bowel into the true pelvis, where it would seriously incommode the functioning of the several organs which already occupy it and is continuous with those that secure the iliac colon. As this new band or mesentery contracts and becomes a definite ligament quite distinct from the normal mesentery, on whose posterior aspect alone it exists, it fixes the large bowel securely at this level, and performs a function of the greatest importance. It is so arranged as to oppose the backward passage of fæces from the pelvic colon upwards into the large bowel, which is likely to take place if this kink or obstruction is not efficiently developed. It is often supposed that the fæcal matter moves continually onwards in its progress through our intestines. That this is not always so is shown exceedingly well in those cases in which this particular kink or obstruction is not developed.

When I first performed ileo-colostomy for intestinal stasis, I divided the ileum and put it into the iliac loop, as it was usually the portion of the large bowel most readily and safely adapted for the performance of this operation. At the same time, great precautions were taken to free the obstruction at the pelvic brim, to which I have just called your attention, by dividing the new mesentery and suturing it over in such a way as to try to obviate its re-formation. This operation was frequently followed by the passage of material back along the descending, transverse, and ascending colon to the cæcum, often necessitating the removal of the large bowel at a later date.

The ovary and Fallopian tube are often involved in these adhesions which fix the colon to the pelvic brim, and the adherent ovary very often undergoes a cystic degeneration. As it increases in size it forms a space in the acquired mysentery in which it moves, its outer wall and the lining of the space enclosing it acquiring a peritoneal covering. Later it destroys the surface of acquired mesentery, which covers its outer aspect, and becomes free except for its attachment to the broad ligament. How far the fixation of the left ovary in adhesions is responsible for the cystic degeneration it undergoes you must judge, but I have seen and demonstrated on many occasions the association of the conditions here described in any stage of development. In some cases the adherent uterine appendages may become infected. The inflammatory process may involve the portion of the colon to which they are attached, and the lumen of the bowel may be diminished

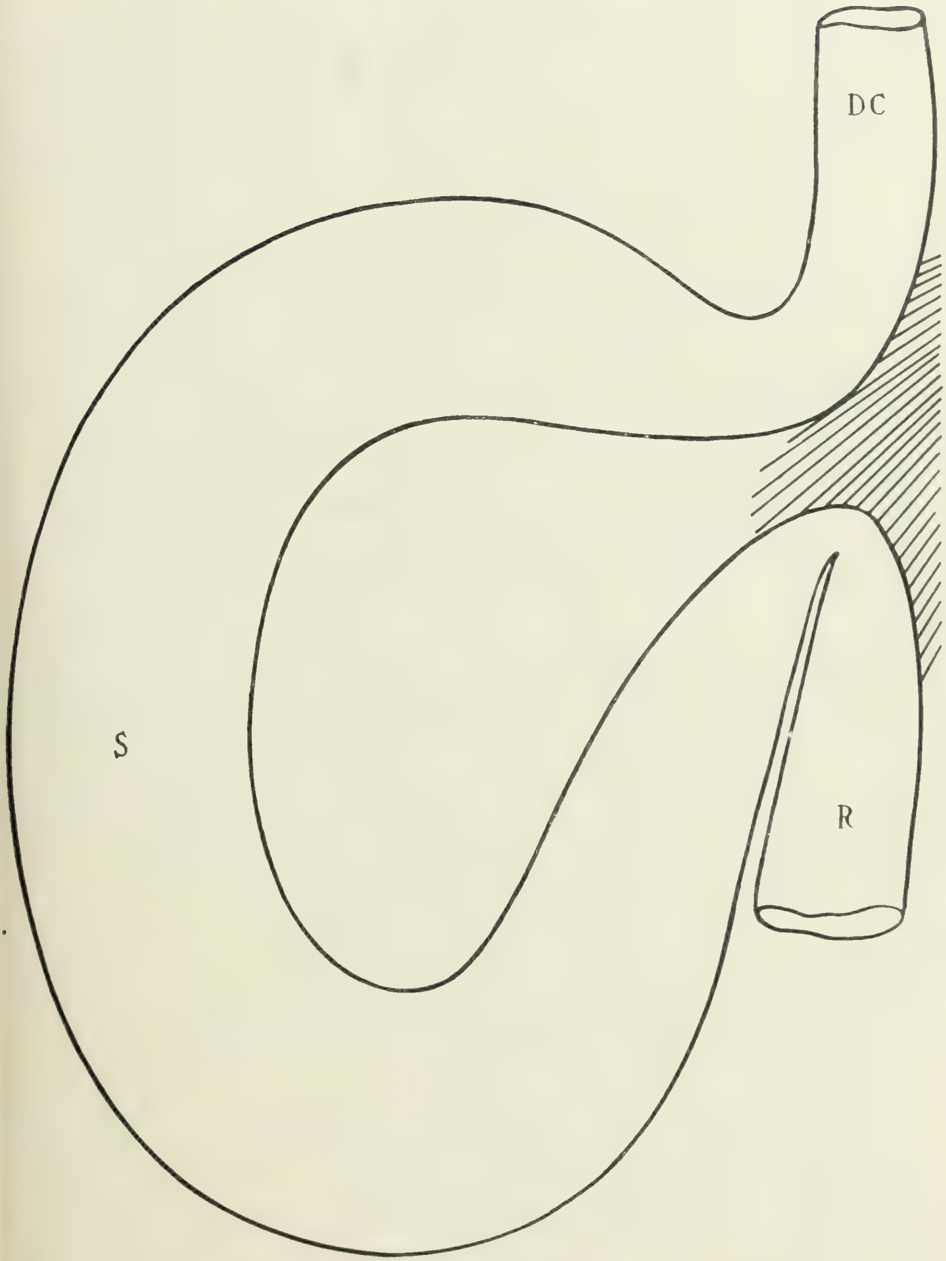

FIG. 47.

sufficiently to produce symptoms of the obstruction and simulate growth. In removing such an infected ovary it is sometimes necessary to excise the portion of colon involved, either because of its constriction or because an abscess in or about the ovary may have perforated its wall or damaged it extensively.

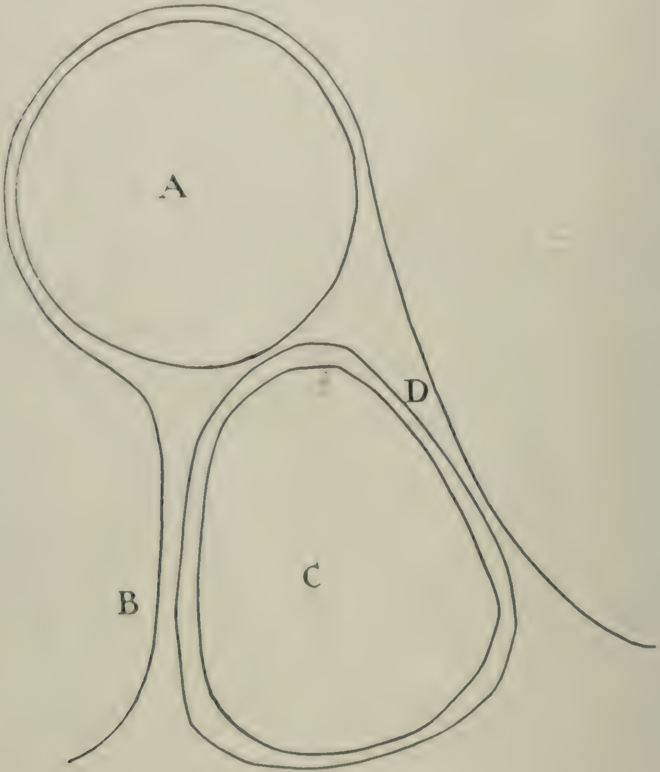

FIG. 48.

A represents the colon in transverse section ; B, the original mesentery ; C, the cystic ovary lying free in a cavity ; and D the remains of the acquired adhesions.

The pelvic colon is sometimes enormously elongated, especially in young life. This is almost invariably a very marked feature in the condition of chronic intestinal stasis, which is associated with tubercle, rheumatoid arthritis, Still's disease, &c. This is true to a lesser extent as age advances. Consequent on the elongation of the pelvic colon this portion of the bowel puddles in the floor of the pelvis and the multiple angulation due to its plication affords an increasing obstacle to the passage of firm fæcal matter through it. The greater the strain exerted by the abdominal muscles during defæcation the more difficult is it for the material to pass round the sharp angles which are accentuated by the pressure. When the large bowel has been removed or short-circuited the fluid contents pass with less difficulty out through the pelvic colon, but even in these circumstances the constipation in the residual pelvic colon may cause much difficulty to the patient and bother to the surgeon. It is easy to realize how readily inflammation or cancerous infection of the pelvic colon may arise in these circumstances at a point of stress where the mucous membrane has been habitually annoyed by the impact of the firm end of a motion.

The greater frequency of chronic intestinal stasis in the female sex is readily explained by the difference in the mechanical conditions in the sexes. In order that women should perform their function of reproduction their anatomy presents certain definite variations from the male, all of which result in a diminution in capacity to perform hard manual work. The calibre of the cavity of the pelvis is relatively greater than in the male: the lower chest is relatively smaller, the sternum is relatively shorter, the manubrium is relatively longer, the interval between the first and second costal arches is relatively greater and the gladiolus is relatively shorter; the upper portion of the chest is relatively longer; the number of ribs articulating with the sternum being seven and often less than seven, the lower costal cartilages being placed almost vertically and having a very insecure grip on the sternum, while the abdomen, or the interval between the sternum and pelvis, is relatively greater. There are many other differences, for details of which I would refer you to a paper in the *Obstetrical Society's Transactions*, on the chief factors which determine the form of the pelvis in the two sexes.[1] The result of these changes is to render the woman less capable physically than man to meet the conditions of civilization. The habits of life, and the nature of dress, and the concomitants of pregnancy, all assist in reducing the capacity of the abdominal muscles to retain the several viscera in their normal position. The variation in the mechanisms of the abdomen in the sexes also accounts for the somewhat different behaviour of the same organs in men and in women.

As a result, then, chiefly of the habitual assumption of the erect posture, certain mechanical or evolutionary changes take place in our drainage scheme which result in a delay of the contents in part of the tract or throughout its whole length. I felt that the term "chronic intestinal stasis" describes most clearly the condition to which I wish to call attention.

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[1] *Trans. Obstet. Soc.*, Lond. (1887) 1888, xxix, pp. 351-68.

By chronic intestinal stasis I mean such an abnormal delay in the passage of the intestinal contents through a portion or portions of the gastro-intestinal tract as results in the absorption into the circulation of a greater quantity of poisonous or toxic material than can be treated effectually by the organs whose function it is to convert them into products as innocuous as possible to the tissues of the body. This condition can be best studied in those whose habit it is to stand for long periods of time. Butlers, footmen, waiters and those leading similar lives form excellent subjects for illustrating these changes.

Again, those who habitually make such overdrafts on their capital energy as result in temporary exhaustion readily develop these conditions. As one would expect, subjects handicapped by a feeble physique react more rapidly and extensively to the same mechanical factors than do the powerful and robust.

What I want to impress on you is the fact that in our usual state of civilization the trunk of the individual is erect, either in the standing or sedentary posture, from the time he gets up till he goes to bed, and while the trunk is erect, even for an instant, the tendency to the falling of the viscera from their normal position exists, and after a time in favourable circumstances that tendency must become an actuality. This tendency to drop is exaggerated by the results of improper feeding early in life. Even the usual attitude of defæcation is artificial and harmful.

An intimate knowledge of the manner in which the changes in the mechanism of the gastro-intestinal tract come about is of the greatest importance to the physician as well as to the surgeon.

A knowledge of the pathology of delayed or defective drainage of the intestinal tract and its consequences is of paramount importance, since in our state of civilization it affects vitally so many members of the community; indeed, some physical evidence of its existence can be demonstrated in almost any adult subject. This is what one would naturally expect, since we are all exposed in a varying degree to the causes which produce it.

After I had called attention to the mechanics of chronic intestinal stasis the accuracy of my statements was challenged by many. For instance, both the prolapse of the intestines and the acquired mesenteries and adhesions were absolutely and categorically denied by physicians experienced in post-mortem work, who appeared to think that, because they had escaped their observation like many things have done and will continue to do, they were justified in denying their existence in the most dogmatic manner possible.

The question having been raised, these changes have since been the subject of more careful investigation, and now it is asserted that they are so universally observed that there cannot be any association between them and intestinal stasis. This would all seem to suggest that the methods we adopt are rather crude and the reliance to be placed on them correspondingly small ; also that we are inclined to pursue detail rather than attempt to grasp general principles.

The chief difficulties which I have experienced in explaining the several changes which exist is that the presence of the adhesions described by me are regarded as evidence of inflammation. These adhesions, however, do not result from inflammation, but are developed to oppose the displacement of viscera, the tendency to which exists whenever the erect posture of the trunk is assumed.

Having regard to the changes referred to, I think that, however our opinions may differ as to the treatment called for in any particular case, there can be no doubt that the pathological changes which are present in these conditions of imperfect drainage are most obvious and important. Any mechanical interference with the functions of the gastro-intestinal tract is far-reaching in its capacity for harm, and once the mechanism of the intestines has deviated materially from the normal, such alterations, together with the evil results consequent upon them, can only be met by extensive surgical measures, and at a risk commensurate with the degeneration present.

In order that we may be in a position to discuss their treatment we should clearly understand their mechanics, as I have dealt with them fully. You may not accept my description of these changes as accurate, or, if you allow their existence, you may regard the explanation given of their causation as incorrect. Yet I am sure that when your attention is called to them, if you have not already done so, you will take the first opportunity of testing their accuracy in the living body, and will not merely be satisfied to say that you have not previously observed them and that therefore you doubt their existence. Their presence or absence is capable of absolute demonstration.

We will now consider the symptoms which result from the delay in part or in the whole of the gastro-intestinal tract in consequence of or in association with the conditions I have described. I will divide them into two groups—namely, those produced by obstruction and those resulting from auto-intoxication.

The symptoms produced by obstruction are those grouped roughly under the head of indigestion, a term used to cover a great deal of ignorance and much varied treatment. They are chiefly pain, tenderness, flatulence, eructation, &c. These result from an inability to transmit the intestinal contents at a normal rate and from the decomposition of the material consequent on the delay in its transit.

I do not propose to discuss the symptoms of the subsequent consequences of this distension, such as inflammatory changes in the mucous membrane or in the ducts and glands communicating with this tract, or of the last stage in the sequence—viz., cancerous infection—since they must necessarily vary with the prominence of the particular end result. The amount and degree of pain depend upon the character and locality of the obstruction, as does also the degree and extent of the flatulence. While the mechanical results of obstruction to the effluent in the drainage scheme are very important, they are trivial as compared to those brought about by the absorption of an abnormal amount of toxins into the circulation.

When these poisons or the products of their conversion exist in excess in the circulation they produce degenerative changes in every tissue and in every organ of the body. It is probable that the textures of those organs whose business it is to convert, carry, or eliminate them suffer more than do the other tissues of the body which are merely permeated by them. In proportion as the stasis is prolonged, so under the influence of a progressive strain greater than they are able to bear these several organs undergo a degeneration which proceeds with increasing rapidity as the condition advances. What the organs are which convert and excrete these poisons, what share each takes in the process, and how any organ is affected in its physiology are very difficult to define accurately, especially as far as the ductless glands are concerned. We believe that the liver is the most important converter of these poisons, and that the kidneys and skin are the chief excretors of the products of conversion.

As to the part taken by the thyroid gland, the pituitary body, and the suprarenals, we possess little precise information. Our observations in this direction are purely experimental, and if our several operations on the drainage scheme have done nothing more than demonstrate the damage done by these poisons, the remarkable power of repair which the several tissues of the body exhibit on being freed from their malign influence, and the extraordinary improvement in the functioning of the organs of the body after operation, they have been fully justified. They have, however, done more than this, since they have thrown a light on intestinal conditions which has simplified their treatment most materially, and has put them upon a definite mechanical basis, replacing what was little more than a nomenclature.

As corroborating the wisdom of attending to the drainage scheme of our bodies I would call your careful attention to some of Dr. Carrel's experiments, since they throw a most important and instructive light on the subject. When I saw Carrel more than two years ago he showed me living tissues growing in a sterile medium on a microscopical slide. At that date they had been growing for twelve days. I asked him what would result from the defective drainage of the tissues, since the quantity of serum was limited and the products of growth must be ejected into it. He said he did not know, but expected to learn shortly. After a few days he found that the vitality of the tissues became diminished, and that the growth of the cells was slowed and that it ceased very soon after. Death of the tissue took place obviously because of the imperfect drainage, just as our bodies and tissues become depreciated and finally die from chronic intestinal stasis. To meet this when growth commenced to flag, he opened up the specimens of growing tissue and washed away the toxic products, and by so doing he gave the material another period of growth extending over about fourteen days. This he was able to repeat many times before death finally took place. A line indicating the extent of growth was for a time level, then descended gradually, and finally very rapidly, illustrating exactly what takes place in chronic intestinal stasis in the human subject. By attending more carefully to the drainage of the toxic products of his specimens he was able to carry the growth along a horizontal line with regular undulations. Again, by feeding them with the juice of fœtal thryoid or spleen, he increased the growth of these tissues in an extraordinary manner, the increase during twenty-four hours varying between three and forty times the bulk of the original material. In this way he replaced the gradually descending line, representing the tissue growth ending in death, by one which is rapidly ascending, and shows no indication whatever of termination in death of the tissue. In other words, by attending carefully to the drainage and by giving suitable foods he gave that tissue a perpetual life and a rate of growth enormously in excess of the normal. He proved in an indisputable manner that decay is due to an inability of the tissues to eliminate waste products. Carrel's observations are of the greatest service at the present moment since they afford us the same confidence in our method of treatment that X-rays did in the case of operation for simple fracture when the opposition to that form of treatment was intense, and to a great extent unscrupulous in its method. I am relating this to show how very simple and yet how very important is the problem involved in the consideration of chronic intestinal stasis. I have recently seen him perform the same experiment on a larger scale. He eviscerated a cat and so arranged the drainage of the intestine that it continued to perform its function normally during artificial respiration for a period of about ten hours. He hopes by preventing the death of the organs by peritonitis to prolong their life for a much longer period. The improvement in the growth of tissue which he obtains by attending very carefully to the drainage is well illustrated in the great and very rapid increase in the growth and weight of the child suffering from tubercle or rheumatoid arthritis which takes place after the large bowel has been excluded from the gastro-intestinal tract.

I cannot illustrate more dramatically the condition of excessive growth following on improved drainage than by describing a case of rheumatoid arthritis in a young girl. Her colon was disconnected from the gastro-intestinal tract by an ileo-colostomy below the last kink.

A female, aged 10½, a helpless cripple suffering from rapidly progressive rheumatoid arthritis in spite of careful and constant treatment. She was short-circuited in November, 1911. This chart shows her weight before the operation to be 49 lb. She lost 2½ lb. during the fortnight following the operation, her weight on November 13 being 46½ lb. On October 1, 1912, thirteen months after the operation, her weight was 87 lb., so that between November 13, 1911, and December 24, 1912, her weight had increased by 40½ lb., or in other words, in thirteen months she had nearly doubled her original weight. On consulting Dr. Still's work on "The Common Disorders and Diseases of Childhood," he puts the normal increase in weight at this age at 6 lb. in twelve months. This shows that the improvement in her drainage scheme had produced an increase of more than six times the normal. Besides this abnormal increase in the growth of the child her disease was stopped abruptly and she is now a vigorous, active and healthy child. She has been shown to the Medical Society by Mr. Barrington Ward, who described her case (*see* fig. 49).

The magnificent work that Carrel has done in the Rockefeller Institute has confirmed my views on the importance of chronic intestinal stasis in the most complete manner possible. The results of his experiments on the growth of tissues are perfectly obvious and indisputable. In reference to this I here quote a few lines from the *Scientific American*, October 26, 1912, on "The Immortality of Tissues," by Genevieve Grandcourt: "It has now become evident that, under the right circumstances, the artificial growth of tissues can be utilized in the study of many problems, such as malignant

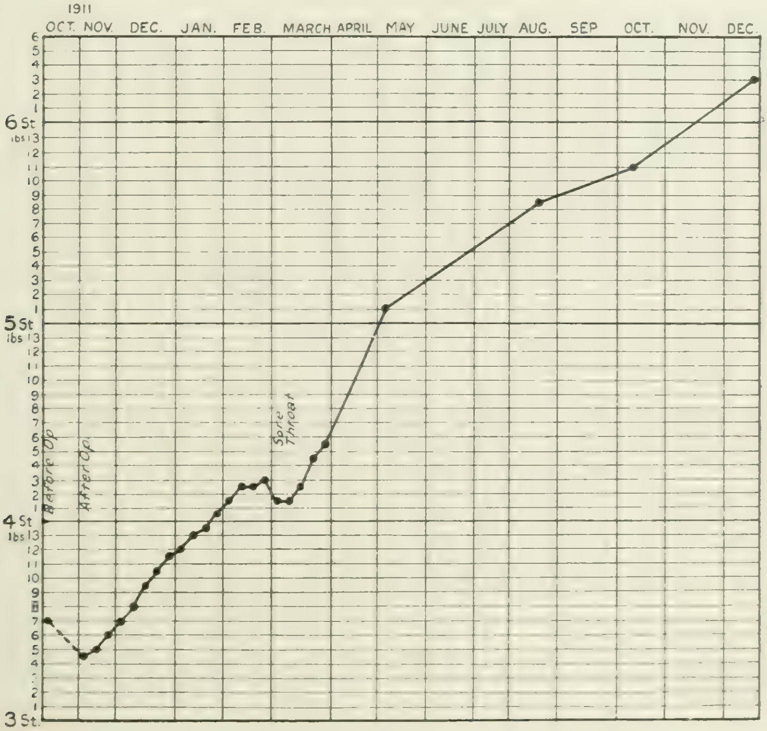

FIG. 49.

growth of tissue, certain problems in immunity, as, for example, the production of antibodies and antitoxins of certain organisms; the reintegration of tissues, the regulation of the growth of the organism; rejuvenation and senility, and the character of the internal secretions of the glands, such as the thryoid, which plays a rôle most important in physical and mental development."

The questions to which Carrel will apply himself to investigate by these methods will certainly modify our views on the treatment of disease very greatly.

Professor Arthur Keith has done a great deal of original work on the functions of the large bowel. The following quotation from a lecture delivered at the Royal College of Surgeons and published in the *British Medical Journal*, December 7, 1912,[1] has an important bearing here. The title of the paper is "The Functional Nature of the Cæcum and Appendix":—

> Every year the opinion gains ground that the great bowel, from appendix to rectum, has become, so far as man is concerned, a useless and dangerous structure. Exactly ten years ago (October, 1902) Dr. Barclay Smith, of Cambridge, gave a clear expression of this new conception.
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> > "If there is any truth," he wrote, "in the suggestions offered in this paper, they have an important practical application as regards the large intestine of man. From the nature of his diet a reliance on extrinsic digestive aid as is furnished by bacteria is no longer a physiological necessity. The statement is perhaps a bold one, but I am convinced that the large intestine is practically a useless encumbrance to him."
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> In the following year (1903) appeared Metchnikoff's famous book on "The Nature of Man." In that work (p. 69) the new conception received a more decisive statement:—
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> > "It is no longer rash to say that not only the rudimentary appendix and the cæcum, but the whole of the large intestine are superfluous, and that their removal would be attended with happy results."
>
> Before either of these statements had been made, Mr. Arbuthnot Lane had reached the conclusion that the human cæcum and the ascending colon served, in a certain class of cases described by him, as a "cesspool," and put his new conception into practice either by excluding the great intestine from the digestive tract by "short-circuiting," or, at a later date, by its complete excision. The result of his operations shows (1) that life is possible without a great intestine; (2) that in certain cases the conditions of life are improved. It is very apparent that Mr. Lane's pioneer operative measures are finding every year an increasingly wide application, and that the views of Metchnikoff and of Barclay Smith are gaining in acceptation amongst medical men.

I will now consider the effect on the several organs and tissues of the body of an excess of toxins or poisonous products in the circulation. Perhaps the most conspicuous result is the removal of fat. This brings about not only an appearance of premature senility, but also a series of changes of infinite importance to the individual, and especially to the female, in whom, for reasons I have already indicated, fat plays a far larger share in supporting important organs and structures than it does in the male. The changes in the position of the several organs which follow on the loss of fat serve to exaggerate the existing stasis in the gastro-intestinal tract and to produce a vicious circle. The removal of the pelvic fat results usually in a backward displacement of the fundus of the uterus, which rests upon the concavity of the rectum. When the woman strains to evacuate the contents of the rectum she drives the large gorged fundus vertically downwards and the rectal lumen is compressed between it and the sacrum, or the uterus may be bent forwards, partly on account of the loss of fat and to a large extent because of the degeneration of its muscular wall which exists generally throughout the body. Consequent on the engorgement and the displacement or kinking of the uterus, a number of changes take place in that organ which call for the attention of the gynæcologist. As we shall see, auto-intoxication plays so large a part in the development of diseases of the female genito-urinary apparatus that the gynæcologist may also be regarded as a product of intestinal stasis. If women were not imperfectly drained the gynæcologist would not have been evolved. The removal of fat from the true pelvis permits of the greater descent of the cæcum and small intestines into that cavity and exaggerates the obstruction of the ileal effluent correspondingly. The kidneys move freely in the space behind the peritoneum in which they were originally supported by a cushion of fat and changes ensue in these organs should the escape of blood or urine from them be controlled by their altered relationship to adjacent structures.

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[1] *Brit. Med. Journ.*, 1912, ii, p. 1599.

As regards the attractiveness of the woman, a matter of vital importance to her happiness, the loss of fat is a most serious factor. The formation of wrinkles, the prominence of bones, &c., are all most distressing and conspicuous features. The buttocks also become flat and flaccid, instead of firm and round, partly because of the disappearance of fat which enters so largely into their formation and partly because of the associated degeneration of the large gluteal muscles. The breasts also waste and flop downwards, and the whole form and contour of the woman alters conspicuously in the most objectionable manner. The skin undergoes remarkable changes. It becomes thin, inelastic, sticky, and pigmented, especially where it is exposed to any pressure or friction. This pigmentation is observed first in the eyelids, whence it spreads gradually over the face. The neck becomes brown and later almost chocolate-coloured. The skin of the axillæ, abdomen, adjacent aspects of the thighs, and that covering the spinous processes of the vertebræ, becomes progressively darker and darker, and defined areas of even darker pigmentation may develop on these stained surfaces. The secretion from the flexures also becomes abundant and offensive. In some of the cases I have operated on this symptom has been such a marked feature as to render the patient very objectionable to others.

Here I would like to call attention to the extraordinary variation in the resisting power of the individual as manifested by the colour of the hair, not only to the changes in the fat and colour of the skin already referred to, but to all other consequences of auto-intoxication. The darker the hair the lower is the resisting power to auto-intoxication and the more conspicuous are the changes which result from it. On the other hand, if the hair is red or of a peculiar towy colour the individual has a maximum of resisting power to the action of these poisons, and that resisting power varies directly with the distribution and with the intensity of the redness of the hair. This is manifested very conspicuously in the influence exerted by the toxins on the appetites of the individual. The darker-haired subject will loathe the sight of food and frequently abhor any sexual relationship, while the red-haired subject rarely manifests these effects, even in the extreme conditions of intestinal stasis. This influence of toxins on the normal appetite is of far-reaching importance in our present state of civilization and is the source of much misery, discontent, and trouble, to which I will not do more than allude here.

Some time ago I called attention to the influence prostatic secretion exerts in combating the effects of auto-intoxication. I pointed this out to Metchnikoff, who thoroughly agreed with me on the subject, and afforded me additional confirmation of its truth by the results of experiments on the action of prostatic fluid upon spermatozoa. To attempt to meet this condition Messrs. Armour make in their works in Chicago "tabloids" of extract obtained from the prostates of rams, and these have been administered very generally and apparently with marked benefit.

Other changes in the skin are those dependent on a damping down of the heart's muscle by the toxins, and upon a breaking up of the red corpuscles, which is called in the most marked condition "microbic cyanosis." The limbs become very cold and this coldness becomes exaggerated as the extremities are approached. If the hand be passed over the shoulder it crosses abruptly from an area of warmth to one of comparative coldness. This corresponds to a line drawn transversely round the centre of the deltoid. The skin of the back of the upper arm is very thick and feels as if it were affected by a firm, brawny œdema. Its colour is bluish, and in some cases even livid. It is liable to be covered with hard, pointed papules. This condition exists to a much more marked degree in the girl than in the woman, and may be sufficiently conspicuous to render the wearing of short sleeves impossible. The skin of the forearm and hand is mottled, being bluish and yellow in patches, while the fingers may be quite blue or cyanotic. The legs present the same condition and usually in a greater degree. The patient frequently complains that she has no feeling up to her knees even in moderately warm weather. These people are better in warm weather and in fairly high altitudes, and are always worse in the cold, and by the sea. In some cases the condition was so marked as to have been called Raynaud's disease by experienced observers.

The muscular system degenerates in a very marked manner. The muscles waste and become soft, and in advanced cases tear with the greatest facility. In consequence the individual assumes positions of rest. In young life the muscular debility produces the deformities which are called dorsal excurvation or round shoulders, but which are more scientifically described as the "symmetrical posture of rest of the trunk," lateral curvature or scoliosis, which is better designated as the "asymmetrical posture of rest of the trunk," flat-foot, and knock-knee. These conditions are still further exaggerated by pressure changes in the epiphyseal lines. To subject these cases to exercises or to fix them in apparatus without also removing the primary factor in their causation—namely, auto-intoxication—is of little service.

The relaxation of the muscle wall of the abdomen deprives it of its function of compressing the viscera efficiently in defæcation and results in the accumulation of fæcal matter in the pelvic colon. This elongates proportionately and renders the evacuation of its contents more and more difficult. The abdominal muscles cease to exert upon the several viscera that firm pressure which is requisite to keep them in their normal relationship to the spine and to each other, and the errors in drainage become further accentuated. The normal mechanical disadvantages of the female abdomen render these changes much more conspicuous than in the male subject. As I have already pointed out, the uterus suffers in the same manner, so that it flops or bends about in response to gravity and intra-abdominal pressure, and much trouble in it and in other structures ensues in consequence.

The muscular wall of the intestine wastes in a similar manner, so that in an advanced case of stasis the ileal wall is very thin and bluish or livid in colour, resembling the appearances seen at a post-mortem, and they give out a distinctly earthy or fæcal odour. The intestine has no rounded form, but being inelastic puddles like jelly in the floor of the true pelvis, forming innumerable bends through which its contents are transmitted with great difficulty.

The heart-muscle is influenced by the poison in the same manner. Here, however, we get two distinct conditions arising, varying, I believe, with complications in the most important excretory organ—the kidney, as well as in the circulatory system itself. In one group of cases the heart is soft, flabby, and the blood-pressure subnormal; while in the other the left heart is definitely enlarged, the aorta dilated and its walls atheromatous, as are those of all the vessels, and the blood-pressure is abnormally high. I am greatly indebted to Dr. Jordan for demonstrating most clearly to me the changes in the heart and aorta in this class of case. Generally speaking, the soft heart and low blood-pressure are more common in the female subject, while the enlarged heart and high blood-pressure are more frequently observed in the male. Inflammatory or degenerative changes in the kidney are much more commonly associated with the second group than with the first.

If, unfortunately, the patient is the subject of syphilis, the combination of this disease with auto-intoxication produces an extreme degree of change in the wall of the large vessels, and especially in those of the trunk.

Dr. James Mackenzie has recognized and described the heart changes consequent on auto-intoxication so thoroughly in his work on the subject that any further reference to them by me is unnecessary. I would merely add that the normal degree of blood-pressure is soon restored to the patient when the large bowel is excluded by operation.

The toxins appear to exert a special depreciating influence upon the respiratory centre. How far this is produced in the nervous centre, and how far it results from a degeneration of the respiratory muscles, is very difficult to determine. Dr. Vaughan and Miss Wheeler[1] assert that proteids can be split into a poisonous and non-poisonous portion, and they are of opinion that the deleterious component is a respiratory poison. It can produce acute, subacute, or chronic poisoning, depending on the manner and the rate of its introduction into the body.

The toxins exert upon the nervous system a most distressing and depressing effect. This is perhaps the worst feature of the effects of chronic intestinal stasis. The patient is usually miserable. Sometimes the depression and misery is so great as to constitute melancholia, or imbecility. Several of my patients have contemplated suicide and have used the intention to do so to induce me to operate on them. Headache, varying in character, is very frequent. It may be a dull ache that appears on awakening, or it may be an intense headache accompanied with vomiting. I removed the large bowel in a patient who had been regarded as insane by some medical men and in whom one of our ablest brain specialists diagnosed a tumour in the frontal lobe and urged operation. This was fortunately refused by the patient. This headache disappeared at once when the large bowel was excluded from the drainage scheme.

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[1] "The Effects of Egg-white and its Split Products on Animals," *Journ. of Infect. Dis.*, Chicago, 1907, iv, pp. 476-508.

I had under my care a girl who had been confined to bed for months. She was unable to stand, and was so stupid that she appeared to be an imbecile. The removal of the large bowel was followed by a very rapid and complete return to health. She is now perfectly intelligent and is earning her living. Another case of epileptiform tic of the right fifth nerve which had existed for eight years and which had rendered the patient's life unendurable, was cured by excluding the large bowel. These represent the more serious types of headache, neuralgia, or neuritis, but lesser degrees are extremely common in these patients and are cured by removing the auto-intoxication which would appear to produce them.

The favourite term for these cases of auto-intoxication in which the nervous system suffers in a marked manner is neurasthenia. They used to be treated by rest in the supine position, massage, and feeding. By all these means the effluent from the ileum was improved and fat was put on. The patient derived a fleeting or more or less permanent benefit from the treatment, which varied with the extent of the infection of the dammed-back contents of the ileum. Toxic patients sleep badly and have unpleasant dreams, or they may sleep very soundly all through the night and frequently fall asleep at any time of the day. They usually awake feeling miserable and unrefreshed by their sleep.

The breast behaves in a characteristic manner in auto-intoxication, so much so that it may be regarded as the barometer of the degree of poisoning. It presents at first induration, which commences in the upper and outer zone of the left breast, extending subsequently to the entire organ on both sides. Cystic or other degenerative change may ensue, and at a later period cancer appears with remarkable frequency in these damaged organs. I have found as many as seven distinct nodules of cancer in a hard, lumpy breast in which the presence of that disease was not suspected. One of the most remarkable results of removing the source of auto-intoxication is the rapidity with which even extreme degrees of degeneration of the breast disappear, and the organ regains its normal form and texture after operation.

Perhaps the organ which surprised me most by its behaviour was the thyroid, for though I had long recognized the dependence of breast conditions on stasis I had obtained no certain evidence that the several changes that the thyroid undergoes were produced by it also. I had short-circuited patients who were clearly exophthalmic, or who had been so most distinctly at an antecedent period and were improving, and had not been altogether surprised at the rapid and complete disappearance of their symptoms, as one knows they may occasionally subside, or even disappear, as it is called, spontaneously. A patient was admitted under my care with marked stasis who had been driven to resort to operation by abdominal pain and vomiting, and by the associated mental and physical misery and depreciation. For eight years she had suffered from an enlarged thyroid which projected forwards in her neck and which interfered with respiration. It contained several large adenomata. As the anæsthetist hesitated to administer an anæsthetic for resection of the large bowel because of the goitre, I urged her to consent to the removal of part of her thyroid in the first instance. This she fortunately refused to submit to, saying she would have her bowel removed or go out of the hospital. Consequently this was done without any serious cause for anxiety. Within a few days of the operation it was obvious that the thyroid was diminishing steadily in size, and this diminution continued till when she left the hospital it was but little larger than normal. I understand that at the present time it is not larger than normal. When this was related to Metchnikoff, he exclaimed, "C'est un cas qui coûte," and his rapid grasp of the importance of this result has been fully borne out by subsequent operations on similar cases accompanied or not by exophthalmic symptoms.

Another most important consequence of auto-intoxication is the lowering of the resisting power of the tissues to the entry of organisms. There are organisms which exist in large quantities in our drainage scheme, and which are only able to secure a foothold in our tissues when their capacity to deal with these organisms has been reduced. By far the most common cause of this lowered resisting power is auto-intoxication, resulting from intestinal stasis. One sees that from the earliest period. The resisting power of the mucous membrane being lowered allows of its infection by organisms which extend or involve the lymphatic tissue in the nasopharynx, producing adenoids and large tonsils, and later changes in the ear and nasal sinuses. These secondary manifestations of stasis produce cumulative symptoms and add their share to the depreciation of the vitality of the tissues.

In these toxic people the organisms which exist normally in the mouth are able to secure a foothold in the interval between the teeth and gums and manifest their presence as an infective or inflammatory process. This extends deeper and deeper into the socket and results in the loss of the affected teeth. This process usually commences, as one would expect it to do, about the lower incisors. As a quantity of organisms is grown in these nurseries the absorption of their products into the circulation, gastro-intestinal tract, and respiratory apparatus all assist in the general process of depreciation. Many observers are inclined to regard these secondary foci as primary, since a marked improvement in the general resisting power may result from their effective treatment. A little consideration shows the absurdity of this supposition. In a considerable proportion of the patients who are driven to operation by the results of intestinal stasis, all, or almost all, the teeth have been previously removed for this reason. The removal of the teeth has not relieved the symptoms of the stasis, which was the primary factor. Again, however bad the condition of infection of the gums may be at the time of short-circuiting, an immediate and extraordinary improvement in this disease follows on the cleaning up of the small intestine.

In women who are toxic the infection of the genital tract is an extremely common and distressing feature. It manifests itself usually as an infection of the mucous membrane or the muscle of the uterus, and is called endometritis or metritis. The continued presence of this infection brings about many troublesome consequences, the last link in the chain being here, as elsewhere in the body, cancer. It is the last chapter in the three-volume story of "Chronic Intestinal Stasis." The bladder of the toxic woman is also very often infected by organisms. Fortunately, the extension of organisms from the bladder to the kidneys is not as frequent as one would expect, yet it is quite common enough to produce much disease of these organs. The urinary apparatus in the male is also affected, especially in early life.

I do not think I need call the attention of the obstetricians to the important part played by pregnancy in relieving the disabilities of the drainage scheme. The ascent of the uterus in the abdomen serves to raise the portions of the drainage scheme which have prolapsed, to tend to stretch retaining bands, and to improve materially the effluent from the ileum, &c. Consequent on this the woman puts on fat, and pillows up the several organs, and tends to obviate their subsequent prolapse. It is in this manner that a toxic, thin, miserable girl may be converted into a plump, clean, happy one by a pregnancy.

Perhaps one of the most interesting efforts of Nature to obtain similar advantages is by the formation of an ovarian tumour. This, as is true of all evolutionary processes, tends to benefit the individual in the first instance, but destroys her later by interfering with the functions of other organs. I have already shown how the bands or membrane forming the last kink secures the left ovary and causes it to become cystic. This forms another illustration of the third law I have referred to earlier in the paper.

The eye also shows very definite changes in auto-intoxication. The condition of the conjunctiva is very characteristic. The sclerotic appears dull and opaque and lacking in brightness, and if the lid be depressed the reflection of the conjunctiva shows what appears to be a quantity of fluid in its texture. The cornea shows degenerative changes around its margin, and the lens and muscles of accommodation suffer in a degree corresponding to that of the stasis. I believe that many of the degenerative changes in the eye are due to auto-intoxication. All these conditions improve at once, and obviously, after the intestines have been cleaned and freed from stasis.

The hair of the head falls out, either because of impaired nutrition of the cells or from the invasion of the roots by organisms. In the young subject, associated with this there is a new growth of a fine down over the cheeks, lip, chin, down the back and over the forearms, all of which conditions are very disfiguring and very distressing to the sufferer. These all disappear, more or less completely, with an improvement in the drainage.

The joints of toxic people, and especially of children, are very loose, and permit of considerable over-extension. We know that the fit or security of a joint varies directly with the development of the muscles which control it, and the feebleness of the poisoned muscles readily accounts for the insecurity of the joints. The skin is very liable to invasion by organisms, which form pustules, &c.

There are many diseases which cannot attack a subject unless the vitality of the tissues have not been depreciated by auto-intoxication. There is no limit to the number of diseases in which stasis affords the chief, if not the entire, factor in their causation. I will confine myself to three to illustrate this point, as they lend themselves so readily and so obviously to demonstration. They are tubercle, rheumatoid arthritis, and Still's disease. I do not believe it is possible for either of these diseases to obtain a foothold except in the presence of stasis. I would exclude the direct inoculation of tubercular organisms through a wound, but this is a very uncommon mode of infection.

In all our cases of these diseases we have carefully examined the condition of the patient clinically, both generally and by manipulation of the abdomen, for evidences of stasis, and we have confirmed this by bismuth meals and X-ray observations. We have been able to satisfy ourselves that in these diseases stasis, and the auto-intoxication consequent upon it, is a necessary antecedent factor in their development. We have also been able to demonstrate that the effectual drainage of the patient is followed by a complete cure of the condition, except in such cases of tubercle where infection other than that of tubercle is superadded. The degree of improvement which follows an operation on the drainage scheme in tuberculosis varies with the purity of the infection.

The rheumatoidal cases show the most abrupt and conspicuous change following within a few days, and in some cases even hours, an ileo-colostomy. I have seen patients who have been unable to move their joints for months or years acquire a free movement within four days of the operation. Even more rapid is the abrupt disappearance of pain. The striking and rapid and, to my mind, wonderful alteration that follows in rheumatoid arthritis is familiar to any surgeon who has performed this operation, or who has observed cases after operation. It is even more striking than the change in the temperature and feel of the extremities, the sudden disappearance of headache and misery, the change in the colour of the skin and in the nature of its secretion, the alteration in the texture and feel of the thyroid and breast, &c., since the structures which enter into the formation of a joint are much denser and less vascular, and one would expect them to be less liable to undergo rapid change. I will quote from the letter of a well-known and energetic surgeon the following details, since it affords an independent view of the condition :—

February 15, 1913.

Since I saw you operate I have operated on a poor woman concerning whom I was in despair until you showed me your cases in the hospital. A case of rheumatoid arthritis was in this hospital for weeks and weeks under a colleague. Deriving no benefit, she was sent to Droitwich and returned worse. Some three months ago she could scarcely crawl into the out-patient room because of the pain in her ankles. Hearing her history I tried to see if I could find a source of poisoning in any region. Discovering much discharge from the uterus, and a very unhealthy os uteri, I curetted her and used the actual cautery to the os. Then I injected her with anti-streptococcic serum. She was very ill for a time in consequence. Then she remained a chronic invalid with knees that would neither bend nor straighten. A bismuth meal showed marked stasis. I therefore performed your operation, and next morning she said: "I can move my legs anywhere without any pain in my knees." In fact, she eats and drinks all she can get, although the operation was only done a week ago.

I could furnish you with the records of many cases of tubercle and rheumatoid arthritis, should you care for them. I would refer to cases published by Mr. Harold Chapple, Mr. Barrington Ward, and Mr. Schlesinger.

There are one or two points of interest in relation to abdominal tubercle which would appear to show that the observations of the physiologists as made upon animals do not give us a very correct idea of the possibilities in the human subject. In evidence of this the following report throws some light. I removed the large intestine of a girl affected with extreme stasis. This portion of the bowel was much ulcerated and it perforated at several points while it was being removed. She progressed perfectly for nearly four weeks, when a fistulous opening appeared in the middle line through which a portion and then all the contents of the bowel escaped. She was too feeble to make any further operation at the time advisable, so we fed her with very nutritious foods and watched her. She steadily but slowly increased in weight during the two years following on the operation. During this time she led an active life and met the discomfort of the fistulous opening by clever little dodges which she herself devised. We gave her bismuth and examined her tract and found that not more than 18 in. of jejunum were functioning. At the end of this time she wished the opening closed. We therefore opened the abdomen, confirmed the length of small intestine which had been sufficient to meet her daily requirements of energy and also to add to her weight, and we established continuity of her small intestine.

Having learnt what a small portion of the small intestine is requisite to carry on life, I have not hesitated to remove the large bowel in cases in which the glands of the small intestine were matted up with a large caseous mass, and I have had no reason to regret doing so. In tubercle of the abdomen more than elsewhere does rapid disappearance of the disease follow disconnexion of the large bowel by ileo-colostomy with or without colectomy.

I will relate briefly the details of a typical case of Still's disease in a boy, aged 6½, showing glandular and splenic changes, stiffness in the neck with painful and tender joints which showed no bony changes. There was fluid in the joints. He was unable to walk. He had been ill for two years, during which time he was treated at the Evelina Hospital. He was extremely wasted, and appeared to have an adherent pericardium. Dr. Still saw the case and confirmed the diagnosis, and approved of operative interference as offering him the only chance of recovery. The accompanying temperature chart (fig. 50) shows his condition from his admission on August 28, 1912, till the date of operation, September 24, 1912. Several rises of temperature took place with corresponding changes in the pulse and respiration. In these attacks the child was very ill, and his blood gave a pure culture of *Staphylococcus citreus*. He showed distinct clinical and X-ray evidence of chronic intestinal stasis. After the operation of short-circuiting the child had no subsequent rise of temperature. This fluctuated below normal till October 4 or 5, 1912, and then continued uninterruptedly at a normal level. At the operation the end of the ileum presented a well-marked ileal kink. The mesentery contained a great number of large glands. The pelvic colon was very long, a condition we have observed to be constantly present in tubercular and rheumatoidal affections in young life. A swab taken from the contents of the ileum gave a culture of *Staphylococcus citreus*. The general condition of the patient and the size and mobility of the joints improved rapidly almost immediately after the operation, and within a few weeks the child was able to play about the ward, where he remained till January 8, 1913. Since that date he has improved steadily, both as regards his joints and his general health.

I do not propose to consider in detail such complications of intestinal stasis as membranous and ulcerative colitis and other consequences of superadded infection of the large or small bowel, as we are very familiar with their symptoms. Nor do I intend to supplement the evidence I have put forward by skiagrams showing the results of the administration of bismuth meals, since that has been done so well by my colleague Dr. Jordan. I might point out that his great success is largely due to the fact that he has not been satisfied to diagnose, or in other words to guess at the condition of the patient's drainage scheme by an examination of X-ray results only, but has made a habit of being present at operations. By seeing the changes in the tract, he has been able to examine his own work critically, and by correlating these facts

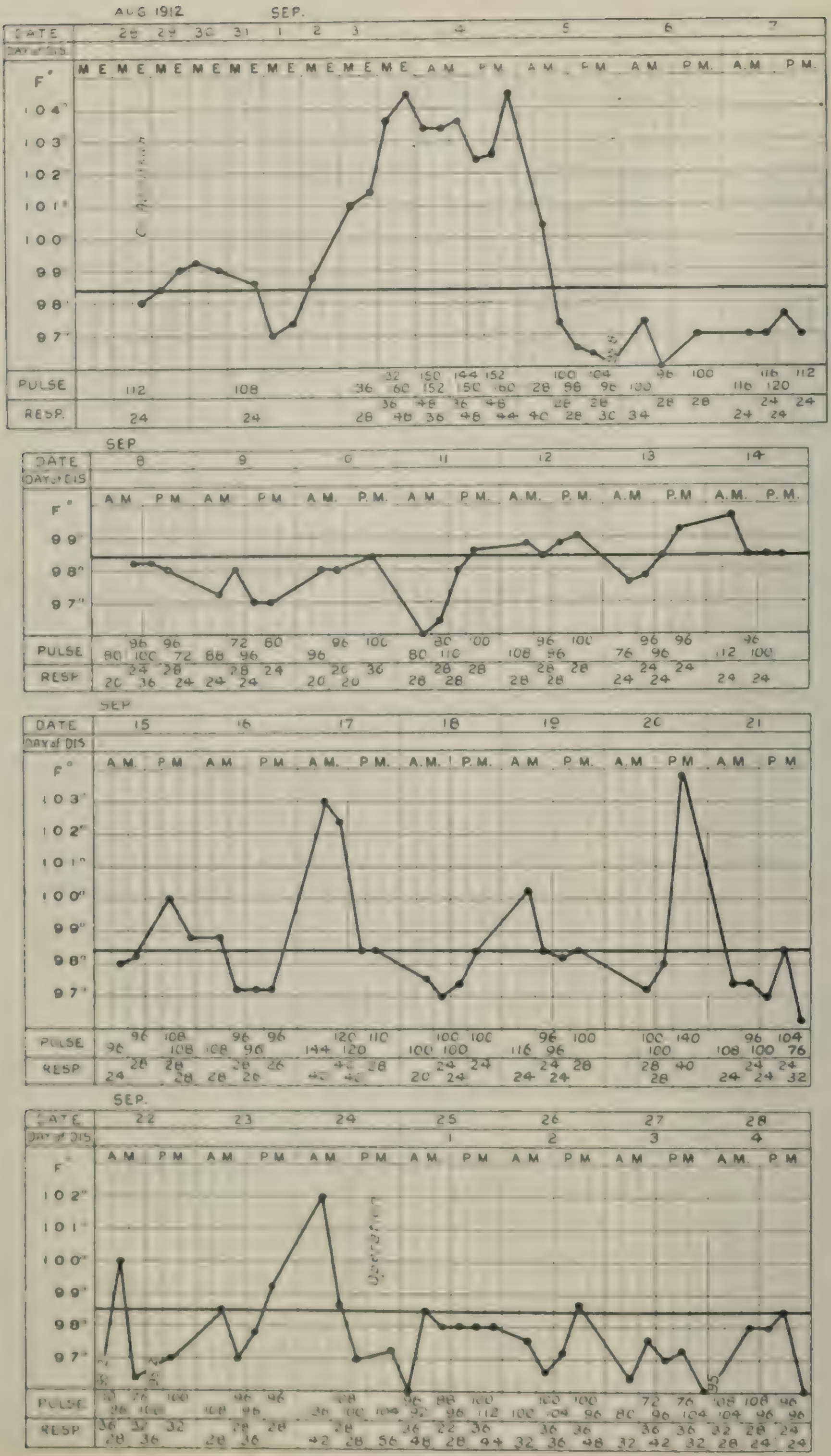
AUG 1912
SEP.
DATE
DAY of DIS
F°
104°
103
102
101°
100
99
98°
97°
PULSE
RESP.
28 29 30 31 1 2 3 4 5 6 7
M E M E M E M E M E M E M E M E
A.M. P.M.
SEP
8 9 10 11 12 13 14
15 16 17 18 19 20 21
22 23 24 25 26 27 28
1 2 3 4
Operation

Fig. 50.

to obtain better results. The reason why so many fail in what is euphoniously called diagnosis is that they rarely see the inside of a living patient's abdomen.

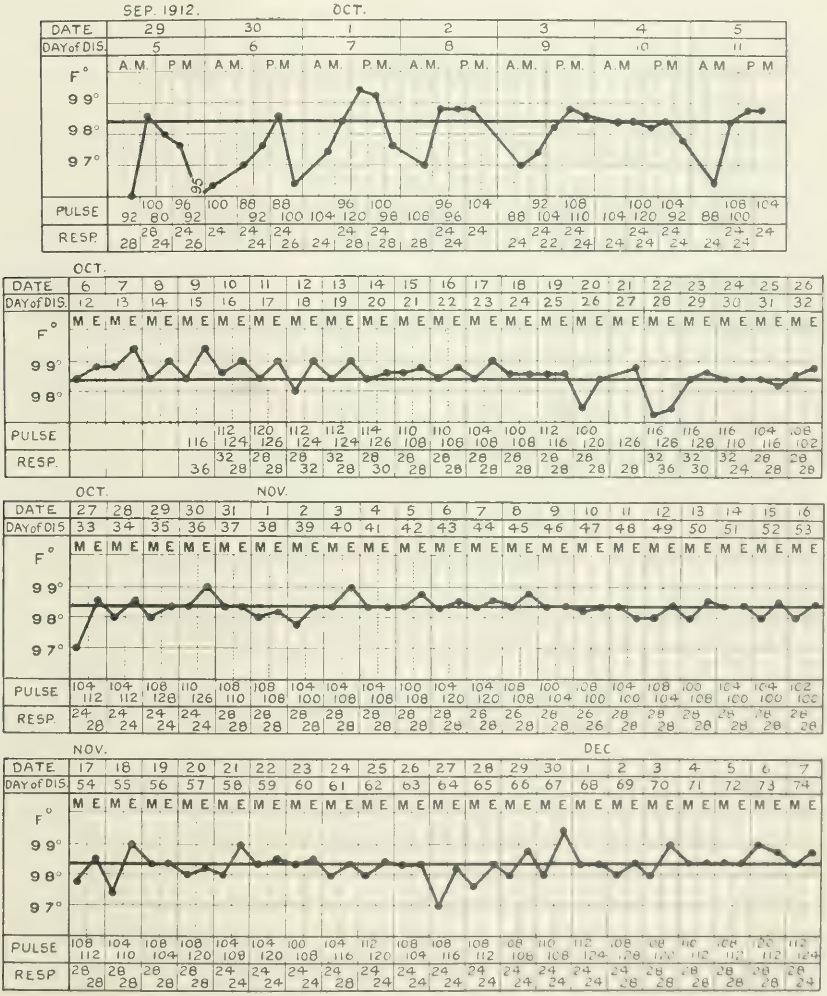

FIG. 50 (*continued*).

I take it that before submitting a patient to any surgical risk whatever it is our duty to employ every scientific means at our disposal to determine exactly what is wrong, remembering that a so-called exploratory operation on the intestinal tract is of little value, since it is performed on a drainage scheme which has been previously thoroughly emptied of its contents by means of powerful purgative drugs and enemata. That is the reason why an exploratory operation only too often ends in the removal of an anchored appendix, a result that appears to satisfy the ambition of the surgeon, and the curiosity of the patient and the friends. I trust that this phase of surgery is rapidly passing away, together with the obscurity that was associated with the medical treatment of the results of chronic intestinal stasis.

From the surgeon's point of view, the treatment of chronic intestinal stasis consists in facilitating the passage of material through the several portions of the gastro-intestinal tract, and so obviating the mechanical and chemical results of any fault or faults which may develop along its length consequent on the peculiar mechanical relationship of the individual to surroundings as involved in the complex conditions of the civilization of the present day. In the vast majority of cases the use of a lubricating material such as pure paraffin, which precedes the passage of food, application of some spring support to the lower abdomen which tends to keep the viscera up, and to control the delay of material in the small intestines and cæcum, and the avoidance of the use of such proteid foods as poison the tissues if retained for an abnormally long time in the intestine, are sufficient for the purpose. When these methods fail, resort must be had to operative interference. The object of such operative treatment is usually to facilitate the effluent from the ileum, and so to remove at once from the area of the drainage scheme, from which toxins are chiefly absorbed, the filth which supplied them.

As I have pointed out, the measures to be undertaken to bring this about depend entirely on the nature of the mechanical conditions which produce the stasis, and also on the state of the patient at the time. For instance, where the ileal effluent is controlled by an appendix which is hitched up behind the termination of the small bowel, the removal of the constricting band frees the lumen of the small intestine and restores it to its normal function more or less completely. At the same time, if in consequence of the stasis there are present marked rheumatoidal changes or tubercular infection, the emptying of the contents of the altered and dilated small intestine into the cæcum may not result in such an effectual clearing of the small intestine as will afford the sufferer sufficient spare energy to destroy the disease. In these circumstances, the only effectual way of bringing this about is by dividing the small bowel and introducing its extremity into the pelvic colon.

The same applies with much greater force to the control of the ileal effluent by the acquired membrane which produces the ileal kink. This condition of obstruction is much more serious than that which is brought about by the appendix, since it is very liable to recur whatever means are adopted to obviate its recurrence. Obviously, such a recurrence cannot arise in the case of the appendix. Also, for some reason or other, the free division of these bands and membranes occasionally results in the production of a peritonitis which may cause serious anxiety. Consequently, when the ileal kink is produced by any extensive arrangement of acquired bands, especially in the female subject, I prefer to short-circuit rather than merely divide the constricting bands. I believe that in many cases the risks of short-circuiting are less than those of division, while the possibility of recurrence by the re-formation of these bands is permanently removed. The necessity of following the same occupation which determined the obstruction originally in the case of the man makes such a procedure all the more advisable. The convalescence after the short-circuit is also much more satisfactory than that which follows the division of bands.

If tubercle, rheumatoid arthritis, or any advanced condition of auto-intoxication be present, there is to my mind no doubt whatever as to the value of short-circuiting the patient. The more I see of these cases and of the excellent and far-reaching results that follow on this procedure, the more inclined I am to adopt it, rather than to effect what is frequently an unsatisfactory and temporary compromise. I believe that the operation that I call short-circuiting, which consists in the introduction of the ileum directly into the upper part of the pelvic colon, has not succeeded so generally as one would wish because the details of the operation have been imperfectly carried out. I have known many complaints of pain and diarrhœa after such an operation. This unfavourable result is frequently due to the fact that the surgeon has been satisfied with establishing the anastomosis alone, and has not closed in the interval between the mesentery of the ileum and that of the pelvic colon. The intestines have fallen down behind the junction till the termination of the ileum has done so also. This has resulted in a permanent or recurring torsion of the end of the ileum on its own axis, producing a varying degree of obstruction of its lumen and consequent pain and diarrhœa, these being due to incontinence caused by over-distension of the bowel behind the obstruction. The chief disadvantage of short-circuiting is the occasional tendency which exists for the material which passes from the ileum into the pelvic colon, if not evacuated very shortly, to ascend into the iliac colon. This can be met in the large majority of cases by the fixation of the colon to the posterior abdominal wall at the pelvic brim by exaggerating the last kink, should this kink be not already efficient. If this is done effectually the chances of material passing upwards into the large bowel are very much diminished. As I have already pointed out, one of the purposes of the formation of the last kink would appear to be to obviate the ascent of fæcal matter from the pelvic colon in normal conditions, should the contents not be evacuated at the proper time. The presence of a quantity of fæcal matter in the pelvic colon is inconvenient to the functioning of the other pelvic organs, so that if it is not expelled it is displaced upwards into a more commodious area for the time being. If the last kink be well developed this is obviated and the material continues to set up a reflex which leads to its being forcibly expelled. In a certain proportion of cases it is advisable to remove the large bowel also. This may be required by the ascent of fæcal matter after an ileo-colostomy, or it may be called for if the colon be much dilated, and especially if its mucous membrane has been chronically inflamed. In such a condition as is called Hirschsprung's disease, the removal of the large bowel is generally required and the distension of this portion of the intestine facilitates its removal. It is not easy to draw a sharp line between the cases of stasis in which it is advisable to do a short-circuiting only and those in which the large bowel may be removed as well with advantage. If the patient is wasted and the abdominal wall is loose and flaccid, especially if tubercle or rheumatoid arthritis or other result of stasis are present, I much prefer to remove the large bowel at the same time as I short-circuit, but if I feel I can save the patient any risk whatever by doing the operation in two stages, I prefer to do so.

The only risk of short-circuiting or removal of the large bowel which is associated with the operation itself is the formation of adhesions. Against this there are apparently no certain means of prevention. We have tried many things applied to the intestine and have fancied that we have obtained better results from the use of silk cloths soaked in paraffin than with any other application. We are still in great doubt as to whether the paraffin is of any use, but feel pretty sure that the best way to prevent the formation of adhesions is to avoid any unnecessary exposure of or damage to the intestines in the performance of the requisite manipulations and to be most careful in one's aseptic precautions. To me, the formation of adhesions and their subsequent behaviour is somewhat of a mystery, except in so far as I believe that they are due to sepsis in one form or another. If one could prevent the formation of adhesions the removal of the large bowel would involve no risk whatever to life. In the old subject who has had auto-intoxication for many years and also is feeble and wasted, there is a freedom from the development of adhesions which would appear to be due to an acquired immunity of the peritoneum to the organisms which are probably responsible for their development. At one time we were much troubled by suppuration in the incision in the abdominal wall in advanced cases. This we attempted to meet, on the suggestion of Sir Almroth Wright, by the cultivation of organisms from the urine, if they were present in it, and by the injection of vaccines made from them. We obtained very little benefit in this way. However, by the use of hot compresses frequently applied we have reduced the frequency of suppuration in the wound to a minimum. It is probable that by these means the patient is protected by the products of organisms which exist in the blood or tissues and which collect in the wound.

The Consequences and Treatment of Alimentary Toxæmia from an Odontological Point of View.

By J. F. Colyer.

I propose to preface my remarks on the consequences and treatment of alimentary toxæmia from the dental point of view by a short reference to the two main sources of oral sepsis—namely, (*a*) caries, and (*b*) gingival disease leading to periodontal disease.

(*a*) Caries consists in the progressive destruction of the tooth substance eventually leading to the opening of the pulp cavity. A septic inflammation is set up in the pulp, and in time leads to a similar condition in the periodontal membrane about the apex of the tooth. The amount of oral sepsis arising directly from carious teeth is small compared with the sepsis from the conditions started by their presence. The effect of caries is to render the teeth themselves and their immediate neighbours functionless, with the result that stagnation areas are formed of food débris, micro-organisms, &c., and a marginal gingivitis is started. The sepsis arising in connexion with the latter is far more serious in its effects than that from the septic material present in the carious cavities.

(*b*) *Gingival disease leading to periodontal disease* is by far the most fruitful source of oral sepsis. The condition known as pyorrhœa alveolaris has long been recognized in medical literature. A considerable amount of misconception, however, exists as to this disease, and as the early recognition of the condition is important, I hope you will permit me to dwell for a few minutes upon its clinical aspects. The disease in the early stages is characterized by a slight thickening of the interdental papillæ due to damage from the accumulation of food débris, &c.,

between the teeth. The attachment of the periodontal membrane at the neck of the tooth is next destroyed, and deep pockets are formed around the teeth. The margin of the alveolar process becomes involved, and slowly but surely it is destroyed, and the tooth is lost. The destruction of the alveolar process proceeds at a far greater rate than the destruction of the gum tissue, with the result that the disease is often well advanced before any recession of the gums is apparent to the patient. In the course of the disease infection occurs, and there is a free discharge of pus, giving rise to the condition familiarly known as pyorrhœa alveolaris.

The points I wish to emphasize about the disease are:—

(*a*) The gums may appear to the untrained eye almost healthy, yet the tooth socket may in places be destroyed almost to the apex of the tooth, and pus may be present in the pocket around the tooth.

(*b*) The disease is slow in its progress, more especially in the early stages.

(*c*) The early stages of the disease can often be recognized in quite young adults.

(*d*) The extent of the disease can only be accurately gauged by examining each individual tooth with a fine probe to ascertain the depth of the pockets.

(*e*) The disease is more intractable and more serious in its results in those who are mouth-breathers.

The relationship of nasal obstruction to dental disease has not received the attention it deserves. The effect of mouth-breathing in the child leads to a persistent marginal gingivitis, most marked around the lower incisors, and this eventually leads to periodontal disease. I think it may be safely asserted that anyone who breathes through the mouth, even for a short period during the day, cannot possibly have a healthy mouth. Nasal breathing is essential for a healthy condition of the mouth.

The consequences of dental sepsis can be considered under the headings of (1) children, (2) adults.

(1) In children the carious teeth cause the food to be bolted, and the insufficiently masticated food plus the septic matter from the stagnant areas in the mouth lead to gastro-intestinal sepsis and all the sequelæ that follow that condition. Clinically, we know that children with marked oral sepsis are usually below the normal weight. We know also that caries of the teeth associated with mouth-breathing causes more harm than when caries occurs in children who are normal breathers.

(2) In adults there is ample ground for thinking that dental sepsis

is a most important factor in pathology. Dental sepsis may lead to other pathological processes in two ways: (*a*) By direct absorption of the organisms or toxins, or (*b*) by the constant swallowing of the septic matter, setting up a gastritis which in its turn leads to general intestinal disease.

In those cases in which the skiagraphs show well-marked rarefying osteitis, the general symptoms are often severe, and I think it is possible that in this group of cases the absorption of the toxins is direct. It is important to try to ascertain exactly how the dental sepsis leads to general trouble, and it is therefore desirable that cases should be carefully recorded from the medical as well as from the dental aspect.

In forming an opinion as to the possible relationship of dental sepsis to general alimentary conditions, we must keep clearly in view that dental disease is very prevalent, and that therefore the association with another condition does not necessarily imply cause and effect. Still further, one must not omit to search for other causes of sepsis. That the effects of dental sepsis are far-reaching there can be but little doubt, but I do not think that the effects are different from what we might expect from sepsis in any other part of the body. Given the fact that you may have direct absorption from the body of the bone, it is conceivable that the organisms may form infective thrombi in any part of the body, or the toxins may damage a part the resistance of which is already lowered. If, too, there is constantly passing into the stomach food charged with septic matter, it is natural that a gastritis may follow and lead to intestinal sepsis with all its sequelæ.

The duty of the dental surgeon in the treatment of alimentary toxæmia is to place the teeth in such a state that they may not at any time be a potential source of disease. The treatment must be directed towards rendering the mouth functional: (1) By correcting nasal obstruction if present, and (2) by treating the teeth themselves.

In children it is absolutely necessary that the teeth should be free from tenderness, and experience teaches that unless the carious cavities can be easily filled the teeth are better removed. In cases of extensive caries, the free removal of teeth is called for, and we should have no hesitation, if necessary, in rendering the young children edentulous. The rapid improvement in health that follows the removal of the teeth is often remarkable. Let me quote one example: A small boy, aged 3, came under observation with his teeth so carious that there was but little of the crowns remaining. He was suffering from gastric trouble, and weighed 29 lb., which is about 6 lb. below the normal for the age. In the year following the removal of the teeth he increased 8½ lb.

in weight, and seemed in perfect health. The free removal of the deciduous teeth undoubtedly in many cases leads to a narrowing of the dental arch, and in that way may produce a crowded arrangement of the permanent teeth. This, however, can easily be remedied by treatment. The great advantage to the general health by cutting off the source of sepsis by extraction far outweighs the possible disadvantage to the dental arch that may follow on the adoption of that line of treatment.

In the adult, the treatment of dental sepsis practically resolves itself into a question of the treatment of periodontal disease. In my opening remarks I referred to the fact that periodontal disease consists in the progressive destruction of the tooth socket and the consequent formation of deep pockets. Treatment, therefore, must be directed towards effectually draining the pockets. Simple as this problem may appear, it is in practice extremely difficult to carry out, and it is this fact that makes the treatment of the disease so extremely unsatisfactory. It is claimed by some authorities that vaccine treatment will produce cures of periodontal disease. On this point there is a considerable diversity of opinion, the crux of the whole situation being "what is to be considered a cure." That vaccines may benefit the general condition and raise the local resistance of the tissues is quite possible, but it is difficult to see how they can produce a cure in the real sense of the word, that is, remove from the patient the potential source of disease in the mouth—namely, the pockets around the teeth. When it is established that dental sepsis is causing harm, are we justified in allowing a possible source of sepsis to remain?—and as long as the pockets exist it does remain. There is a section of dental practitioners who consider that free extraction of the teeth affected with periodontal disease is the only safe and reliable treatment, and with them I am inclined to agree.

Another point to be remembered is that the longer the source of sepsis is permitted to remain, the greater is the damage to the tissues. In young adults the most marked and rapid improvement in a general condition will often follow dental treatment, while in those of advanced years this improvement is often slow and not so complete. This difference is naturally to be explained by the fact that in the young the tissues have not been damaged beyond recovery, whereas in those of more advanced years the tissues are damaged beyond repair. This, to my mind, is a most important point, and with increasing knowledge of these cases, I think the tendency will be to insist more and more on the early removal of teeth in cases of dental sepsis, and so place the patient, as far as the mouth is concerned, in a safe position.

(The discussion was adjourned until April 14.)

The Royal Society of Medicine.

April 14, 1913.

Sir Francis H. Champneys, Bt., President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.[1]

Mr. J. B. Lawford: After some hesitation I have come to the conclusion that I shall best meet the exigencies of the occasion by dealing with the subject before us in general terms. I shall, therefore, omit all clinical and pathological details of individual cases, and this hiatus will, I hope, be filled by subsequent speakers. Moreover, I do not propose to discuss the many doubtful points as to the exact methods by which alimentary toxæmia is induced, the active agents concerned in its production, or the clinical and laboratory tests indicative of intestinal putrefaction. These questions have been already discussed or will be discussed by speakers much more competent that I am to tackle such problems.

In a study of the literature of the subject I have found it no easy matter to sift the evidence. The fluidity of our knowledge concerning alimentary toxæmia has been perhaps sufficient excuse for the publication of much that is of doubtful value.

In considering toxæmia, resulting from disorders of the alimentary tract, in its relation to ocular disease, it must be borne in mind that we are in great measure wandering in the realms of hypothesis. Although for several years such toxæmia has been widely accepted, not only as a possible, but as a probable cause of certain forms of disease of the eye and its adnexa, we are still, in the majority of instances, unable to furnish proof that the toxæmia and the ocular lesions are cause and effect. The note of caution so emphatically sounded by the opener of this discussion (Dr. Hale White) is one which should be (but has not always been) borne in mind by every observer, not only in recording cases which are ætiologically grouped under the title "intestinal or alimentary toxæmia," but also in the investigation of cases in which such toxæmia is one of several possible causes. The invocation of toxæmia as an explanation of pathological conditions of obscure ætiology is easy and attractive, and I am inclined to think some observers have too readily succumbed to the temptation.

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[1] Adjourned from March 10.

It has long been recognized that a close association exists between gastro-intestinal disturbances and some forms of eye disease, and that therapeutic measures directed to the derangement in the intestinal tract are not infrequently followed by amelioration in the ocular condition. But in order to establish with certainty the causal connexion between intestinal sepsis and lesions of the eye, or any other organ, something more is requisite than an improvement in the local malady subsequent to treatment of the intestinal tract. The "Diagnosis Therapeutica" is no doubt valuable and helpful sometimes, but as frequently it is an unreliable witness.

As evidence of the nebulous condition of our knowledge of the connexion between enterogenous toxæmia and ocular lesions, I may draw attention to the fact that, with a few exceptions, the authors of recent text-books of ophthalmology, in various languages, either do not refer to the subject, or do so very briefly, and with a conspicuous absence of dogmatism.

In this country comparatively little has been published upon the relation between alimentary auto-intoxication and ocular disease, by those engaged in ophthalmic practice. Hence it seems to me that this discussion is very opportune, and I trust will prove of great value in eliciting the opinions, and recording the observations of many of those engaged in clinical and laboratory investigations. In other lands, especially in Germany and America, a considerable number of papers upon this subject have appeared during the last seven or eight years. Many of those to which I have been able to refer are of decided interest and often very suggestive. Perusal of them shows that considerable difference of opinion exists concerning the ætiological connexion between intestinal disorders and disease of the eyes, and no little doubt as to the reliability of some of the generally accepted evidence of intestinal putrefaction. This uncertainty is one of the many difficulties with which we have to contend in our endeavours to solve the problem now under discussion. What may we accept as conclusive evidence of an intestinal abnormality, of such kind, or of such degree, as to be a likely cause of disease of distant parts, as for example the eye? The answer to this question may possibly be an outcome of this discussion.

Nearly five years ago a well-known ophthalmic surgeon[1] stated that there is no ocular disease *known* to be due to intestinal auto-intoxication, and laboratory examinations have so far failed to isolate any definite toxin to which disease of the eye can be attributed. I believe it is still true that no toxin has been isolated which can be considered responsible for the ocular lesions we wish to label toxæmic. On the other hand, numerous observations have been recorded since the above statement was made (some of them by the author of it), which, to say the least, render it increasingly probable that alimentary toxæmia is a contributory factor, if not the sole agent, in inducing these lesions.[2]

We shall probably be on safe ground if we consider auto-intoxication from the alimentary tract, in causal relation to disease of the eye, as a good working hypothesis and one which helps to explain the clinical manifestations of certain cases better than any other.

I think it may be confidently stated that there is a general agreement in the view that the ocular tissues most liable to suffer in alimentary toxæmia are the uveal tract and the corneo-sclera. Some writers also include the ocular nervous apparatus, and others the eyelids, as peculiarly prone to involvement. It would be an easy matter, however, to produce from published records examples of lesions of practically every part of the eyeball and its immediate surroundings (the eyelids, the ocular muscles and nerves) ascribed, often with great probability, to this form of toxæmia.

There is ample experimental and clinical evidence that the uveal tract is more prone to infection by certain micro-organisms than are other parts of the eyeball, and also that this tunic, or part of it, is very liable to suffer in the toxæmia induced by certain microbes—e.g., the gonococcus—and in other toxic conditions due to perverted metabolism—e.g., diabetes. This cannot be put forward as evidence that these structures are susceptible, or especially susceptible to the unknown toxins manufactured in the gastro-intestinal tract, but it may not unreasonably be held to lend support to that view.

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[1] de Schweinitz.

[2] One of the most interesting and valuable papers on this subject containing the records of very thorough clinical and laboratory examinations of cases is that by de Schweinitz and Fife, entitled "A Contribution to the Possible Relationship of Auto-intoxication to Certain Diseases of the Cornea and Uveal Tract" (*see* bibliography).

For the purposes of this discussion it is convenient to divide the alimentary canal into two parts: (1) the mouth and fauces, (2) the gastro-intestinal tract. That toxæmia may and does result from septic conditions of the mouth, especially that due to chronic gingivitis and pyorrhœa alveolaris, no longer admits of any doubt. The relation of oral sepsis to disease of the eye has received very close attention in recent years. For obvious reasons the observations upon eye disease attributed to oral sepsis are more numerous and more convincing than those in which intestinal toxæmia is assumed to be the cause. The association of pyorrhœa alveolaris and chronic iridocyclitis is well known to all ophthalmic surgeons. Credit is due to Mr. William Lang as being one of the first to recognize the significance of this association. The frequent coincidence of these two conditions; the marked improvement in the ocular lesions when the diseased teeth are removed and the gums suitably treated; the recurrence of the iridocyclitis with a fresh outbreak of pyorrhœa, leave little room for doubt that the ocular lesions are caused by the septic conditions in the mouth, and probably through the agency of toxins. The iridocyclitis in these cases is usually of a quiet, mildly plastic type with abundant punctate deposits, and is marked by great chronicity. We must not, however, overlook the fact that the large majority of individuals in whom pyorrhœa alveolaris exists do not suffer from iridocyclitis, and also that iridocyclitis with precisely similar characteristics is met with in patients whose mouths and teeth are above suspicion.

Recurrent and relapsing forms of scleritis, episcleritis, and sclerokeratitis are met with in patients suffering from pyorrhœa, and may often with reasonable probability be attributed to it. My personal experience in these forms of disease does not seem to support this view of their ætiology. Careful and thorough treatment of the mouth has frequently failed to exert any beneficial influence upon the ocular conditions, and vaccine treatment has in most instances been disappointing.

Another form of oral sepsis in which the tonsils are the seat of purulent foci, or areas of putrefaction, has been not unreasonably considered as the cause of ocular inflammation. There are grounds for the belief that the tonsils may be the portal of entry of some acute infective diseases, and it is permissible to assume that when diseased they may produce toxins capable of inducing ocular lesions.

There are clinical records of the association of chronic inflammation of the tonsils and iridocyclitis.

Although no actual proof can be adduced, there is valuable evidence that choroiditis may result from oral sepsis, including both chronic periodontal disease and tonsillar disease. That septic conditions play a part in the ætiology of choroidal inflammation has long been the view of careful clinical observers; the occurrence of infection of the choroid in septicæmic conditions—e.g., puerperal septicæmia—is well known. Some years ago in a discussion before the British Medical Association, I referred to the possible toxæmic origin of some cases of choroiditis. Since then further evidence in favour of this view has been forthcoming in my own experience and in records of other observers. The variety of choroidal inflammation which I believe to be most frequently associated with, and possibly dependent upon, the toxæmia induced by pyorrhœa alveolaris is that generally known as *localized exudative choroiditis*, the clinical characteristics of which were well described many years ago by Nettleship. It is quite certain, however, that this form of disease of choroid owns other causes and is often seen in patients quite free from pyorrhœa alveolaris or other septic conditions in the mouth. I have not hitherto seen any example of widespread choroidal lesions in which oral sepsis seemed a likely cause.

Certain functional disorders of vision have been ascribed to oral sepsis. Asthenopia of varying type, unrelieved by the correction of refractive errors and muscle imbalance, is reported to occur in patients with pyorrhœa alveolaris, and to disappear when the oral disease has been treated. I have never met with such cases. It seems reasonable to assume that the asthenopia may be an indirect result of toxæmia resulting from pyorrhœa and attributable to the general malnutrition engendered by auto-intoxication.

Leaving the buccal portion of the alimentary tract, and directing our attention to the relation between ocular lesions and enterogenous toxæmia, we are confronted with a more difficult problem. The diseased conditions in the intestine are invisible, and the tests for putrefaction and other pathological processes which are certainly, or possibly, causes of toxæmia are not yet absolutely reliable. Consequently, we have to depend largely upon such evidence as the exclusion of other likely causes, the existence of other signs of toxæmia, and the result of therapeutic measures directed to the gastro-intestinal tract. Positive results from laboratory examinations of the urine and fæces are, of course, extremely helpful, and there is ample reason to think that such aids to diagnosis will become more definite and more certain in the near future.

Until recent years syphilis, rheumatism, gout, and gonococcal infection were the accredited causal agents of many of the inflammatory affections of the eye, but in a considerable proportion of cases of keratitis, episcleritis, sclero-keratitis, iridocyclitis and choroiditis, all these causes could be excluded, and the ætiological factor remained undiscovered; it was then usually thought to be latent rheumatism or gout, and treatment in accordance with this belief was adopted. It is these ætiologically obscure cases for which an explanation is sought in intestinal toxæmia, and recent observations and experience seem to endorse this hypothesis, at least in an appreciable proportion of them. We must, however, be on our guard lest we repeat an old error in accepting toxæmia as a cause of all cases which we fail to account for in other ways. The reports published by some observers leave little doubt in my mind that they have sometimes accepted intestinal toxæmia as a cause of ocular disease on insufficient evidence.

Some well-known maladies, as for example dysentery, in which toxins are abundantly present in the intestinal canal, are accompanied by ocular lesions, such as retinal hæmorrhage, retinal phlebitis, and choroiditis. Although such cases are not primarily examples of auto-intoxication, the analogy between them and putrefactive changes in the gut occurring independently of extraneous poisons may be reasonably close.

Inflammatory lesions affecting the uveal tract occupy a foremost place in the category of ocular disease ascribed to enterogenous toxæmia. One of the earliest records is that of a case of relapsing iridocyclitis reported in 1899 by Bacquis: this occurred in a patient suffering from stenosis of the intestine, and the relapses coincided in time with recurrent blockage of the gut.

Recurrent iritis and iridocyclitis have been reported by most writers on this subject, and Elschnig is of opinion that in all cases of iritis in which syphilis as a cause can be excluded, examination should be made for evidence of septic conditions of the intestinal canal. The type of ocular disease is similar to that known to be associated with oral sepsis; there are usually abundant punctate deposits on Descemet's membrane, posterior synechiæ, cloudiness or opacity of the vitreous; recurrences are frequent, and often sight is seriously damaged and sometimes lost.

Choroiditis of varying type, localized or widespread, running a chronic course and often recurrent, is met with in patients with marked indicanuria and other signs of intestinal derangement, in whom other likely causes of choroidal inflammation can be excluded. These cases, however, present no recognizable feature which can be regarded as pathognomonic of a toxæmic origin.

The development of cataract has also been attributed by more than one writer to intestinal toxæmia. It is common knowledge that cataract is a frequent sequel of any form of chronic uveitis, and this is probably the correct explanation of its occurrence in association with toxæmia.

As we find to be the case in oral sepsis, inflammatory lesions of the cornea and sclera are attributed with considerable probability to toxæmia of intestinal origin; and numerous examples have been reported. Episcleritis and sclero-keratitis have more especially been noted, and in such conditions, if tubercle as a cause can be excluded, their possible dependence upon toxæmia should be carefully considered. I have seen examples of relapsing sclero-keratitis in patients in whom the urine contained *Bacillus coli communis* in large numbers, and in whom other signs of toxæmia were present.

The evidence in reference to corneal inflammation due to intestinal toxæmia is less abundant: a few examples have been published, notably one by de Schweinitz and Fife, and one by Lawson, in both of which the toxæmic origin of the keratitis can scarcely be doubted. The association of recurrent attacks of phlyctenular keratitis with gastro-intestinal disturbance in children is well known, and some writers consider this form of eye disease as one of the most frequent manifestations of toxæmia. Certain it is that treatment of the gastro-intestinal tract usually exerts a beneficial influence upon the ocular trouble. Some anomalous forms of keratitis of the quiet non-ulcerative type, sometimes described vaguely as "nutritional," have been ascribed to toxæmia, but their dependence upon this condition is still problematical.

Good authorities, such as Professor Uhthoff, have expressed doubt that any causal connexion exists between gastro-intestinal auto-intoxication and lesions of the optic nerve and retina; but there are records of cases which at least suggest the possibility that certain well-known forms of optic nerve inflammation may have a toxæmic origin.

Many years ago Horner expressed the opinion that the gastric derangement induced by tobacco and alcohol was responsible, at least in part, for the optic nerve lesions met with in cases of poisoning by these drugs, and the almost invariable occurrence of gastro-intestinal disturbance in cases of tobacco and alcoholic amblyopia has been noted by most observers.

In recent years de Schweinitz and Edsall, from examination of the urine, concluded that the gastritis following abuse of tobacco and alcohol was toxæmic in nature.

Acute retro-ocular optic neuritis, the ætiology of which is often very obscure, may be one of the results of auto-intoxication, and cases are on record which support this view. One striking example is recorded by Wirtz, in which retro-ocular neuritis occurred in a patient in whom abundant indicanuria was present, the urine being free from albumin and sugar: rapid improvement followed upon treatment of the deranged intestinal canal. The optic neuritis associated with chlorosis has been attributed to toxins developed in the intestines, and this seems a reasonable explanation of such cases.

I have failed to find any records of cases of oculo-motor paralysis attributed, with probability, to auto-intoxication. One instance of paralysis of the ophthalmic division of the trigeminal nerve, with keratitis neuro-paralytica, has been reported, and I have seen one case in which the same paralytic conditions may possibly have resulted from intestinal toxæmia.

Recurrent hæmorrhages in the retina and vitreous in young men, described by Eales about thirty years ago, have been attributed to toxæmia due to oral or intestinal sepsis. It is well known that obstinate constipation is nearly always present in these cases, and it is by no means improbable that the intestinal stasis, and the putrefactive results thereof, constitute the cause of the ocular lesions. Further investigations of these cases, with the help of modern laboratory methods, should be undertaken.

The causal relation which alimentary toxæmia, including oral sepsis, may bear to infective processes in eyes on which operations have been performed, is one of great importance. All ophthalmic surgeons are familiar with troublesome and unexpected iritis and iridocyclitis arising several days, perhaps a week, after an uncomplicated operation, such as extraction of cataract. It is conceivable, and not improbable, that these cases are examples of toxæmic troubles, and that the altered conditions of life and the diminished tissue change inseparable from operations and confinement to bed, may favour the development of toxins which manifest their activity in that part of the uveal tract which has been subject to surgical interference.

In conclusion I think I may state, without fear of contradiction, that even if positive proof of the dependence of certain ocular lesions upon alimentary toxæmia is still wanting, much evidence from clinical and laboratory investigation may be adduced in support of this contention. It is, therefore, incumbent upon us so to pursue our investigations that we may ultimately be able to dispel the doubt which now exists as to the effect of alimentary toxæmia upon the eye.

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Professor W. E. DIXON, F.R.S.: The term "toxin" in modern times has come to mean a poison, which when injected into an animal gives rise to antibody formation, but alimentary toxæmia includes poisons of a much wider group. Now it is by no means certain that true toxins are absorbed from the intact mucous membrane of the alimentary canal. We know, for example, that abrin and ricin can be taken with impunity by the mouth, though in doses of about $\frac{1}{25}$ mg. per kilo of body-weight the former is poisonous when injected. Large doses of tuberculin can be given by the mouth to tuberculous calves without eliciting a reaction, though a minute dose under the skin at once produces the typical tuberculin reaction. In the same way snake venom, diphtheria and tetanus toxins are harmless when taken by the mouth. This general rule, however, may have some exceptions, and it is known that in certain people, after they have partaken freely of eggs, such complicated substances as egg albumen may find their way into the urine. In general, however, I think that it must be accepted that alimentary toxæmia is poisoning produced not by "toxins," the term being used in its strict sense, but by relatively simple chemical substances, certainly not of a more complex chemical nature than many of the alkaloids we are in the habit of administering. These poisons must originate as a result of digestion or putrefaction of food, and our attention may be concentrated almost entirely on the digestion of the proteins. It is true that under certain conditions a kind of toxæmia may be induced from carbohydrate metabolism; for example, in certain conditions of the bowel an excess of lactic acid may be found leading to an excess of ammonium salts in the urine, and some four or five times the normal excretion of calcium; the result may be calcium starvation, of which one symptom is probably joint trouble.

When the protein molecule is broken down by the action of acids, alkalies, or by digestive ferments, amino-acids are ultimately produced, and it is in this form that proteins are absorbed, and it is known that the administration of the final cleavage products of an artificial, sterile, pancreatic digestion will serve to preserve nitrogenous equilibrium in an animal as well as the administration of intact proteins.

These amino-acids, of which a considerable number are known, and which vary in kind with the proteins from which they are formed, are then non-poisonous substances and, so far as we are aware, do not give rise to evil results in the animal body. Under the influence of putrefactive organisms these amino-acids may, however, be altered slightly in composition and become toxic. It is not necessarily true, however, that bacteria are out of place in the alimentary canal. It is much more probable that many of them may exert a beneficent effect on digestion and are living in symbiosis. We, for example, possess no ferment in the alimentary canal which will act upon cellulose, but bacteria can hydrolyze it and so assist our digestion. Or, to refer to another of their beneficent functions, the case of lecithin may be mentioned, which is broken up by the pancreatic juice into glycero-phosphoric acid, fatty acids and choline; the last substance if absorbed in quantity might well induce certain toxic effects; but bacterial enzymes break up choline into carbon dioxide, marsh gas and ammonia, and thus the organism is protected.

I propose to mention only some three or four substances which are known to be formed in putrefaction in the alimentary canal, and will refer first to ammonia. Folin has produced evidence to show that the ammonia which is well known to be present in the portal vein comes from the large intestine as the result of putrefaction, and it has long been recognized that much ammonia may be formed in putrefactive conditions of the bowel.

Rovighi[1] thinks that an excess of ammonia or carbamic acid absorbed from the alimentary canal produces hepatic lesions. His experiments were made on dogs; he injected subcutaneously various aqueous solutions of carbamate of ammonia, and obtained an acute intoxication by injecting doses of 0·75 to 1·5 grm., and a chronic intoxication by injecting daily 0·1 to 0·2 grm. The animals lived from fifteen to thirty days, and on post-mortem examination showed certain lesions. In acute poisoning marked dilatation of the hepatic capillaries and vacuolation and shrinking of the protoplasm was present. In chronic poisoning the liver was indurated and small; the capillaries, especially the central vein, were dilated, the hepatic cells turgescent, the nucleus stained badly and some fatty degeneration was present. If the poisoning had lasted a month an increase of connective tissue was observable.

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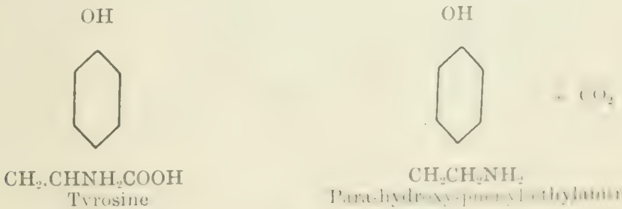
[1] "Ricerche sperimentali," *Il Morgagni*, 1899, xli (i), pp. 149-64.

Joannovics[1] produced a like intoxication by administering the carbamate of ammonium by the stomach—a much more convincing method—since it is by no means certain that the drug would reach the liver in the same form in which it was put into the stomach. He made two series of experiments on dogs. In the first he obtained subacute poisoning in his animal using carbamate of ammonium, and in the second carbonate of ammonium. His animals lived varying times up to seventy-four days, during which period they received up to 6·5 grm. carbamate of ammonium. He gives a detailed histological account of the post-mortem effects on the liver, spleen, and kidneys, and states that the effect of ammonium carbonate and carbamate are alike. His results are in agreement with those of Rovighi.

Many of the amino-acids are acted upon in the alimentary canal by micro-organisms and changed to other substances which may cause poisoning of the system, either because they are produced and absorbed in great excess, or possibly because of some deficiency in the natural protective mechanism. One function of the liver is to destroy such poisonous substances and to render them innocuous, and this may for the time be in abeyance. The simplest method by which the harmless amino-acids are made toxic is by decarboxylation. A few examples will make this clear. Tyrosine may be so changed to para-hydroxy-phenyl-ethylamine or tyramine:—



Tyrosine

Para-hydroxy-phenyl-ethylamine

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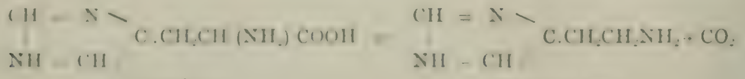
[1] *Archives Internat. de Pharmacodynamie et de Thérap.*, Gand et Paris, 1903, xii, p. 26.

Now tyramine is not formed in a sterile pancreatic digestion, but Barger and Walpole have shown that it can be produced in a medium containing tyrosine which has been inoculated with fæcal bacteria. It is true that Emerson[1] and Langstein[2] believed it could be obtained from tyrosine by digestive ferments alone, but Abderhalden showed that this was not true, and that it is formed only by putrefaction.[3] Tyrosine is pharmacologically inert; tyramine is poisonous.

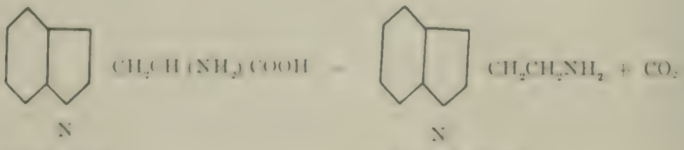
Histidine, though not strictly speaking an amino-acid, is related to them, and may be included in the same group. Histidine is a normal product of digestion, but the decarboxylated compound is poisonous.



Histidine. Histidine base.

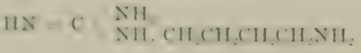
Ackermann[4] obtained a large yield of the poisonous base by submitting histidine to the action of putrefactive organisms.

Tryptophane can also be changed by the agency of putrefactive micro-organisms into its corresponding poisonous amine.[5]



Tryptophane Indolethylamine

Agmatine is another active principle obtained from arginine by the loss of carbon dioxide.



$$HN = C \left\langle \begin{array}{l} NH_2 \\ NH.\,CH_2CH_2CH_2CH_2NH_2 \end{array} \right.$$

Agmatine.

As further examples there are leucin $\left(\begin{smallmatrix}CH_3\\CH_3\end{smallmatrix}\ CH-CH-CH.NH.COOH\right)$ which, by the loss of carbon dioxide, becomes $\left(\begin{smallmatrix}CH_3\\CH_3\end{smallmatrix}\ CH.CH_2CH_2NH_2\right)$ iso-amylamine, and phenylalanine

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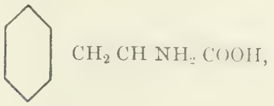
[1] *Beitr. z. Chem. Physiol. u. Path.*, Braunschweig, 1902, i, p. 501.

[2] *Ibid.*, p. 507.

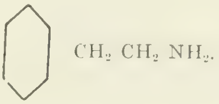
[3] *Lehrb. d. Physiol. Chem.*, Berl., 1909, 2 Aufl., p. 353.

[4] *Zeitschr. f. Physiol. Chem.*, Strassb., 1910, lxv, p. 504.

[5] Laidlaw, *Bio. Chem. Journ.*, Liverpool, 1911, vi, p. 150.

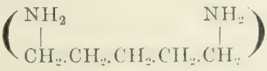
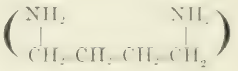


which becomes phenyl-ethylamine—

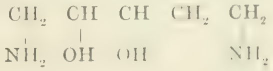


But in each case the law holds that the amino-acid is non-poisonous, whilst the base exerts distinct physiological activity.

But, besides this group of derivatives from amino-acids, other compounds, more or less poisonous, may be produced from amino-acids by putrefactive micro-organisms. Thus, indol and skatol, the crystalline volatile compounds which impart the smell to fæces, are formed from the bacterial putrefaction of certain amino-acids of which tryptophane is one. The introduction of tryptophane into the large intestine of the rabbit causes the formation of large quantities of indol and skatol.[1] But the production of these two compounds in a normal alimentary canal requires two factors: (*a*) Intestinal sepsis; (*b*) proteins containing the tryptophane group. Gelatine, for example, could give rise to no indol.

Cadaverine  $\left(\begin{array}{l}\underset{\textstyle CH_2.CH_2.CH_2.CH_2.CH_2}{\overset{NH_2\qquad\qquad\quad NH_2}{|\qquad\qquad\qquad\;\;|}}\end{array}\right)$ and putrescine  $\left(\begin{array}{l}\underset{\textstyle CH_2.CH_2.CH_2.CH_2}{\overset{NH_2\qquad\qquad NH_2}{|\qquad\qquad\quad\;|}}\end{array}\right)$

are other substances derived from putrefaction of the corresponding amino-acids in the bowel (Ellinger). Cresols and phenols are also formed, but bodies of this type are so little poisonous, and the body is so well able to protect itself against them, that it is doubtful whether their presence is of any great significance from the point of view now under consideration. One of these bodies, however—sepsin—which is usually prepared from putrid yeast or blood, is a violent poison. It is, however, a very unstable substance, easily converted into the harmless cadaverine. The formula suggested by Faust is as follows:—



$$\underset{NH_2}{\underset{|}{CH_2}}\;\underset{OH}{\underset{|}{CH}}\;\underset{OH}{CH}\;CH_2\;\underset{NH_2}{CH_2}$$

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[1] *Beitr. z. Chem., Physiol. u. Path.*, 1903, iv, p. 171.

It may be noted that subsequent observers have not succeeded in preparing this body, and the formula suggested is chemically improbable.

Sepsin or Dioxycadaverine.—The effect of sepsin may be noted from the following experiment: A large dog was injected intravenously with 20 mg. of sepsin sulphate. In a few minutes violent vomiting and diarrhœa occurred, and later bloody stools. The dog died in a few hours, and inflammatory changes of a most intense character were found throughout the alimentary canal (Faust).[1] Without entering into this question further, it is well to remember that the ingestion of putrid meat may give rise to the most violent symptoms as the result of this poison, but it is a substance which is not likely to be formed *de novo* in the intestines, since under the conditions present in the intestines it seems to be converted into cadaverine.

I will now pass to the detailed consideration of the toxic effects of the amino-bases. The immediate action of many of these amino-bases is upon the vascular and muscular systems of the body. In 1907 Dr. Taylor and I found that extracts of normal human placenta contained a pressor substance which was subsequently found to be due to incipient putrefaction. Abelous had previously obtained (1906) from putrid horseflesh a base with similar properties.[2] Barger and Walpole[3] identified two of these pressor substances with certainty, iso-amylamine and para-hydroxy-phenyl-ethylamine. Both these bases injected into animals have a marked similarity in action to that of adrenalin; their action is restricted to such muscle fibres and gland cells as receive a sympathetic supply, and is of the same nature as that obtained by stimulating the sympathetic nerves to those tissues. Iso-amylamine is not so strictly limited to these structures since it causes constriction of the pulmonary and coronary vessels, which are for all practical purposes not innervated. Weight for weight, the immediate effect of adrenalin is considerably greater than that of either of the other two amino-bases, but the action of the latter is slower in onset, though more prolonged than that of adrenalin. It may be mentioned here that both these bases have been shown to arise as the result of putrefactive changes in the cod's liver, and were constituents of cod-liver oil prepared by the old method. Tyramine has also been shown to occur in ripened cheese, being in this case produced from tyrosine by bacterial action in much the same way as it is formed in the alimentary canal. Both these amines differ decidedly from adrenalin in that their action is obtained when they are given by the mouth, and also when injected subcutaneously.[1]

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[1] *Arch. f. Exper. Path. u. Pharm.*, Leipz., 1904, li, p. 248.

[2] *Compt. rend. Soc. de Biol.*, Par., 1906, i, pp. 463, 530.

[3] *Journ. of Physiol.*, Camb., 1909, xxxviii, p. 343.

Indolethylamine, when rapidly absorbed, produces a transient stimulant effect on the central nervous system, causing clonic and tonic convulsions, tremors of the limbs, and vaso-constriction with a rise in blood-pressure. It has a direct stimulant action on plain muscle, which is most marked in the arterioles, the iris, and uterus.[2]

The histidine base, which has been investigated by Dale and Laidlaw,[3] is different from those so far described. The injection of from 10 to 30 mg. of this base into the cat causes immediate vomiting and purging, profuse salivation and laboured respiration with a subsequent period of collapse and light narcosis. During the narcotic stage the pupils are constricted. The fundamental feature of its action is its direct stimulation of plain muscle, by which it produces exaggeration of rhythm with increased tonus; the most responsive plain muscle appears to be the plain muscle of the uterus and the muscular coat of the bronchioles. One curious feature in its action is that it gives rise to a depressor action on the circulation, and is probably responsible for the "vaso-dilatin" which Popielski has found in the mucous membrane of the alimentary canal. It causes also a considerable fall in the rectal temperature. In guinea-pigs, which possess a thick corrugated bronchiolar mucous membrane, this base readily causes death by completely occluding the bronchioles; and a similar death by asphyxia can be produced in cats. It is well recognized that certain cases of morning cough are associated with flatulent dyspepsias; this might be due either to a nervous reflex or be the result of a chemical messenger, and it is conceivable that this histidine base if produced in excess in the alimentary canal might, by producing some bronchial constriction, give rise to an effect of this kind.

Among other amines formed by decarboxylation of amino-acids, it is only necessary to refer to agmatine from arginine; its action is weak and insignificant, but it causes some contraction of plain muscle when in considerable concentration.

It is probable that putrefaction in the alimentary canal, in some degree, is a normal process, in which case it would be expected that we should find evidence of these poisons in the urine of normal people. Abelous obtained a pressor base from the urine of normal men which had many of the properties of adrenalin, and to this he gave the name "urohypertensine." Barger and Walpole consider it probable that Abelous's urohypertensine is iso-amylamine, and nearly all the pressor action is due to a substance which is volatile with steam. A second pressor base can be found in the urine after the complete removal of the first with ether; it is probable that this is para-hydroxy-phenylethylamine.[1] Bain has also drawn attention to the fact that in patients suffering from high blood-pressure the urine contains little or none of these bases, and he thinks that this indicates that they are retained in the body, and play an important part in the production of the high blood-pressure. It should be noted that a vegetable diet reduces the amount of these bases found in the urine, and eggs and fish also reduce the quantity to some extent. There is no evidence to show the reason for their absence from the urine when the blood-pressure is high. Bain's view may be correct, or their destruction may be more complete.

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[1] Dale and Dixon, *Journ. of Physiol.*, 1909, xxxix, p. 25.

[2] Laidlaw, *Bio.-Chem. Journ.*, 1911, vi, p. 141.

[3] *Journ of Physiol.*, 1910, xli, p. 318, and 1911, xliii, p. 182.

As it is now clear that poisons are normally produced in the alimentary canal from bacterial action, and that these are absorbed and excreted, it will be well to note the means which the body takes to protect itself from these poisons, since if no protection existed poisoning would be present, perhaps, as a normal condition rather than as a rarity. It has been pointed out already that some alkaloids like choline may be destroyed in the alimentary canal by micro-organisms, and that others, like sepsin, if formed would probably be converted almost immediately into the harmless cadaverine. Bodies of the nature of carbolic acid combine with sulphuric acid to form compounds which can be rapidly excreted and are non-poisonous, and the amount of these aromatic sulphates in the urine for many years has been supposed to indicate the degree of putrefaction going on in the alimentary canal. Benzoic and salicylic acids combine with glycocoll, and again form innocuous compounds which are very easily excreted. The resulting salicyluric acid, for example, has no poisonous properties like salicylic acid.

Indol and skatol are oxidized in the body, and excreted combined with sulphuric acid, the resulting compounds being :—

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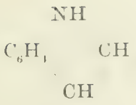
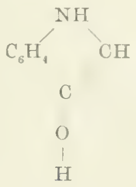
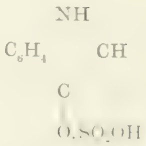
[1] Bain, *Lancet*, 1910, i, p. 1190, and 1911, i, p. 1469.



Indol



Indoxyl



Indoxyl sulphuric acid

without any poisonous effect. Para-hydroxy-phenyl-ethylamine, according to Ewins and Laidlaw, is partly oxidized to para-hydroxy-phenyl-acetic acid, but much of it disappeared completely, and could not be traced.

It has long been known that the liver possesses a remarkable power of destroying toxins and alkaloids. Heger and others showed many years ago that animal tissues possess the power to destroy certain alkaloids. If the tissue juice squeezed out from a frog's liver, and filtered so as to be entirely free from microscopical particles, be mixed with a little hyoscyamine, and kept at 22° C. for two or three hours under suitable antiseptic conditions, the alkaloid disappears; both chemical and physiological tests yield negative results. The same effect can be obtained in the rabbit, but it is less marked; the dog's liver, on the contrary, has little or no destructive power. Cloetta, following on these lines, has recently given an explanation of the natural tolerance of rabbits to atropine.

Lauder Brunton found that the liver had also considerable power of destroying diphtheria toxin, and in some recent work by Lee and myself we showed that tolerance to nicotine depended on the ability of the tissues to destroy the alkaloid, and that destruction occurred mainly in the liver.

Effect of Chronic Poisoning.—Arterial disease in man may begin either in the intima (atheroma) or in the media, when the change is usually spoken of as arteriosclerosis. Of the exact causation of these conditions we know little except that poisons, such for example as syphilis and lead, play a considerable part. Experimental arterial disease in animals has been produced in a great variety of ways. As early as 1889 Gilbert and Lyon showed that injections of bacteria and their toxins produced fibrocalcareous changes, especially if the vessel wall had been mechanically injured. Injury to the vessel has been shown by many to result in severe inflammatory conditions. Josué, in 1903, first drew attention to the action of drugs in producing arterial disease in animals. He injected adrenalin into the veins of rabbits, and produced extensive changes in the middle coats of the arteries. In man it has often been shown that the first sign of this form of arterial disease is fatty degeneration, and Klotz has suggested that it is this fatty degeneration which is responsible for the attraction and deposition of calcium salts.

In recent years it has been shown by different workers in our Cambridge Laboratory that any drug which has the power of considerably raising blood-pressure will, when injected into the circulation of healthy animals, bring about degeneration of the middle coat of the arteries. Generally about six injections must be administered before any changes are observed, and the effects occur alike in young and middle-aged animals. It has been shown that digitalis, squill, apocynum, barium, lead, adrenalin, nicotine, and even the inhalation of tobacco smoke, will all bring about these changes. Hence it may be concluded that any considerable absorption of pressor bases from the alimentary canal might give rise to this condition.

The last experiments to which I desire to draw your attention are, perhaps, the most important—namely, the effect of absorbing small doses of those poisons which are produced in the alimentary canal over a prolonged period. Unfortunately the reliable experiments are not many, but still a number of experiments have been made with some of these amino-bases, and have produced remarkable results. Harvey[1] has noted typical arteriosclerotic changes in the vessels from administering para-hydroxy-phenyl-ethylamine to rabbits, whether by the mouth or intravenously; and Etienne[2] has observed the same type of effect by using the urohypertensine of Abelous, which, as pointed out already, is probably isoamylamine. The plaque formation in the aorta in these cases begins with granular degeneration in muscle fibre. The muscle fibre losing its function, tone in the affected portion of the vessel is lost; the blood-pressure dilates this somewhat, and the elastic fibres lose their crenated form in the middle coat, and appear as straight fibres. Considerable leucocytic activity can be observed at this time. Calcium salts are now deposited around the straightened elastic fibres; so that at this stage sections stained in silver nitrate, and fixed in sodium hyposulphite, show a black band in the substance of the media, whilst the intima may be either quite unaffected, or appear a little thickened. The elastic fibres show a normal reaction to elastic stains, and do not appear to be in any way affected.

[1] *Journ. of Path. and Bact.*, 1911, xvi, p. 95.

[2] *Journ. de Physiol. et de Path. gén.*, 1913, xv, p. 105.

In many of these experiments the heart was found enlarged, and exhibited fatty degeneration. In Harvey's experiments the most remarkable changes were found in the kidneys, and these were present both in the experiments in which the drug was injected, and those in which it was administered orally. The change consisted in a chronic nephritis showing many similarities to the large white kidney of man. He regards the changes in the kidney as secondary to a vascular sclerosis. The first changes to be produced in such experimental animals are the plaques in the aorta: these are followed or associated with thickening of the walls of the renal arteries, but at this stage without alteration in the glandular substance. Later the reaction of the renal epithelium to stains is impaired, and dilatation of some of the tubules and glomeruli becomes evident. This dilatation later may be very marked, and desquamated cells and albuminous material are often observed occupying the dilated lumen; Bowman's capsules are frequently thickened. The tubular epithelium has a ground-glass appearance, and the cells are flattened. In more advanced cases round-celled infiltration may be observed round the damaged glomeruli and tubules, and freshly formed connective tissue eventually replaces in part the glandular structure. It is a simple matter to produce acute nephritis in animals, but a chronic nephritis of this nature produced by a non-irritant substance is not common, and it must be remembered that this may result from the absorption of the amino-base by its natural path, the alimentary canal.

Dr. J. C. G. LEDINGHAM: My interest in the question of alimentary toxæmia is naturally confined to the bacteriological and biochemical evidence which has been advanced by the school of Metchnikoff in support of their master's thesis. That thesis asserts that the poisons elaborated by the micro-organisms of the intestinal tract, and particularly those harboured by the large intestine, are the most potent factors in the production of a long array of symptom-complexes loosely grouped under the term "auto-intoxication," and, further, that these same poisons are the essential causes of the sclerotic changes in the organs which accompany old age. The thesis, as you can understand, is, therefore, a perfectly straightforward one, and one which ought to admit of both observational and experimental bacteriological proof. In the absence of proof capable of satisfying the bacteriological critic, the thesis must remain little more than a fascinating hypothesis.

It will be my purpose, therefore, to discuss as briefly and succinctly as the time at my disposal permits, the main bacteriological evidence which Metchnikoff and his pupils have brought forward during the past four years in support of the theory of intestinal intoxication. This evidence has been sought for in the following types of research: (1) An investigation of the flora of the large intestine or of the faeces with a view to the classification and identification of the various bacterial groups and their bio-chemical properties. (2) Experiments on the toxic action of filtrates of special groups of these micro-organisms in animals. (3) Analogous experiments with substances of definite chemical composition, especially indol. (4) Alteration in the intestinal flora as the result of food changes, constipation, and operations undertaken for the removal of the large intestine.

In connexion with the first of these lines of research the main work has been done by Metchnikoff himself and his pupils, especially Distaso, who have built on the earlier work of Tissier, and I would here point out that French authors are too prone to the assertion that our knowledge of the intestinal flora has been in the main the outcome of French work. Such, of course, is by no means the case. In fact, the chief contributions to our knowledge of the coliform group have come from workers in this country and in America. It is, however, to the anaerobic flora particularly that Metchnikoff and his pupils have directed their attention, as it is these proteolytic anaerobes which they specially incriminate as playing the chief rôle in what they call "intestinal putrefaction." It would be out of place to enter into a description of these various anaerobes, as I am convinced that the question of the classification and identification of these groups is far from settled, and there are probably far more names than bacteria to correspond. It is doubtful if much that is new on this subject has emerged from recent French work. The chief putrefactive organisms whose cultural and biochemical properties have been studied in connexion with intestinal putrefaction have been *Bacillus perfringens, Bacillus sporogenes, and Bacillus putrificus*, but it cannot be said that we have yet accurate methods of differentiating anaerobes. One must also mention the acidophile or acetogenic organisms which, by the French observers, are regarded as highly beneficent organisms by virtue of the inhibitory effect exercised by their acid products on the growth and activity of the putrefactive types.

Of these acetogenic organisms, which have the property of producing acid from lactose and of being able to grow in acid media, the most important perhaps is the *Bacillus bifidus*, of Tissier. This author found that this organism is the chief representative of the flora of the breast-fed child—a statement which more recent work has confirmed. It may be noted that *Bacillus bifidus* is a strict anaerobe and is Gram-positive.

A very similar organism, to which attention has been paid, is the *Bacillus acidophilus* of Moro. As the child passes on to more varied food there is a gradual change in the flora from the purely fermentative type represented by the acid-formers above mentioned to a combined flora of the fermentative and putrefactive type—using the term "putrefactive" in the sense of ability to break down nitrogenous constituents to the stage of peptones and proteoses, and mono- and diamino-acids. Further, katabolism of these cleavage products is effected mainly by the representatives of the coli group, many of which have the power of producing indol from tryptophane and some of producing phenol from tyrosin.

Now, it is seen that in the adult normal human flora, the Gram-positive acetogenic organisms represented by *Bacillus bifidus* are rare and never predominate, as in the young child. The predominant group is that of the *Bacillus coli*, and in addition there are the representatives of the putrefactive anaerobic group (*Bacillus putrificus*, *Bacillus perfringens*, *Bacillus sporogenes*, &c.).

Indol is formed chiefly by coliforms, and only to a small extent by these putrefactive anaerobes. It is, therefore, all the more surprising that Metchnikoff and his school should have drawn so much attention to the anaerobes as the chief factors in intestinal putrefaction, while at the same time their energies should have been devoted to the investigation of the toxic action of indol—a product chiefly of coliform activity. How far the indican in the urine is an evidence of a measure of intestinal putrefaction by micro-organisms is not yet completely settled, but admitting for the sake of argument that it does come from the action of micro-organisms in the intestine, it is not at all clear why it should be regarded by Metchnikoff and his school as the chief toxic agent produced by their action. Indol, of course, is a substance of definite chemical composition, while the known bacterial toxins are of unknown chemical composition. How far, then, the Metchnikoff school is justified in taking a simple substance like indol as the toxic product *par excellence* of the action of putrefactive intestinal microbes is extremely doubtful. Recently the opinion has been revived by Emmerich that in cholera the symptoms of intoxication are not due to specific vibriotoxins but simply to the nitrites, which, as you know, the *Vibrio choleræ* is capable of forming in the test tube.

Little support has been given to this view in the case of cholera, and it would, therefore, seem all the more gratuitous to regard indol as the main toxic product of putrefactive organisms, the pathogenic action of which has been only very insufficiently studied. Let us see, therefore, what has been the main result of the experimental work performed with these putrefactive anaerobes and with their presumed chief toxic product, indol. In one of his earliest papers Metchnikoff tested the toxic action of filtrates of some of these anaerobes. Filtrates were tested intravenously on rabbits in doses of 7 to 8 c.c. per kilo of body-weight. These are very large doses. For an ordinary full-grown rabbit this would mean at least 20 to 25 c.c., and it is not surprising that in some cases a fatal result ensued. Great variation, however, was apparent in the toxic power of these filtrates (*Bacillus perfringens*). Nevertheless the result of these experiments led Metchnikoff to state that in the putrefactive organisms of the digestive tube we have a source of auto-intoxication. Further experiments on this line were performed by his pupil Korentschewsky, who tested filtrates of anaerobic organisms isolated from rabbits' fæces. Occasionally quite potent filtrates killing in 1·3 c.c. per kilo were obtained, but in other cases doses of 20 c.c. per kilo did no harm. The strongest filtrate was prepared from *Bacillus perfringens*. Experiments were also performed on rabbits by intrarectal injection of filtrates, and there was some very slight evidence that antibodies to these organisms appeared in the blood serum.

In the case of dogs fed daily with broth cultures of *Bacillus perfringens* (in very large doses, 200 to 500 c.c.) progressive loss of weight occurred with slight anæmia, and when the animals were killed some evidence of sclerotic changes in the kidneys was found. It is surprising that nothing worse happened after such heroic dosing.

It must not be supposed, however, that the putrefactive organisms are the only defaulters. The flora of the large intestine is, according to Metchnikoff, wholly bad and therefore we have other series of experiments with preparations of staphylococci, coli, &c.

According to Manouélian atheromatous plaques appeared in the aorta of a large proportion of rabbits inoculated repeatedly with staphylococci (in seventy-three out of eighty-six) and also in monkeys. These plaques occurred at the level of the arch of the aorta. It must be noted, however, that Weinberg found spontaneous atheroma in 4 to 19 per cent. of rabbits. More recently Bertrand and Berthelot have isolated from the fæces of patients suffering from chronic auto-intoxication an organism which, when grown on a special medium containing histidine, yields a highly toxic product apparently identical with β-imid-azolethylamine, a base previously obtained by Ackermann in the putrefaction of histidine and also by Barger and Dale in ergot. The organism which yielded this toxic product belonged to the coliform group, and, according to the authors, was closely related biochemically to the *Bacillus lactis aerogenes*. The authors make the statement that this particular organism was never found in the fæces of normal healthy people. This is a very important statement and confirmation must be awaited before it can be accepted.

I turn now to the animal experiments performed with definite chemical substances which are understood to arise from the action of micro-organisms in the intestinal tract. In these experiments by Metchnikoff, Dratschinski, and others, potassium phenylsulphate and indol have been the chief substances whose toxic action on animals has been investigated. With potassium phenylsulphate atheromatous changes in the aorta of rabbits, guinea-pigs, and monkeys were produced, and small cell infiltrates in the liver and kidney. Rabbits received by the mouth 0·04 grm. of paracresol during a period of one to four months, and 61 per cent. of the rabbits showed atheromatous plaques. Dratschinski gave similar doses of indol daily to guinea-pigs by the mouth, and obtained evidence of atheromatous degeneration of the wall of the aorta with chronic interstitial processes in the organs, such as the liver, kidney, and suprarenal capsules. These foci in the wall of the aorta invariably contained calcareous deposits, and the question to be decided was whether these foci met with in the animals intoxicated with indol were merely further stages in the development of the cartilaginous foci met with in similar situations in normal guinea-pigs, especially those of advanced age. Such foci were met with in 67·5 per cent. of normal guinea-pigs, weighing 250 grm. and over. The author was of opinion that hyaline and calcareous foci encountered in the intoxicated animals were distinct from the cartilaginous foci found in normal animals, and concluded that intoxication with indol was the exciting cause. It does not appear to me, however, at all proven that the indol, if it effected anything at all, did any more than hasten the calcareous transformation of these normal cartilaginous foci, and the question of the influence of indol in this connexion must still be considered as undecided.

I now come finally to discuss the changes which have been stated to occur in the composition of the intestinal flora in cases of intestinal stasis and in cases which have suffered resection of the large intestine. This question has recently been attacked by Distaso, who had the advantage of obtaining material from Mr. Lane's cases. Distaso roughly classifies the organisms of the intestinal tract according as they do or do not produce indol. In the normal adult man the fæces, as is well known, show a preponderance of indologenic organisms of the coliform group. In constipation, according to Distaso, there are practically no Gram-negative organisms, but there is a great abundance of spores, the indol-formers being now of the sporulating type. There is also an absolute diminution in the quantity of bacilli present. The conclusion drawn by Distaso that the flora of constipation is an index of intoxication is entirely unsupported by the evidence he adduces. In cases operated upon for removal of the large intestine it was observed that the flora showed not so much a qualitative change as a quantitative change, with a consequent alteration in the functional activity of the organisms concerned. Before operation the flora was one typical of constipation, while after operation the indologenic organisms had largely given place to the acetogenic (i.e., *Bacillus bifidus and Bacillus acetogenes*) which are regarded as highly beneficent. The effect of this change, according to the author, is that the acid produced by the latter organisms inhibits the activity of, or actually destroys, any putrefactive organisms present. The evidence on which this statement is based was the fact that, when the fæces were inoculated on a potato medium, marked acid formation took place, while growth on broth containing boiled white of egg only rarely was followed by proteolysis, indicating an action on the part of the putrefactive organisms.

It is very questionable how far it is legitimate to assume that the function of a flora in the body can be determined by an *in vitro* experiment of this rough order. To prove a change in flora of this character would require far more extensive bacteriological and biochemical research than has so far been devoted to the subject, and it is indeed remarkable that the author should make the astounding statement "that after this study the theory of chronic intoxication by the work of intestinal organisms cannot any longer be in doubt." As a matter of fact, it is not clear from the paper how often the fæces were examined in these cases and whether samples were taken at operation from the bowel above the resection. If, as stated, only two examinations of the fæces at a year's interval were made, the data so obtained would be of little value. Such cases would require to be examined repeatedly before and after operation and under all conditions of diet.

Distaso further asserts that the intestinal flora of the normal adult is noxious, and yet he asks the reader to rejoice with him over the microscopical appearance of the fæcal flora of one of these operated cases. This flora, of which a drawing is appended, is characterized as "une des plus belles," but certainly the flora of the constipated person—a drawing of which is placed alongside—seems far the more attractive. With regard to the work done by the Metchnikoff school on the influence of food on the composition of the fæcal flora, I would merely refer to certain experiments of Bertrand, who found that rabbits fed on potatoes always gave a marked indican reaction in the urine, while those fed on carrots gave no such reaction. When a carrot rabbit was placed on potato diet indican appeared. The intestinal flora of the two groups did not differ qualitatively, but the potato-fed rabbits had more *Bacillus coli* in the fæces. No quantitative data on this point are supplied. Bertrand's view was that in the potato-fed rabbit there was much less acid production from the fermentation of sugar than in the carrot-fed rabbit, and consequently there was nothing to inhibit the formation of indol by the coliforms present. Also it is stated that the flora of the potato-fed rabbit was very poor in amylolytic species, but data on this point are wanting.

It cannot be said that work of this kind advances the subject far. One is tempted to ask why chemical examinations of the intestinal contents were not made at various levels and accurate records given of the organisms met with at these levels. In all the recent work on auto-intoxication and on sour milk therapy it has to be remembered that the inhibition of intestinal putrefaction by milk and milk constituents, especially milk sugar, was known years before Metchnikoff popularized the administration of milk soured by lactic acid bacilli.[1] As pointed out by this author, the effect of such treatment may be due not to acid produced by the bacilli supplied but to that produced by the *Bacillus coli* acting on the sugar, which is generally increased in the diet. Protein is thus spared, and consequently protein decomposition products do not appear. A bacteriological basis for the view that the activity of lactic acid bacilli inhibits that of the putrefactive organisms is still lacking.

In conclusion, I am of opinion that the investigation of the biochemical action of intestinal organisms yields a very large and promising field for future research, and may throw a flood of light on this question of auto-intoxication, but it does not seem to me that the results achieved in the types of researches so far published by the Metchnikoff school lend any very marked support to the thesis.

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[1] *See* an excellent survey of this subject by Kendall, *Journ. Med. Research*, 1912, xxv, p. 117.

Dr. James Galloway: From the days when we were house physicians, the occurrence of alimentary toxæmia, as the term is understood in this discussion, must have been familiar. Many of us may recollect the occasion when we were called to the casualty department late on a Saturday evening and found awaiting our services a vigorous looking young man, whose features were scarcely recognizable on account of an outburst of severe urticaria. We found on inquiry that his week-end dissipations had been stimulated by a diet of stout and winkles; and our judgment as to the cause of the urticaria was supported when we found that the best treatment in such cases was the administration of an emetic, followed by a copious black draught. No doubt remained in our minds that the urticaria was due to alimentary toxæmia, caused by poisons absorbed from his alimentary tract.

In such a case as this, the relation in time between the disturbance of the alimentary tract and the visible indication of disease is so close that their causal relationship is admitted by all. But in the case of most other indications of intestinal and visceral disturbance the relationship between the probable cause and its manifestation cannot be so easily traced. Even with the knowledge now at our disposal as to the nature of the poisons derived from various fermentative processes, the chain between cause and effect cannot be distinctly demonstrated; mathematical methods of proof are not applicable, so that all we have to offer in support of our beliefs are the judgments drawn from individual or collective clinical experience. Nevertheless, little doubt is left in the mind of the clinician that causal relationships do exist, and it may be well to point out at this time some of the more obvious external signs of disease of the alimentary tract.

The poisons capable of producing toxæmia resulting in these manifestations of disease seem to come from two sources: First, those contained in the food itself, or arising from the food by the action of ferments in the alimentary tract; these ferments may be those normally produced by the various accessory glands, or may have some abnormal origin, owing to disease or other causes. Second, those resulting from fermentative or putrefactive processes due to the action of organisms which may be normal denizens of the alimentary tract or interlopers. Most of the external manifestations seem to be more easily traceable to the first group of poisons rather than to the second, though this may very possibly be due to the fact that the noxious consequences of the action of many of these organisms, such as bacteria, are more difficult to appreciate and to trace to their original source.

The outward manifestations group themselves easily in four main categories:—

(I) Those resulting from disturbance of the blood-vessels which disappear leaving no permanent trace. In this category may be grouped the varieties of urticaria, evanescent or of long duration, the types of erythema including exudative and vesicating forms.

(II) Morbid conditions in which an inflammatory lesion progresses to actual infiltration of the integuments with cellular elements and terminating in persisting deformity, such as dilatation of blood-vessels, atrophy, persistent dermatitis, ulceration, and scarring.

(III) Purpura, in its different forms—punctate, diffuse, and often associated with other hæmorrhagic manifestations.

(IV) Pigmentation due to increase or abnormal distribution of pigments closely related to those of the normal type, such as melanin, or in other cases to pigments more recently derived from hæmoglobin and due to hæmolysis resulting from the circulating intestinal toxins.

Group I.

Of the first group the various forms of simple or acute urticaria are by far the most common. The relationship between the ingestion of an unusual article of food, such as shellfish or certain fruits, with the consequent outburst of urticaria, is so close as to leave no reasonable doubt that the poisonous material is contained in the ingested substance, or is very easily elaborated from it by the digestive ferments. A little more difficulty is felt when dealing with other varieties of the disease, such as the recurring type of urticaria, in which the lesions are usually not so severe or extensive as in the acute single attack; or the chronic variety in which the lesions are usually diffusely scattered, though not large individually, and leave indurated lesions, giving rise to the term "papular urticaria." These papules not infrequently develop long-standing pigmentation, and may even resemble the rare condition known as "urticaria pigmentosa," of which the exact morbid relationships are still unknown.

In reference to the causation of these less common types of urticaria, the connexion between ingested poisons and the production of the lesion is not so easily traceable, but information gathered from clinical investigation strongly indicates the existence of the connecting chain. For instance, in one case it may be discovered that some individual article of food little suspected to be the cause has by a process of elimination been at length excluded from the dietary. As soon as the sufferer denies himself this treasured delicacy the troublesome and long-standing urticaria disappears. Also, although the treatment of the chronic and papular varieties of the disease is by no means satisfactory, and often very disappointing in its results, the largest amount of success follows treatment by careful selection of the diet—sometimes by restriction of proteins, at other times by refraining from carbohydrates, accompanied by treatment adapted to keep the alimentary canal in a healthy state.

The relation between the various forms of erythema and alimentary toxæmia is more obscure. That such a relation does exist is borne out by the clinical experience of the causation of some of the simpler forms of erythematous eruption. Take, for example, the simpler types of non-exudative erythema. It is well known that such outbreaks are apt to occur after the disturbance of the lower bowel and its contents by the administration of injections, or as a consequence of lavage of the rectum and colon. The cause in this case is not likely to be due to absorption of the fluid used for purposes of washing out the bowel, but to substances pre-existing in the alimentary tract, or rapidly formed in consequence of the interference with its contents. The material absorbed into the body producing attacks of erythema in cases such as these may, with some degree of likelihood, be ascribed to the effects of bacterial decomposition. No doubt, at any rate, exists in our minds as to the causal relationship of the erythema and the administration of the injection. It is reasonable, therefore, to suspect that other conditions of the large intestine, such as those produced by disease of the mucous membrane, may manifest themselves by erythematous eruptions on the surface, and as a matter of observation simple erythematous eruptions are by no means infrequent in the course of the various forms of inflammation of the bowel. These outbreaks are very apt to be purpuric.

The types of erythema exudativum in which, in addition to dilatation of the capillaries, escape of the contents of the vessels occurs into the tissues—either the blood serum or the blood cells—are by far less common than the simple erythemas. We are less likely, therefore, to observe the association between eruptions of erythema exudativum and affections of the alimentary tract, but that the relationship does exist in many cases is more than probable. The close connexion between outbreaks of exudative erythema and diseases of internal organs has frequently been noted and emphasized by various writers, and the toxæmia producing these attacks may arise with greater probability from the alimentary canal than from other viscera. Instructive examples are quoted by Osler and other observers.

The general impression given by clinical observation of this group of external manifestations is that the urticarial type is more frequently produced by the direct absorption of noxious ingesta or as a consequence of the more simple chemical changes in foods occurring in the upper portion of the alimentary tract from the action of the natural alimentary ferments; while eruptions of erythematous nature more frequently arise from affections of the intestines, especially the large intestine, in which the terminal processes of digestion take place, the poisonous materials involved being of a more subtle character, or perhaps due to the results of fermentations produced by bacteria or other organisms. The purpuric types of erythema seem especially to indicate that the changes occur in the colon or large intestine.

GROUP II.

The external lesions which we have been considering are those due to vascular phenomena, which leave no permanent trace behind. Closely allied to these lesions are certain others in which, in addition to the erythematous condition, tissue changes occur. Of these the criterion is the appearance of new cellular elements, neither red blood cells nor white blood cells, but newly formed cells, characteristic of various types of inflammatory infiltration. When these new cells are observed destructive tissue changes must occur in some degree, though it may be very slight, and as a result we may observe erythematous lesions with the same symmetrical disposition as some of the others we have discussed, but which do not vanish, leaving as their result atrophic or even ulcerating destruction of the surface. The type of this morbid state is the disease usually known as lupus erythematosus, a condition closely resembling in its distribution erythema multiforme, but terminating in some degree of destruction or atrophy. The ætiology of this mysterious affection is by no means well understood, but its connexions with visceral disturbances should never be overlooked. A good deal of attention has been drawn to its peculiar relationship with certain forms of disease of the kidney. In other cases a poison arising from the alimentary tract may be suspected of being the cause. A characteristic example is as follows:—

A woman, aged about 35, came under observation with an acute outburst of lupus erythematosus affecting symmetrically not only the face but also the extremities and the trunk, accompanied by fever and much constitutional disturbance, resembling, therefore, the well-known type of the disease sometimes called "exanthematic lupus erythematosus." The duration of this case was about three years. The investigations into the possible causes of the disease were at first unfruitful, but soon it was noted that the patient was gradually developing the signs and symptoms of cirrhosis of the liver. It was not, however, till well on in the course of her malady that it became known that the patient, who in all other respects was exemplary in her social behaviour, secretly indulged in alcohol to an excessive degree.

Such cases occurring in the course of medical experience naturally give rise to the belief, and indicate with a good deal of certainty, that certain forms of alimentary toxæmia may result, not only in such conditions as hepatic cirrhosis or fibrosis of the kidneys, but also in the outward and visible sign of a chronic inflammatory erythema, ending in induration or atrophy of the skin. In this relationship notice should be taken of numerous cases in which permanent dilatation of blood-vessels results in the production of the different clinical types of telangiectases, or even of angiomata. A good deal of attention has recently been given to the different forms of these permanent vascular lesions. Some of them seem to occur almost certainly as the result of circulating poisons affecting the blood-vessels mainly. They are frequently seen in cases in which cirrhosis of the liver and kidneys have followed toxæmia arising in the first instance from the alimentary canal.

GROUP III.

The probability of alimentary toxæmia being the cause of certain cases of purpura has long been recognized clinically, for the connexion between the outburst of purpura and the disturbance of the alimentary canal is sometimes so close that it gives as clear evidence of their relationship as the outburst of urticaria following the eating of some unusual article of food. This relationship is well seen in some of the cases producing the type of the disease known as Henoch's purpura. Many purpuric lesions arise, no doubt, by a peculiar development, or simply by intensification of the conditions already noted producing erythema. They form, however, on account of their aspect, so characteristic a group that they claim special attention.

One of the most remarkable conditions in which the connexion between poisoning from the alimentary tract and the outburst of purpura is shown is in certain stages of hepatic cirrhosis. In these cases of cirrhosis, sometimes at an early period of the disease, a patient is attacked by a violent outbreak of purpura, the lesions occurring throughout the body affecting large areas, and showing profuse extravasation of blood. These outbreaks are often associated with internal hæmorrhages, most commonly from the lower part of the bowel. Such attacks of purpura seem to occur in the early stages of obstruction to the portal circulation caused by the contracting of the newly formed connective tissue in the liver. As a result the portal blood containing the crude materials absorbed in the alimentary canal is forced to seek collateral channels to regain the general systemic circulation. Much of it returns directly by means of the vena cava, thus closely reproducing the conditions established by the well-known porta-caval fistula of Eck. The blood returning by these newly formed channels of circulation carries with it the products of intestinal digestion, or possibly abnormal noxious substances due to the impaired action of the alimentary canal. Under normal conditions the liver is the organ in which these materials are further elaborated, thereby being rendered innocuous, or are destroyed; but in the unusual conditions produced by portal obstruction the tissues, the blood-vessels, and the blood itself are poisoned for the time being. It is under these circumstances that certain exceedingly violent attacks of purpura arise and they strongly suggest the causation indicated. It is not surprising to find that hæmorrhages from the bowel are associated with these purpuric outbreaks. New channels are being discovered by the returning blood, such as those uniting the superior and inferior hæmorrhoidal vessels and the radicles of the internal iliac vein; extravasation is likely to occur under these circumstances from the dilated blood-vessels, quite apart from the noxious effects produced on the tissues by the abnormal constituents of the blood. In such a case the attack of purpura may be the first indication, drawing attention to the visceral and intestinal disease. Complete recovery from the hæmorrhagic symptoms may occur, the patient being even able to resume his usual work in life. The cirrhosis of the liver, of course, remains, or gradually progresses, but the gland apparently resumes its functions as the purifying organ of the portal blood to a sufficient extent to protect the body, or the tissues of the body succeed in protecting themselves in some other way against the action of the poisoned blood. It is likely that the poisonous materials producing these changes in the tissues and in the blood are the results of the ordinary digestive processes or the simpler forms of chemical decomposition rather than of bacterial fermentations.

Another variety of purpura presents small hæmorrhagic areas, or petechial or punctiform lesions. The relationship between this condition and alimentary toxæmia is very distinctly shown in the variety of disease to which the name of "Henoch's purpura" has been given. An instructive example occurred in the case of a young girl, who presented herself for treatment two or three years in succession with attacks of intestinal pain, melæna, hæmaturia, and numerous purpuric lesions. On every occasion the attack followed an autumn visit to the country, and the eating of unusual foods such as berries and nuts.

The same type of purpura is very familiar in association with chronic inflammatory conditions of the colon, such as the varieties of colitis and dysentery. The attacks are especially prone to appear when other evidences of toxæmia or septicæmia become prominent.

There are grounds for associating the type of purpura characterized by large areas of cutaneous hæmorrhage with absorption of poisons from the upper part of the alimentary tract. These poisons may arise from infected food materials or may be produced by the normal, or only slightly abnormal, processes of intestinal digestion. The petechial and punctiform types of purpura, on the other hand, appear to occur as the result of the terminal digestive changes or abnormal fermentative processes occurring in the lower reaches of the bowel, possibly influenced by the micro-organisms present in the alimentary tract. This is the type of purpura most often seen in cases of severe septicæmia which seem to be traceable to the pyogenic inflammations of the mouth, gums, or nasopharynx.

GROUP IV.

A fourth group of the visible manifestions of alimentary toxæmia includes the abnormalities of pigmentation. The unhealthy, dusky discoloration of the skin occurring in various forms of indigestion is a matter of ordinary observation. Much attention has been drawn in the course of this discussion to the discoloration occurring in the cases of disease to which the name of intestinal stasis has been applied. It is difficult to be quite sure of the exact nature of this pigmentation. It is partly due, no doubt, to disturbances of the circulation producing unusual conditions of opacity or translucency of the skin, but in other cases, no doubt, it is due to a definite increase in the amount of the pigments, identical with, or nearly related to, the normal skin pigment. The cases, however, which are most striking are those in which the pigmentation is due to unusual or abnormal pigments.

The first to which reference should be made is the peculiar yellow tint, probably resulting from severe hæmolysis, followed by liberation of hæmoglobin in the blood serum and the formation of pigments, of which urobilin is one. This hæmolytic, or "urobilin jaundice," as it is sometimes called, is well seen in the severer forms of anæmia, many of which are associated with septic disorders of the alimentary tract; the poisoning in these cases giving rise, as one of its consequences, to abnormal hæmolysis is probably of bacterial origin. It is very usual to find these hæmolytic changes associated with obvious purpuric lesions.

The most striking of all the varieties of abnormal pigmentation is seen in the cases to which the name of "hæmochromatosis" is given. The pigment in this condition is not so close a blood derivative as is urobilin, and probably more than one variety of pigment may be present Of these a pigment still containing iron has been identified with some certainty, to which the name of "hæmosiderin" has been given; while another pigment, free of iron, is known as "hæmofuscin." The staining and discoloration of the tissues resulting from the formation and disposition of such pigments is often of a most remarkable character. In some cases it is of a pronounced slate-black colour, while in others the tint is a full mahogany brown. There can be no doubt also that the depth of the pigmentation may vary from time to time in the same case. The relationship between some of these cases of hæmochromatosis with poisoning arising from the alimentary tract is very close. Cases are on record in which inflammatory conditions involving the abdomen and interfering with the action of the intestine have coincided with the development of the slate-black pigmentation already mentioned. The discoloration may show itself on the surface of the body in a degree which can scarcely be imagined unless the opportunity has arisen of seeing such a case. In one such case recovery from disease affecting the intestine following operation was accompanied by the gradual disappearance of discoloration of the most intense character. This patient was well, in vigorous occupation and health several years after treatment.

On the other hand, the type of disease associated with enlargement of the spleen, progressive loss of health, with severe anæmia, and frequently with repeated discharges of blood from the bowel, and in which cirrhosis of the liver occurs as a terminal feature, shows pigmentation of the red-brown tint. In a case presenting these features, of which this drawing is shown, the pigmentation of the surface was of a pronounced mahogany-red colour. In this category may also be included the cases of discoloration due to alterations in the hæmoglobin.

to which the names of methæmoglobinæmia and sulphæmoglobinæmia have been applied.

In many of these abnormal pigmentations the relationship to alimentary toxæmia is a close one—probably that of cause and effect.

DR. A. MANTLE: Large numbers of cases of functional disorders of the alimentary canal find their way to Harrogate from all parts of the world, and amongst them many are seen with symptoms which I think we are right in considering due to absorption of chemical poisons, so-called "toxins," the symptoms varying with the particular tissue affected. In endeavouring to seek the source of the poison in such cases, a careful history must be taken, and tropical influences must be considered in some; and above all we must have a picture in our mind's eye of the mucous tract from the nasopharynx to the rectum, for it is this particular structure which is at fault, for we are dealing with a mucous disorder. We are told by bacteriologists that in the nasal and buccal mucous membranes bacteria abound, and careful investigations of apparently trivial infections of the mucous membrane of the nose and tonsils have explained the cause of the lesions in the joints in not a few cases of rheumatoid arthritis, which condition we know is not primary but usually secondary to the absorption of micro-organisms from other places, notably mucous membranes. In cases of alimentary toxæmia with or without joint symptoms, we therefore first inquire into and examine the mucous membrane of the nose, tonsils, and mouth, including the teeth. We cannot lay too much stress on pyorrhœa and dental caries as common causes of the early disturbances of the alimentary canal; for although the acid secretion of the gastric mucous membrane is very often effective in destroying the bacteria swallowed into the stomach when this mucous membrane is in a healthy condition, yet should it be in a condition of catarrh with a diminution of motility, and more or less stasis of its contents, the same destruction of bacteria does not take place, and they pass on to the intestine to infect *its* mucous membrane.

The paramount importance of a mouth being free of septic organisms is by no means yet fully recognized by doctor or dentist, and it is sometimes a little difficult to make the patient understand how detrimental the condition of pyorrhœa really is. I am accustomed to point out that it would be very little good to attempt to cure the inflammation caused by a foreign body in one's hand if we let it remain to excite further inflammation, for on removal a cure results. They usually accept the simile, and let the dentist start his work of getting the mouth free of sepsis before, or whilst I commence my treatment. I saw two cases bearing on this point on the Thursday before Good Friday. A well-developed gentleman, aged 34, complained of flatulent distension after meals, and constipation. He was anæmic, and had chronic nasal catarrh, and, on examining the mouth, there were pyorrhœa and eight suppurating stumps covered over by a plate, the fitting of which his dentist had improved only the day before! He was an intelligent man, and, acting on my advice, he had the mouth cleared of all stumps the next day. There was found to be a little enlargement of the liver and a catarrhal condition of the mucous membrane of the stomach and colon, and much mucus was passed. The bowel was treated by lavation, and Ems treatment was given for his nose, and he was massaged. His hæmoglobin steadily increased in value, and he has returned home very much better, chiefly through being rid of a septic condition of mouth. The same day I saw a lady who had had several series of vaccines for cystitis and pyelitis, in which the *Bacillus coli* was active, which commenced eighteen months ago, the vaccines having given good results. She was sent to me, however, for an irritable condition of the bowel with the passage of mucus, and she had some eczema. On examining the mouth, there was found excellent and costly crowning of several unhealthy stumps, and considerable pyorrhœa. The stomach was not in a catarrhal condition, but she had some mucous colitis which was treated. I advised her to make only a short stay, for her doctor agreed with me that she would get the fuller benefit of a course of treatment when she was free of oral sepsis. Having investigated the teeth and the mucous membrane of the mouth, tonsils, and nasopharynx, we come to the stomach, and it was the fermentative changes in gastric dilatation which Bouchard believed were the causes of auto-intoxication. We have for some time considered mucous ulceration to be not infrequently due to a bacterial invasion of the gastric mucous membrane from the mouth in the subjects of pyorrhœa, the ulceration being associated with gastritis; and we are indebted here to Sir Bertrand Dawson for considerable and important bacterial research as to the conditions associated with gastritis and gastric ulceration. I quite agree with him, for my experience proves it, that just as colitis may be an infection from the mouth and stomach, and subsequent to gastritis and duodenal catarrh, so may the stomach be infected from below in association with chronic appendicitis and catarrhal inflammation of the colon. By either route the gall-bladder and pancreas may become infected, and explain the pain in gall-stones being so commonly referred to the stomach, and epigastric pain being suffered with appendicitis. From a large experience I look upon a toxæmia of intestinal origin induced by chronic constipation and catarrhal inflammation of the colon as the most common form met with. It is usually that form of constipation in which there is delay in the passage of the fæcal matter favouring bacterial multiplication, and is often met with in the neurasthenic. Deficiency of motility in the intestinal wall leads to stasis, and the irritation of hardened fæces to catarrh of the mucous membrane. In the other form of constipation which is due to deficient expulsive power, the fæces have reached the pelvic colon, but are not efficiently expelled, and we get the same catarrhal symptoms, the result of irritation. That there are thousands of constipated people who get no bad symptoms, such as have been described, there can be no doubt, for we must remember that with such a strong barrier of defence as we possess in the cells of the mucosa, together with the powerful antitoxic function of the liver, probably it is only when the mucous membrane is inflamed, and its epithelium stripped by abrasions, or is ulcerated—or, again, when the liver or the kidneys become inadequate—that toxins find their way into the circulation.

Before speaking of the most common *symptoms* of alimentary or intestinal toxæmia, I should like to emphasize one point, which is, that there is sometimes a more or less general infection of the mucous membranes, and I am quite satisfied that in some cases it reaches the respiratory mucous membrane, affecting the bronchial tubes and smaller bronchi, and sometimes producing asthma. It is common knowledge that articles of food taken into the alimentary canal will produce rashes on the body such as urticaria and erythema, and in the same way some toxins taken with food are absorbed, and quickly produce active changes in the bronchial mucous membrane and asthma, and patients will tell you that such and such an article of food provokes an attack. It is therefore quite a reasonable assumption that toxins absorbed from the nasopharynx, gastric or intestinal mucous membrane, may also affect the respiratory mucous membrane.

A gentleman, aged 52, was sent to me early last year for compressed air treatment for emphysema and bronchial catarrh. Upon investigating his case I found that before any chest symptoms manifested themselves three years ago he was on board a steamer when a number of passengers and he amongst them, had ptomaine poisoning from food. He had a flatulent condition of bowel with distension of the abdomen, and a tendency to constipation. His stomach was normal, and his teeth and gums, but he had a congested condition of the nasal mucous membrane which he had had for three years. He was given treatment for the nose, and compressed air for his lung condition, but I found that the *origo mali* was in the colon, and intestinal lavation proved this to be the case, for large quantities of mucus in the ejecta pointed to a chronic catarrhal inflammation. With this treatment he gradually improved, but as a matter of precaution he took another short course before the winter, with the result that he has entirely lost his asthma. I have seen several such cases of asthma associated with colitis, in which there seemed to be a general infection of the mucous tract, but all have had some chronic catarrh in the nasal mucous membrane. From one of the worst of these I made an inquiry only a few days ago, and learn that the patient has only had two slight attacks this winter, since his treatment last September.

We now come to the most common symptoms of alimentary toxæmia, but it will readily be understood that these will depend largely upon the structures affected. I believe the toxins from the intestine have a special affinity for the nerve centres, and although I am quite prepared to admit that neurasthenia is the cause of constipation, the intestinal wall and reflexes taking on the lethargy common to the neurasthenic condition, yet the bowel stasis increasing bacterial activity, and possibly absorption of toxins, may increase the nervous symptoms of neurasthenia, and also affect the vasomotor centres, bringing about the circulatory changes which are so common in these cases, for there is usually too little blood in the periphery, and too much in the splanchnic area, with a tendency to congested viscera. There is a feeling of a great sense of fatigue, and patients say they always feel tired. Sir Lauder Brunton says, "the *Bacillus coli* seems to have a special power of producing fatigue toxins, and many people in whose intestines it exists in great abundance suffer from constant weariness and a feeling of fatigue." The skin is usually sallow, and as we may get various skin eruptions, which I have already referred to, from ingested poisons in articles of food, so we may find the toxins of chronic intestinal stasis affect the skin, probably due to their irritation or stimulation of the vasomotor centres, and produce such eruptions as eczema, psoriasis, urticaria and pruritus. The joints may become affected, and I have already incidentally mentioned rheumatoid arthritis as being produced by infection from the nasopharynx, but the same joint symptoms arise from poisons absorbed from the intestinal mucous membranes, and we know many poisons favour the joints.

In five cases, all women, I have seen purpura in association with intestinal toxæmia. Two of the patients have been the wives of doctors, and one, a neurasthenic case, I have watched for several years. The eruption appears in purple spots, circular in shape as a rule, and they mostly appear on the lower extremities, and on the trunk and upper extremities to a very limited extent. They do not disappear on pressure, and are not raised above the surrounding surface, and usually fade in a few days, leaving a brownish stain. Exertion will bring on an attack. In nearly all these cases there have been mucous colitis and pyorrhœa.

When we come to the treatment of the condition I think it will be readily conceded that we must remove all the sources of poison whether in the nose or mouth, or those the result of stasis in the stomach or bowel, and it is surprising what can be done if the point or points of infection are localized. For those who can afford it, probably the regime, waters and massage obtainable at a spa are an advantage, but there are those who cannot have this luxury but who can be well treated at home or in hospital by gentle aperients, intestinal lavage and massage. The chief part of treatment in the opening addresses has been that ably advocated by the surgeon, Mr. Arbuthnot Lane, who, whilst showing a very practical acquaintance with the symptoms of toxic absorption, does not invoke the physician's aid. I agree with Dr. Hale White, who wonders if the surgeon is aware of what can be done by medical means, and sometimes with the assistance of vaccines in these cases. Not only does Mr. Arbuthnot Lane, in his contribution, advocate the excision of the colon, or the short circuiting of it, for chronic constipation and colitis, but he suggests it for this condition when associated with rheumatoid arthritis: but "Is there no balm in Gilead? Is there no physician there?" Has medicine lost its healing art, that an organ when unhealthy, either must be removed or put on to a siding as no longer of any use? I am quite ready to admit that there may be cases of chronic constipation, and one of such was sent to me by a colleague of Mr. Lane in which medical treatment was of little or no avail, and his operation of short circuiting was attended by brilliant results; but this is quite the exception, for I am more and more pleased with the results of spa treatment in colitis and rheumatoid arthritis.

In the treatment of intestinal stasis, which is the most fertile source of alimentary toxæmia, it is then advisable to resort to intestinal lavation, but if the condition of the mucous membrane is not showing signs of catarrh which the absence of mucus would suggest —this treatment need not be long continued and others adopted. I have for some years used in the treatment of these cases, with the best and most encouraging results, 30 oz. of sulphur water, which is an excellent water for lavage at a temperature of 100 F. This is introduced into the colon through a rectal tube by hydrostatic pressure at a height of 2 ft., and is repeated, for the first injection merely clears away the fæces, whilst the second acts as a bath to the mucous membrane. An immersion bath of sulphur water follows, which is not only good for the arthritic pains which are common, but also for the skin and general circulation which is usually much disturbed and unbalanced. That the fluid flowing into the bowel does pass through the whole length of the colon by virtue of hydrostatic pressure is easily proved, for when a case in which appendicostomy has been done is injected with fluid coloured with an aniline dye, the latter is seen to come out of the opening in the abdomen. If there is a just reason for pursuing this line of treatment, which the evidence of considerable quantities of mucus would suggest, I usually continue it every second day for some ten or twelve baths, and this is as a rule sufficient to get rid of the mucus. I think it advisable to repeat the treatment once or twice at yearly intervals, but so much depends upon diet, massage, and abdominal exercises as to the bowel regaining its functions. Every year one meets with cases in which this treatment is successful, not only in helping the physician but the surgeon also. There are some cases operated upon for appendicitis in which the symptoms are not entirely relieved by operation, the explanation being that whilst the appendix to the colon, which has been in a condition of chronic catarrh, is removed, the colon is sometimes left in the same catarrhal condition, and it is then that intestinal lavation may be helpful in clearing up the case.

I congratulate the Council of this Society in bringing together the great branches of our profession as they have done in this discussion, for with the physician being found ready to co-operate with the bacteriologist and also with the surgeon and dental surgeon when deemed necessary, and the surgeon similarly with the physician, we shall by this combined effort accomplish much more in the future in combating disease than we have in the past.

Mr. J. G. Turner: From observation of the results of treatment, I am sure we are justified in asserting that there is a toxæmia due to dental sepsis, and that it is partly of local origin from absorption by the mouth tissues, and partly of true alimentary origin from absorption in the visceral alimentary tract. The results of treatment directed solely to the dental condition in clearing up symptoms such as those described by Dr. Vaughan Harley are so striking that I feel no manner of doubt as to cause and effect.

This dental or oral sepsis is due to four factors: (1) Dental caries; (2) pyorrhœa alveolaris; (3) tartar; (4) artefact conditions leading to sepsis, and these are all governed by the one root factor, stagnation — stagnation of germs and of soft, sticky, starchy, and sugary food. We may call it germ-carbohydrate stagnation, and with certain limitations as to the causation of tartar and pyorrhœa alveolaris which I need not now enter into, germ-carbohydrate stagnation may be taken as the root factor of dental sepsis.

Examination of the lower animals, wild and in captivity, furnishes a striking proof of this statement. The mouths of fish are invariably free from dental sepsis; germ-carbohydrate stagnation is for them an impossibility. So for the same reason are the mouths of wild carnivora; but put them in captivity and feed them on bread, buns, &c., and their mouths become septic with pyorrhœa alveolaris, tartar, and dental caries. Domestic dogs and cats afford everyday examples. Monkeys furnish an interesting study in this respect. In the wild state their fruit-eating habits induce a certain percentage of caries and of pyorrhœa alveolaris, but this latter is localized and due to impaction of fibrous vegetable debris. In the captive state, judging from examination of material afforded by the London Zoological Gardens, the percentage of dental caries is trebled and generalized pyorrhœa alveolaris is common. The captive monkey is fed largely on bread. Dr. Plimmer, pathologist to the Zoological Society, tells me that a considerable number of monkeys die at the gardens of an acute enteritis which he attributes to pyorrhœa alveolaris, not only in the well-marked later stages, but in the far earlier stage when the gums are apparently healthy. At this stage there can be squeezed up from under the gum edge a pultaceous mass consisting largely of germs, and to this Dr. Plimmer attributes the infection causing the acute enteritis. This fact is interesting, because I have already described a similar condition of solid stagnation in man.

In man I have found the same rule to hold good. The more opportunity there is of germ-carbohydrate stagnation the greater the prevalence of dental sepsis. In the large collection of skulls of South Sea Islanders preserved in the Royal College of Surgeons Museum there is a striking frequency and parallelism of occurrence of pyorrhœa alveolaris and caries of the necks of the teeth. Soft coco-nut prepared in various ways forms a large part of the diet of these people. Among British skulls dating from about 1650 to 1840, the incidence of both dental caries and pyorrhœa alveolaris is very small. That is, there was an absence of dental sepsis when bread was made of coarse ground meal and sugar was a scarce article of diet. Nowadays, with impalpably ground flour and a superabundance of sugar, we find dental sepsis in almost every mouth.

If this be the true explanation of the prevalence of dental sepsis we shall expect to find it as often in children as in adults, but in children we shall not expect to find a fully developed stage of pyorrhœa alveolaris. This is exactly what is to be found. Dental caries is rampant, and the early stage of pyorrhœa, a marginal gingivitis, or the stage of solid stagnation incriminated by Dr. Plimmer in monkeys, is equally omnipresent, and the results are seen in biliousness, malnutrition, &c.

Marginal gingivitis is found in children of all ages, amongst both the temporary and permanent teeth, and as time elapses passes into a true pyorrhœa. A fully developed pyorrhœa alveolaris is a frequent occurrence before the age of 20. Making allowance for the time required for the process to establish itself, and recognizing its early stages, pyorrhœa alveolaris equally with dental caries is a disease of young life.

Now I want to point out that dental sepsis is a power for evil in far less obvious stages than that in which a flow of pus is an obtrusive symptom, and long before the teeth are too tender to eat on—at a time when patients argue that their teeth have never hurt them—and I want to indicate a means whereby its presence may be made manifest. If a wisp of cotton-wool be passed between any two teeth, preferably molars, at the gum edge, dipping if possible into the periodontal sulcus, it will push out in front of it a certain amount of stagnant material. If there be much sepsis present this stagnant material will have a fœtid smell, which if not at once obvious may be made so by spreading it over a warm surface, as by rubbing it between the finger and thumb. Swallowing of such putrid potted meat cannot, I imagine, be other than poisonous to the patient. Storehouses for decomposing food and debris are afforded by every carious tooth, by every pyorrhœa pocket, by the gum edges of all tartar deposits, by all overlapping fillings, ill-fitting crowns and bridges and unclean tooth-plates, and the swallowing of it is, I think, an important factor in alimentary toxæmia. This dental fœtor is as common in children as in adults.

I notice that Mr. Arbuthnot Lane considers we are in an absurd position in holding pyorrhœa alveolaris to be a primary disease, because he finds free extraction does not cure all cases of intestinal stasis, and because short-circuiting produces a rapidly beneficial result on the pyorrhœic condition. It seems to me it would be irrational to expect cure to follow in every case of even total extraction when infection has been going on for ten, twenty, or thirty years, and in reply to the second argument it has been long recognized that improvement of the patient's general condition is followed by improvement of the local. Moreover, Mr. Lane must show us that dogs, cats, monkeys, and South Sea Islanders suffer from intestinal stasis.

In the treatment of dental sepsis I am certain that the most radical is the surest, and here we need have no fear of the result of free extraction. Very little inquiry and observation are needed to show that at all ages of life mankind can live well and comfortably without teeth; but at no age is he safe in the presence of sepsis, oral or other. The numbers of healthy people with few or no teeth will come as a surprise to those who have made no inquiry. It is the fashion to insist on the repair and replacement of injured teeth, but no inquiry is made as to the amount of artefact sepsis thereby induced. Overlapping fillings, ill-fitting crowns and bridges, all uncleansable, are only too common, and tooth-plates themselves are extremely difficult to clean; they are commonly coated with a greasy, sour-smelling layer of germs and food debris. In face of this it is surely better, especially in hospital practice, to acknowledge and be glad of the fact that man can live without teeth. But I do not wish to imply that nothing can be done short of extraction; most gratifying results can be obtained by placing the teeth in such a state of cleanliness that it is easy for the patient to maintain that clean state. Everything must be smooth and polished, and every tooth or root surface not united to alveolar tissues by living attachment must be easily accessible for cleansing. This can all be achieved by local measures—e.g., scaling, laying open of pockets, &c.

Of the use of vaccines for the local condition I am not hopeful. Reaccumulation cannot be prevented, nor the advent of a new germ or of a variant of the original germ, nor the putrefaction of debris; and for the same reason I fear the result of vaccines as a treatment of alimentary toxæmia following dental sepsis will be disappointing. Moreover, the good effects of vaccines are apt to be evanescent.

In prevention we have a more hopeful prospect. I object strongly to the suggestion that pyorrhœa is found in people who clean their teeth properly. When present it is proof positive of failure to clean—but, indeed, to clean the teeth is an extremely difficult matter and requires expert instruction. For the prevention of dental sepsis cleanliness of itself suffices. If we could induce a return to the use of coarse-ground cereals we should probably reduce the amount of dental sepsis by three-quarters by cutting off the supply of soft, sticky, starchy food.

Dr. A. F. HERTZ: As my contribution to this discussion I propose to examine the evidence on which a diagnosis of toxæmia due to chronic intestinal stasis can be made. It might be gathered from what some clinicians have said that it is possible to make a diagnosis from the symptoms alone. If, for example, we could accept the statement that chronic intestinal stasis is "a necessary antecedent factor" in the development of all forms of tuberculous disease, the discovery of tubercle bacilli in the sputum would logically lead to a diagnosis of chronic intestinal stasis. Few, however, will agree with a statement for which so little evidence has been brought forward. Even the symptoms, which are by common consent regarded as characteristic of chronic intestinal stasis, are by no means pathognomic. The worst case I have ever seen of intestinal toxæmia, in which the classical symptoms of extreme emaciation, extensive pigmentation, evil-smelling sweat, and cold extremities were present, was due not to chronic intestinal stasis, but to chronic diarrhœa, the result of some intestinal infection contracted by a lady whilst big game hunting in West Africa.

It is not at all uncommon to be told by a patient, who is in the habit of taking large quantities of aperients for constipation, that the lassitude, headache, and abdominal discomfort from which he suffers are better if he forgets to take his aperient and his bowels are not opened. His symptoms appear to be due to the absorption of poisons from the abnormally fluid fæces in his colon, and are therefore a result of the diarrhœa produced by his aperient, and not a result of his constipation. It is important before examining such cases with the X-rays that the patient should not take any aperient for some days. When this has been done I have often been able to demonstrate the complete absence of intestinal stasis, although many of the patients were contemplating some operation for the relief of their supposed constipation. The bismuth reached the rectum in the normal time, but the patients had become so convinced that their bowels would not act without help that they made no attempt to open them, as they did not feel the violent desire which resulted from the use of their aperients. When it was explained to them what the X-rays has shown, and also that a finger inserted into the rectum proved that it was full of fæces only waiting to be expelled, they were persuaded to try to open their bowels and succeeded in doing so. A gentleman, aged 38, whom I saw in April, 1910, was convinced in this way, and he has had his bowels opened regularly ever since without treatment of any sort, although up to that time he was taking an enormous quantity of aperients, and was sent to see me for an opinion as to whether the colectomy, which had been advised, ought to be performed.

It is well known that the frequency of the stools gives no certain evidence as to the existence of intestinal stasis. Thus a condition analogous to retention of urine with overflow may occur in the bowel, the patient being exceedingly constipated, but passing numerous small stools every day. On the other hand, in the condition aptly described as "greedy colon" by Sir James Goodhart, the patient may only open his bowels once every three or four days and yet be perfectly healthy; in such cases I have found with the X-rays that the passage through the bowels is generally normal in rate, but the quantity of fæces formed is so small that several days may be required for sufficient to collect in the pelvic colon to give rise on entering the rectum to the call to defæcation. By giving some charcoal with breakfast one morning and watching for its appearance in the stools, the total time taken in its passage through the alimentary canal can be ascertained; but this method gives no indication as to the part of the bowel in which stasis occurs.

We are therefore driven to the conclusion that the only reliable means of determining whether a patient is suffering from chronic intestinal stasis, and of ascertaining exactly what parts of the alimentary canal are at fault, is a series of examinations with the X-rays. It is, however, first necessary to determine whether large doses of bismuth salts interfere with the normal activity of the bowel; for it has rightly been pointed out that they are among the best drugs for combating certain forms of diarrhœa. In order to test this point, Mr. F. Cook and Mr. E. G. Schlesinger, at my suggestion, took charcoal on a number of occasions at different hours in the day, their bowels being regularly opened in the morning at about 9 a.m. By this means they found the shortest time required for the charcoal to appear in their fæces. On repeating their investigations with 2 oz. of bismuth oxychloride added to the charcoal they found that the time was unaltered. I have since been able to confirm these experiments in a number of constipated individuals. I believe that bismuth salts only influence those cases of diarrhœa in which excess of sulphuretted hydrogen has developed as a result of abnormal putrefaction; this gas is a powerful stimulant to the intestinal movements, but is rendered inert by combining with bismuth. In common with many others, I now use barium sulphate instead of bismuth oxychloride, as it does not combine with sulphuretted hydrogen and under no conditions affects the motility of the stomach or intestines; it is equally non-poisonous, the shadow it throws is almost as dense as that of bismuth salts, and its cost is only one-twentieth.

In some unpublished investigations Dr. Rippmann and I found that the weight of the bismuth or barium salts mixed with the food produces a very slight degree of distortion of the stomach, which is greatly increased in cases of atonic dilatation; it also tends to drag the cæcum and the end of the ileum downwards. It is therefore clear that the smaller the dose used the more accurate will be the results obtained by means of the X-rays. Except in very fat individuals 2 oz. of bismuth oxychloride or barium sulphate are ample for all practical purposes: the use of 6 oz. trebles the error.

Valuable as the X-rays are in the investigation of the motor functions of the alimentary canal they have led to many erroneous diagnoses owing to a want of appreciation as to what constitutes the normal. In a recent discussion at another society cases were related by various speakers, in which a diagnosis of intestinal stasis was made, although the details given of the time at which different parts of the bowel were reached fell well within the normal limits. Thus one speaker concluded that the presence of bismuth between the hepatic flexure and the iliac colon thirteen hours after the meal in one of his cases suggested some mechanical difficulty in the left iliac fossa! It would be rare to find less evidence of "stasis" in a normal individual.

The first examination of a case of intestinal stasis by means of the X-rays was made by me in 1907 after my fellow-workers and I had examined a considerable number of normal individuals. The figures which we published of the average time taken for bismuth to reach different parts of the bowel in normal individuals have since been regarded as the standard, and any case in which delay, when compared with this standard, has been observed has too often been diagnosed as an example of intestinal stasis. The error of this is obvious, when it is remembered that our figures represented the average of considerably divergent times, and more extended experience has convinced me that the normal limits are even wider than we originally supposed. Thus in fifty-eight normal individuals examined by Alan Newton seven hours after a bismuth meal, the furthest point reached by the bismuth was the cæcum in four, the ascending colon or hepatic flexure in twelve, the transverse colon or splenic flexure in thirty-one, the descending or pelvic colon in eleven. Though the average furthest point is in the transverse colon, corresponding to what we described in 1907, it would clearly be incorrect to say that stasis was present in an individual in whom the bismuth had not reached beyond the hepatic flexure after seven hours.

GASTRIC STASIS.

A meal consisting of half a pint of porridge or bread and milk mixed with 2 oz. of bismuth oxychloride or barium sulphate is completely evacuated by the stomach in about four hours. If, therefore, any bismuth is still seen in the stomach six hours later, gastric stasis is certainly present. It is obvious that this rule only holds good if nothing is eaten or drunk between the barium meal and the last examination. Supposing, for example, the barium meal is eaten at 10 a.m., and lunch is taken at 1 p.m., the latter becomes thoroughly mixed with the small quantity of barium and food, which is normally still present in the stomach. Instead of all the barium being evacuated by 2 p.m., some will still be present two or three hours later, when it will again be mixed with the food, if tea is now taken. At 6 p.m., therefore, eight hours after the barium meal, a small shadow may still be seen, as I have found by actual experience in normal individuals, and if dinner is taken between 7 p.m. and 8 p.m. the last trace of barium will not be evacuated until midnight or later. I have repeatedly seen a diagnosis of gastric stasis made because a shadow was still present in the stomach six or more hours after a barium meal, although this was perfectly normal, as more food had been given in the interval, and a subsequent examination, in which this source of error was avoided, showed that the stomach emptied itself in the normal time.

The nearer a barium meal approaches a normal meal in taste and consistence the more reliable will be the results obtained. For this reason the barium sulphate should be mixed with bread and milk or well-made sweetened porridge, instead of with water and lactose, as the latter makes a most unpleasant mess, the nastiness of which inhibits the normal gastric movements and secretion. An example of this has been recorded by Dr. Ironside Bruce. More recently I found some bismuth still present in the stomach twelve hours after a lady had taken a mixture of this sort, which had made her feel extremely nauseated. The next day she took an equal quantity of the same salt of bismuth in porridge, which she liked, and every trace of it disappeared from the stomach within six hours.

When the precautions described are taken, it is found that no connexion of any sort exists between gastric and intestinal stasis. In organic pyloric stenosis, which always gives rise to gastric stasis, the chyme, which passes through the pylorus abnormally slowly, often traverses the intestines at the normal rate. The constipation, which is almost invariably present, is due solely to the small quantity of fæces formed owing to the deficiency of chyme reaching the intestine; too little fæces, therefore, reach the pelvic colon to give rise on entering the rectum to a daily call to defæcation. This view is confirmed by the fact that gastro-enterostomy at once relieves the constipation by allowing an adequate quantity of fæces to form.

Conclusion.—When precautions are taken to prevent errors in diagnosis it is found that intestinal stasis never leads to gastric stasis.

THE DUODENUM IN INTESTINAL STASIS.

Under normal conditions the bulbus duodeni or proximal portion of the first part of the duodenum is more or less constantly visible during gastric digestion. Peristalsis only occurs in it to a very limited extent, the chyme which has come to it from the stomach remaining in it sufficiently long for neutralization to occur by its admixture with the alkaline duodenal secretion, pancreatic juice and bile. The pyloric sphincter then relaxes again, and the entry of a further quantity of chyme causes that already present to overflow into the distal portion of the first part of the duodenum, from which it is immediately conveyed by a rapid peristaltic wave. As the bismuth-containing chyme is greatly diluted in the bulbus duodeni by the digestive secretions, its shadow is less dense and defined as it passes through the rest of the duodenum, and its bulk is never sufficient to unfold and fill completely any part of it after the bulbus duodeni has been left. It generally stops for a short time somewhere in the distal part of the duodenum before being conveyed with equal rapidity into the jejunum. As it passes along the small intestine by frequent very rapid peristaltic waves, which carry it a few inches at a time, it becomes more and more difficult to recognize with the X-rays, owing to the subdivision it undergoes at each stopping-place as a result of segmentation and to its admixture with further quantities of intestinal juice; finally, it is completely lost to sight until the chyme begins to collect in the last few inches of the ileum.

When the stomach empties itself with unusual rapidity, as it does in cases of duodenal ulcer, and less frequently in certain other conditions, the duodenum at any given moment contains more chyme and bismuth than under normal circumstances; consequently, the whole duodenum, which in healthy individuals is never more than half full, even when the stomach is emptying itself most rapidly, becomes filled by the bismuth-containing chyme in its passage to the jejunum. Careful observation shows that there is no stasis; the bismuth passes rapidly into the jejunum, being replaced in the duodenum by a further quantity from the stomach. Owing to an error in the interpretation of the X-ray appearance it has been thought that the duodenum was dilated in such cases, whereas it really is of normal size, but contains more chyme than usual, and therefore gives a denser and wider shadow. The so-called writhing of the duodenum, which has been described as occurring in cases of supposed intestinal stasis as a result of a duodenal kink, is nothing else than the normal peristalsis and segmentation rendered more clearly visible than usual by the great quantity of bismuth-containing chyme present. This "writhing" of the duodenum was recently demonstrated to me in the case of a woman suffering from gastric symptoms; a few days later Mr. Steward operated and found an organic hour-glass constriction of the stomach, but the duodenum was completely free from obstruction and dilatation. As the evacuation of the stomach is always accelerated by lying down on the right side, the duodenum is filled more rapidly and may therefore be seen more readily, if the patient assumes this position before turning on his back for the examination. The skiagrams, which have been published as examples of the dilated duodenum, which is stated to give rise to duodenal ulcer and to be due to obstruction by a duodenal kink, represent, I believe, a duodenum of normal size, which is exceptionally full as a result of posture, as we are told that before the skiagram is taken "it is necessary for the patient to lie on his right side for two or three minutes," whilst in cases of duodenal ulcer the rapid evacuation of the stomach is an additional factor. Moreover, the skiagram is taken in the horizontal position, although those who believe in the frequent occurrence of duodenal kinks state that they are only produced in the erect position and disappear on lying down. In over fifty cases of uncomplicated duodenal ulcer I have never yet seen a dilated duodenum or a duodenal kink, and it is a characteristic feature of the condition that the stomach is hypertonic and empties itself with unusual rapidity, the bismuth passing through the small intestine and reaching the colon with remarkable speed. These observations are in complete agreement with those of Dr. Barclay, of Manchester, Mr. Rowden, who has investigated all of Sir Berkeley Moynihan's more recent cases at Leeds, Dr. Kreuzfuchs, of Vienna, and many others, none of whom have ever seen the kink or dilatation, the radiographic evidence for which depends upon the unconfirmed investigations of a single observer.

The constipation, which is generally present in cases of duodenal ulcer, is not the cause of the ulceration, as in many cases it only develops after the symptoms of ulcer have appeared, and it almost invariably disappears as soon as the ulcer heals—whether as a result of medical treatment or of the performance of a gastro-enterostomy.

Conclusion.—Intestinal stasis does not lead to duodenal kinking, dilatation, or ulceration, and duodenal ulcers are associated with an unusually rapid passage of chyme out of the stomach and through the whole of the small intestine.

ILIAC KINKS, ILIAC STASIS, AND THE ILEO-CÆCAL SPHINCTER.

In 1903 Keith demonstrated the existence of a strong ileo-cæcal sphincter in man. It was thought by many that its function was to prevent the regurgitation of fæces from the cæcum into the ileum when antiperistaltic waves passed down the ascending colon. X-ray investigations have, however, shown that antiperistalsis does not occur in man under normal conditions; moreover, the ileo-cæcal sphincter does not prevent regurgitation into the ileum, as, in common with other observers, I have seen a bismuth suspension pass into the ileum when run into the colon through the rectum at as low a pressure as 1 ft. of water. There can be no doubt that the function of the sphincter is, as Keith originally suggested, to prevent the contents of the ileum passing too rapidly into the cæcum. Some recent observations made with Mr. Alan Newton, of Melbourne, have confirmed and amplified my earlier investigations on this subject. We have found that the bismuth-containing chyme reaches the end of the ileum an hour or even longer before any appreciable quantity passes into the cæcum and that the ileum is often still full four, five, or even more hours after the last traces of bismuth have left the stomach. Consequently an accumulation of chyme occurs in the last few inches of the ileum, where it remains and undergoes digestion actually for a longer period than in the stomach. During the whole of this period active segmentation, but very little peristalsis, can be seen. It is clear, therefore, that the function of the ileo-cæcal sphincter is to prevent the passage of the contents of the ileum into the cæcum until sufficient time has elapsed for digestion and absorption of foodstuffs to be complete, as the chyme which reaches the cæcum contains only traces of nutrient material in solution. Iliac stasis is thus a normal physiological condition of the utmost importance for adequate digestion.

The ileo-cæcal sphincter begins to relax at infrequent intervals some time after the arrival of chyme in the end of the ileum, but only when another meal is taken does peristalsis occur at all actively in the extreme end of the ileum in addition to segmentation; the sphincter apparently relaxes as each peristaltic wave reaches it, a great part of the contents of the end of the ileum being rapidly squirted into the cæcum. In spite of this, if ordinary meals are taken after a bismuth meal, so that some bismuth remains in the stomach (in the manner already described) until the evening, the shadow at the end of the ileum may be visible in normal individuals until late at night.

The normal iliac stasis is increased in all conditions leading to spasm or to the inhibition of the normal relaxation of the ileo-cæcal sphincter. In acute appendicitis, for example, the sounds which normally indicate the squirting of the contents of the ileum into the gas-containing cæcum cease completely, owing probably to spasm of the sphincter, which Elliott has shown occurs in animals when the splanchnic nerve is stimulated or the neighbouring peritoneum is irritated. In chronic appendicitis similar, but less marked delay often occurs. One of the most striking cases of iliac stasis I have ever seen was in such a case. Six hours after the bismuth meal the stomach was empty, but no bismuth was present in the cæcum, all of it having collected in the end of the ileum, though in the average normal individual the shadow by this time would have reached the hepatic flexure. The last few inches of the ileum could be clearly defined, as they were distended with chyme. Palpation under the screen showed that there were no adhesions, the whole of the ileum being freely movable. Twenty-four hours later some bismuth was still present in the last inch and a half of the ileum and a little in the cæcum and ascending colon, all the rest having passed to the rectum, from which some bismuth-containing fæces had just been evacuated. It was clear, therefore, that the only stasis in this patient's alimentary canal was in the end of the ileum. Mr. Rowlands operated and removed a diseased appendix, but found that the ileum was perfectly normal and was free from adhesions. The ileo-cæcal junction was not abnormally narrow, so that it was clear that the stasis could have been due to nothing else than inhibition of relaxation or spasm of the ileo-cæcal sphincter.

The end of the ileum, as it rises from the pelvis to join the cæcum, bends in various directions; as the shadow on the screen is in one plane only, it is natural that it often appears as if it formed one or more acute angles or kinks. It is obvious, therefore, that no conclusion of any sort can be made from skiagrams alone as to the presence or absence of kinks. By palpating the abdomen during the examination, it is easy to demonstrate that most of these "kinks" are only apparent. Sometimes the ileum appears to be fixed where it crosses the pelvic brim, but by manipulation the whole loop can be raised out of the pelvis, and the apparent adhesions and kink disappear. Recently I saw what appeared to be a typical iliac kink, the ileum being fixed when it passed over the brim of the pelvis; no amount of manipulation had any effect upon it, but on inflating the bowel in carrying out Bastedo's test for appendicitis the distended pelvic colon lifted the last part of the ileum completely out of the pelvis, and palpation now showed that there were no adhesions and no kinks. I suggest, therefore, that before a diagnosis of an iliac kink be made with the X-rays the colon should be inflated, if palpation has failed to separate the apparent adhesion.

Although there can, of course, be no doubt that abnormal bands may occur in connexion with the terminal portion of the ileum, I do not feel at all convinced that they are of any importance except in quite exceptional cases, in which clear evidence of antecedent attacks of appendicitis can almost invariably be obtained. The obstruction offered by a genuine iliac kink is hardly ever as great as that normally offered by the ileo-cæcal sphincter, 3 in. further on. Thus in the skiagrams published to illustrate the iliac kink the lumen is not always even narrowed. When it is narrowed, there is nothing to show that the narrowing does not represent a division produced by the segmentation, which normally occurs with great activity in the end of the ileum. Nothing short of almost complete stenosis could lead to stasis of any importance, as the contents of the ileum are always so fluid that they can pass through a very narrow passage without difficulty. A true iliac kink, if it ever really leads to iliac stasis, probably does not do so directly, but indirectly by inhibiting the relaxation of the ileo-cæcal sphincter, just as gastric adhesions may upset the pyloric mechanism.

It has been suggested that iliac adhesions ought to be surgically treated at the same time as an operation is performed for a duodenal ulcer, for gall-stones, and for appendicitis. But in my experience the removal of the gall-stones and drainage of the gall-bladder almost invariably lead to such complete and permanent relief of symptoms that any further operation done at the same time could not improve the results. In the same way gastro-enterostomy completely cures a duodenal ulcer in a considerable proportion of cases, and if the appendix is at the same time removed the chances of recurrence are exceedingly small. I can hardly imagine that the results would be any better if iliac kinks were also dealt with, and there can be no doubt that the division of bands and membranes in such cases may lead to peritonitis, as Mr. Lane pointed out in his opening address; for I have myself performed the post-mortem on a patient with duodenal ulcer, in whom death had resulted from post-operative infection in the right iliac fossa, when adhesions in this neighbourhood had been divided in addition to performing gastro-enterostomy. Again, the removal of the appendix in acute appendicitis, if it is done sufficiently early, is rarely followed by any symptoms which could be ascribed to iliac kinks. It is true that the removal of the appendix in the later stages of acute appendicitis and in chronic appendicitis is not infrequently followed by pain in the right iliac fossa, and sometimes by other abdominal symptoms; but I have also seen this occur when an iliac kink had been simultaneously dealt with.

Conclusion.—Iliac stasis is a normal phenomenon and results from the action of the ileo-cæcal sphincter. Iliac kinks are comparatively rare, and do not directly increase the normal iliac stasis. The normal iliac stasis may be increased by spasm or absence of relaxation of the ileo-cæcal sphincter as a result of disease in the neighbourhood of the cæcum, including perhaps iliac kinks.

KINKS AT THE HEPATIC AND SPLENIC FLEXURES, PTOSIS OF THE COLON, AND COLONIC STASIS.

In normal individuals examined with the X-rays the hepatic and splenic flexures almost invariably appear to be acutely kinked. This again is the result of the shadow being cast in a single plane, as the limbs of the flexures belonging to the transverse colon are in front of those of the ascending and descending colon respectively; the flexures consequently appear to be acute, although they form a wide angle when looked at from the side. It is almost always possible to determine when adhesions are present between the limbs of the hepatic and splenic flexure by seeing whether they can be separated by palpation during the X-ray examination. In my experience it is very uncommon to find that they cannot be separated sufficiently to make it obvious that no great mechanical hindrance to the passage of the fæces can be present.

A pelvic cæcum and a dropped transverse colon are often referred to as if they were invariably associated with constipation. I believe, however, that in a large majority of cases they are quite independent conditions. I have certainly seen a pelvic cæcum just as frequently in strong, healthy individuals with perfect digestion and no constipation as in constipated patients. Moreover, I have never been able to convince myself, when a pelvic cæcum was associated with constipation, that there was any causal relationship between the two conditions, as in the majority of cases dyschezia without delay in the cæcum or in any other part of the colon was present. Ptosis of the transverse colon never leads to kinking at the hepatic flexure, which invariably drops with it, but a very movable right kidney may produce obstruction in such cases. A definite sharp kink is occasionally produced at the splenic flexure, as the phreno-colic ligament is stronger than any other of the intestinal attachments. In one case, to which I shall again refer, the kink was so marked that a short-circuiting operation was required, and the transverse colon, which was situated low down in the true pelvis, was anastomosed with the pelvic colon, with which it was already in contact. When constipation is associated with visceroptosis, the former is generally due to dyschezia, which results from inefficient defæcation owing to weakness of the abdominal and pelvic muscles, this weakness being also the cause of the visceroptosis.

Most cases of constipation are more or less cumulative, the bowels never being completely evacuated, so that more and more fæces are retained. In dyschezia, for example, the rectum is never emptied, and an accumulation of hard fæces forms successively in the pelvic colon, descending colon, and even the transverse colon. If, therefore, a bismuth meal is taken without first thoroughly evacuating the bowels, the bismuth travels with more or less ease as far as the beginning of the fæcal accumulation, beyond which it passes with extreme slowness, corresponding with the rate of excretion of the most distal part. If, however, the colon is first thoroughly evacuated by means of enemata, it is found in such cases that the bismuth often reaches the rectum in the normal time, but owing to difficulty in defæcation it is retained there. It is only possible to distinguish such cases of dyschezia from true colonic constipation, in which delay occurs in the colon itself, by emptying the bowels completely by enemata before the examination begins. Numerous mistakes in diagnosis have resulted from neglect of this precaution.

My investigations, both with the X-rays and by other means, have led me to realize that in a large majority of instances only part of the colon is affected in constipation. Thus in nearly half of the severe cases I have seen in consultation, the rectum, or less commonly the pelvic colon, was affected alone, the condition which I have called dyschezia being present; this can generally be treated with success by re-educating the patient to defæcate by regular habits, exercises for the abdominal and pelvic muscles, and graduated enemata. In rare cases the difficult defæcation depends on pelvic adhesions, and in one such case their division combined with a short-circuiting operation, performed by Mr. Lockhart Mummery after medical treatment had failed, produced complete relief. In other cases the cæcum and ascending colon are alone affected; as soon as the fæces get into the transverse colon their passage to the rectum is normal in rate. Such cases can generally be relieved by diet and drugs, with the aid of massage applied regularly for a prolonged period to the affected part. I have not myself seen a case in which I thought an operation was indicated, but if all medical treatment failed, it seems to me that an appendicostomy or, if that proved unsuccessful, an anastomosis between the ileum and the transverse colon, with or without the removal of the cæcum and ascending colon, as successfully performed by Wilms and de Quervain, would be more rational and less dangerous treatment than ileo-sigmoidostomy combined with complete colectomy. In another group of cases the passage as far as the splenic flexure is normal in rate, but considerable difficulty is experienced in getting beyond this point. This is sometimes due, as I have already pointed out, to severe ptosis of the transverse colon, the splenic flexure remaining fixed, and sometimes to unexplained muscular weakness of the distal part of the transverse colon. If such cases are examined by means of a barium enema, it is generally found that the fluid run in from the rectum passes without difficulty into the transverse colon; moderately large enemata without the aid of drugs or other treatment, with the exception of an abdominal support if ptosis is present, are then often successful. In two of my cases, however, all medical treatment failed to give relief, and Mr. F. J. Steward effected an anastomosis between the lowest part of the transverse colon and the pelvic colon in one instance, and between the two limbs of the splenic flexure in the other with satisfactory results.

The comparatively rare cases, in which the whole of the colon is involved, are generally amenable to treatment by diet, massage, and drugs. I have up to now only seen a single case out of the very large number I have examined with the X-rays, in which delay occurred in the whole of the colon and in which, in spite of every form of medical treatment I could devise, the patient became steadily more and more poisoned and suffered from more and more pain. Mr. Arbuthnot Lane was good enough to operate on this case for me in December, 1911, and the relief, which was almost instantaneous, has proved permanent and complete. I am therefore convinced that when the whole of the colon is involved and when thorough and prolonged medical treatment has proved completely ineffective, but under no other circumstances, the operation of ileo-sigmoidostomy is the only rational treatment.

Conclusion.—Ptosis of the intestines does not lead to stasis except in rare instances at the splenic flexure, a pelvic cæcum and a pelvic transverse colon being compatible with perfect health and normal intestinal action. In most cases of constipation a single part of the bowel, such as the rectum, pelvic colon, ascending colon, or splenic flexure, is involved, and treatment should, therefore, be directed to the affected part only. In those cases in which the whole of the colon is involved medical treatment almost invariably succeeds, but when it does not, ileo-sigmoidostomy, with or without colectomy, is indicated.

Mr. F. COLEMAN: The odontological aspect of this subject is concerned chiefly in the sources of alimentary toxæmia, but also has an important bearing on its consequences and treatment. My remarks will be largely restricted to the mouth as a source of alimentary toxæmia. The mouth acts as a source of alimentary toxæmia only in an indirect manner—viz., by the passage of its contents into the stomach and intestine, for little or no absorption takes place by means of its own lining membrane.

According to Dr. Andrewes's definition of alimentary toxæmia, which has been largely accepted in this discussion, "actual bacterial invasion of the blood" is excluded; and it is therefore assumed that the lining membrane of the alimentary canal remains intact, and absorption takes place by some process akin to osmosis, assisted, no doubt, by the physiological activity of the columnar cells. I mention this definition because, in stating how the mouth may act as a source of alimentary toxæmia, I refer to certain lesions of this cavity for the purpose of describing how their secondary effects—viz., impairment of mastication and increase of the bacterial contents of the mouth—may play an important part in alimentary toxæmia. Granting this premise, the mouth forms a source of alimentary toxæmia in three ways:—

(1) Lesions of the buccal cavity, which largely fall into two classes, lesions of the teeth and lesions of the gums.

(2) Imperfect mastication, which may be due to inefficient teeth or want of teeth.

(3) Mouth-breathing.

There is some overlapping of these classes; thus a tender tooth (i.e., periodontitis) is also a more or less functionless tooth, and so may form a cause of alimentary toxæmia in two ways—viz., by the discharge from the gum, and by its inefficiency as a masticatory organ; still, some such classification serves a useful purpose in bringing our facts together. The first class—i.e., lesions of the buccal cavity—may allow of direct bacterial invasion of the blood, but at the same time some of the bacteria or their toxins are shed into the mouth and swallowed.

We must now consider what are these lesions. The pulp of a tooth, as long as it remains alive, can exert a defensive action against the invasion of micro-organisms, and if teeth could always be *filled* at this stage, conservative dentistry would be a satisfactory branch of healing; but when once the pulp is involved by caries, and its removal (i.e., the pulp) thereby necessitated, conservative work must be regarded as a compromise—the compromise being between retaining a tooth that can be rendered functional and retaining a tooth that is an irritant to the tissues, for a pulpless tooth, being a pathological condition, must, and does, act as an irritant. Without going into too much technical detail, it may be stated that the mucous membrane around a pulpless tooth is rarely healthy; the tooth itself is almost invariably slightly tender to percussion as compared with a sound tooth, and radiography usually shows some rarefaction of bone around the tooth and frequently some exostosis of its root, all of which are indicative of tissue irritation —to which may be added the frequent association of hyperplasia of the lymphoid tissue of the neck. As a large part of conservative dentistry consists in retaining pulpless teeth in the mouth as abutments for fillings, crowns, and bridges, it becomes a difficult question to know how far we are serving the patient's best interests in carrying out this type of work. It is in such questions as these that a free interchange of opinions between the various branches of medicine is of the utmost value.

The effect of diseased teeth has only within recent years been recognized as a fruitful source of alimentary toxæmia, so that many people still regard the absence of discomfort in a tooth as tantamount to its harmlessness. Unfortunately, this teaching has not been appreciated by all practitioners, so that the difficulty is increased of persuading patients to part with teeth that are injurious to their health. The appearance of many of these teeth after their removal, some of which may have supported artificial crowns and "bridges," should form sufficient evidence of their injuriousness.

The speciality of dentistry being a rather restricted one, there is a tendency to judge the harmfulness or otherwise of a tooth by the local conditions alone, besides which there may be great difficulty in correctly assigning to the teeth, as cause and effect, signs and symptoms of disease in other parts of the body.

Carious cavities in teeth that have not involved the pulp (Miller has shown that micro-organisms only penetrate the dentinal tubes to $\frac{1}{10}$ mm. or $\frac{1}{250}$ in.) are a potential source of alimentary toxæmia, although their contained bacteria may have produced no inflammatory reaction on the tissues of the mouth, for these cavities act as incubating chambers, and greatly add to the bacterial content of the mouth and consequently the number of bacteria swallowed. Many of the bacteria that are swallowed become disintegrated and set free intracellular toxins.

There can be no doubt that many of the patients we see with suppurating gums, owing to carious teeth or pyorrhœa alveolaris, develop an immunity to the causative organism or organisms present; and if we could imagine such a condition transferred to a healthy person the result would, without doubt, be very serious, even if not fatal. Caries being a slow and progressive disease which rarely affects all the teeth simultaneously, the patient is able to develop antibodies which in some way neutralize the toxins absorbed from the teeth and gums—a greater development of toxins calling forth a greater output of antibodies, provided the patient's resistance is able to withstand the increased poisoning. This fact is frequently demonstrated by the improvement that results from general treatment, in spite of the fact of the local condition remaining the same, and by the employment of vaccines that raise the local resistant power of the tissues. It is suggested also in a converse direction—viz., by the impairment of health that results if the local condition be suddenly ingrafted on the patient, as in mercurial stomatitis.

If we consider the large area of infection that may result from a suppurative periodontitis involving several tooth-sockets (roughly a square inch per socket), the correlating discharge from such an area, the favourable incubating ground formed by a tooth-socket for the development of micro-organisms and their toxins, and the mechanical difficulties it offers for efficient drainage, is it to be denied that the mouth may be an important source of alimentary toxæmia? A gingivitis will sometimes directly infect the mucous glands of the cheek that lie opposed to the teeth, the mucous membrane appears stippled with greyish-white, opaque nodules, which feel firm and shotty, and are arranged in a linear manner corresponding with the festooned margins of the gum which they overlie. This condition to my knowledge was first noted or described by Goadby and, although I have since observed the condition on a few occasions, it is far from generally present. Goadby has found these glands to contain *Staphylococcus viscosus*, and states that the glands so affected are liable to ulcerate and form small painful ulcers. If it be possible for the mucous membrane of the cheek to become ulcerated merely by contact with a source of infection, it does not seem unreasonable to infer that similar changes may occur from a like cause elsewhere in the alimentary canal.

The second way in which the mouth may act as a source of toxæmia is by inefficient mastication. If mastication, and consequently insalivation, be imperfectly performed, food is swallowed before being thoroughly broken up, and is, therefore, less permeable to the gastric and intestinal juices, while the carbohydrate portion is only partly, if at all, converted into maltose. The effect of this increased bulk of food in the stomach, together with the unchanged carbohydrate, is to throw more work on the stomach and so impair digestion, besides which the bactericidal property of the gastric juice is rendered less effective on those bacteria swallowed. The detergent effect of fibrous foodstuffs and the scouring action of the tongue and lips is correspondingly diminished by defective mastication, so that debris of food, mucus, and micro-organisms collect on and between the teeth. The micro-organisms placed under such favourable conditions rapidly multiply and thus increase the number swallowed, while the starchy portion of the retained debris is converted into acid, thereby affecting the teeth, and so establishing a vicious circle. The deficient blood supply to the teeth and gums resulting from inefficient mastication renders these tissues less resistant to the invasion of micro-organisms. The artificial means of cleansing the mouth are insignificant in their value as compared with those brought about by efficient mastication.

The third way in which the mouth may act as a source of alimentary toxæmia is brought about by mouth-breathing. Mr. Colyer has mentioned one factor connected with this—viz., a gingivitis in the incisor region. The mouth, like the nose, is not lined with a ciliated epithelium, nor provided with an olfactory sense, both of which ensure some purity of the air inhaled. The nasal passages are physiologically adapted for warming the inspired air by their uneven surface and free blood supply, and the inhalation of cold air, which may also be impure, is, without doubt, an important factor in the hyperplasia of the lymphoid tissue around the fauces, and thus an indirect source of alimentary toxæmia.

CONSEQUENCES OF ALIMENTARY TOXÆMIA.

I will not refer to the various gastro-intestinal disturbances or "neurasthenias," as they have been termed, that may result from the continued swallowing of infected material from the gums—these have been fully dealt with by Hunter and others—except to suggest that probably more harm is done during the sleeping hours, when the gastric juice is not being secreted and its bactericidal property, therefore, in abeyance.

TREATMENT OF ALIMENTARY TOXÆMIA.

I have already indicated how a tooth becomes an irritant to the tissues, and an indirect source of alimentary toxæmia, when its pulp is infected with micro-organisms, and that subsequent treatment must be regarded as a compromise.

I am inclined also to be in agreement with Mr. Colyer in his statement that the only reliable treatment of sockets affected with pyorrhœa alveolaris is the removal of their contained teeth. The corollary of these two statements is that only by the removal of pulpless teeth and teeth affected with pyorrhœa alveolaris can these possible sources of toxæmia be eliminated.

I should like to make a comment on the statement made by Dr. Hale White in his opening address, with reference to the removal of teeth affected with pyorrhœa alveolaris. He states, and with good reason: "It is dangerous to remove too many affected teeth at once, for a rapid absorption of micro-organisms may take place by the damaged vessels." This statement is correct as far as it goes, but does not take into consideration the liability of the sockets of the extracted teeth becoming further infected or reinfected by the remaining "pyorrhœic" teeth; and although the wound is more extensive if all the affected teeth be removed at one operation, there is the compensatory advantage of rendering the buccal cavity in a more favourable condition for healing.

Dr. GUSTAVE MONOD (Vichy): I propose to limit my remarks to the special view of alimentary toxæmia taken by the so-called Vichy school, and in doing so I refer, as you may suppose, to the brilliant work of Glénard on the relationship between toxæmia and enteroptosis. So extensive, however, is this subject that I am compelled in the short time at my disposal to deal with conclusions only. These are: Toxins circulating in the blood of patient A., and causing great disturbance might, if run into the system of patient B., do no harm whatever, thus showing that the soil is as important as the seed. In many cases, indeed, as Dr. Hale White pointed out, the final outcome depends on hepatic adequacy. By *hepatism* we mean a diathesis, hereditary or acquired, characterized by inadequacy of the liver cells. A vicious circle is thus created, the toxins set free reacting in turn on the cells of the liver. Disturbance of the intrahepatic circulation follows, and, in sequence to this, there arises a like disturbance of circulation in the bowel. The subsequent effects observed in the bowels are diminution of muscular tonicity, and lowering of the intraperitoneal pressure. Reduction of the lumen of the gut, well marked in the transverse colon, follows, after which enteroptosis begins. The first abdominal organ to descend is the hepatic flexure of the colon. A kink is formed and the cæcum is then found distended and palpable. The second organ to feel the effects of ptosis is the right kidney. Then in order the stomach, the left kidney, the liver, and the spleen. This, the complete picture of enteroptosis, is a very rare condition; for usually, according to the teaching of Glénard, the syndrome concerns only the colon and right kidney. In passing I wish to emphasize that Glénard holds strongly that movable kidney is, in most of the cases, a sequel to toxæmia. I am only sorry that I cannot now discuss this assertion. Clinically, these patients are commonly diagnosed as dyspeptics, neurasthenics, or the subjects of calculosis, and are treated on these lines with little success. They require daily saline aperients, and, paradoxical as it may seem, a meat diet. They should wear a belt which effectively supports the abdominal organs. One way of attempting to break the vicious circle is by means of spa treatment, and in this connexion we believe that some, at any rate, of the natural alkaline waters exercise a specific action on the cells of the liver.

The Royal Society of Medicine.

April 21, 1913.

Sir Francis H. Champneys, Bt., President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.[1]

Alimentary Toxæmia from the Surgical Point of View.

By P. Lockhart Mummery, F.R.C.S.

In his opening address Dr. Hale White pointed out that we know very little about so-called "Alimentary Toxæmia," and that even the name is a bad one. With that opinion I thoroughly agree. We are, I think, only on the borderland of knowledge in respect of this very interesting condition, and it is sincerely to be hoped that we shall have some real facts brought out during the progress of this discussion which will aid us in building up a sound knowledge of the subject. Medicine and surgery, we all trust, will become exact sciences in course of time, and one of the first essentials of science is that nothing shall be taken for granted, everything must be proved beyond all question.

In studying this condition, one is struck by the fact that in the more severe cases the patient seems quite unable to deal with the poisons generated in the colon, and the least amount of delay causes toxic symptoms, such as depression, nausea, headache; while in the less severe cases it is only when stasis has been present for some days that there are acute symptoms. I think the probable explanation of this is that the liver, which is the organ that habitually deals with these toxins, becomes damaged after a time in the same way that the kidneys get damaged in chronic alcoholism, and then it allows the toxins to get straight through into the blood. It is to be hoped that some of the pathologists will make a study of the condition of the liver in these cases.

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[1] Adjourned from April 14.

I have read with interest Mr. Arbuthnot Lane's introductory paper, and I think he attaches too much importance to the effects of gravity in producing secondary changes in the peritoneal attachments of the large bowel. I agree with Mr. Lane in thinking that the adhesions so often seen are only seldom inflammatory in origin. Personally, I believe that they owe their origin to chronic constipation and overloading of the bowel in earlier life, and that if more attention were paid to the rational treatment of constipation in early life these cases of chronic stasis would be much less frequent. I cannot follow Mr. Lane's ingenious explanation of the formation of these adhesions, nor do I think it is correct. Like Mr. Lane, I have for a number of years been making a careful study of these curious bands, which one finds causing kinking of the bowel, and I have taken every opportunity of observing them. There is an important point in respect of these so-called adhesions which I have not seen previously mentioned—namely, that they are often not adhesions at all, but folds of normal peritoneum. It is a little difficult to explain exactly what I mean by this, but a reference to the diagram will make the point clear. When one cuts away, or strips up, one of these bands of adhesions, instead of finding, as one would expect, a raw surface beneath, one not infrequently finds another layer of normal glistening peritoneum. So that at the position of the band there are apparently four layers of peritoneum instead of one. I am not going to attempt to offer any explanation of this curious fact, as I think we cannot explain it until we know considerably more about the morbid physiology of the peritoneum. What I desire to point out is that these bands are not, as Mr. Lane assumes, mere thickenings in the normal peritoneum, but are actual double folds of this membrane. Apart from this, however, Mr. Lane seems to me to disprove his own case by admitting that these bands are found in young children in whom unquestionably the forces to which he attributes their origin have not yet begun to act. (Mr. Lane says he found these bands well developed in a child aged 2 years.)

The real cause of the patient's symptoms is that some poisonous material, of which we do not know the nature, is being formed in the alimentary canal, and is passing from it into the tissues via the bloodstream. It is obvious that we can remedy this in three ways, by preventing the formation of the poisonous material in the alimentary tract, by preventing its absorption, or by removing that portion of the alimentary tract in which it is formed. The first can often be done by alterations of diet—more often, I believe, than is generally supposed. The second can only be secured by not allowing the material to remain in the alimentary tract long enough for the formation of the poison. It must be remembered, however, that stasis is not necessary for the production of toxæmia, and that, on the other hand, one sees plenty of instances of severe stasis where no toxæmia ever occurs. This is best seen in those extraordinary cases of congenital dilatation of the colon where the bowels only act a few times in the course of a year, and yet I have not seen one of these cases suffering from toxæmia. I think it is also not sufficiently recognized that we may have toxæmia even in a marked degree without stasis. There can, however, be no doubt that there are a large number of cases of alimentary toxæmia in which all the evidence points to stasis as the cause of the condition. The relief of stasis in these cases by the removal of some obstruction, by keeping the large bowel washed out through an appendicostomy opening, or by

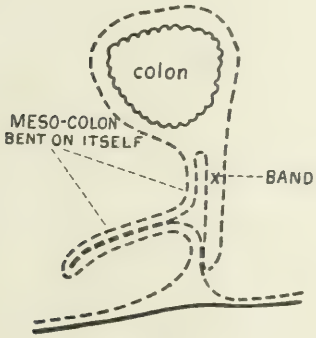

FIG. 1.

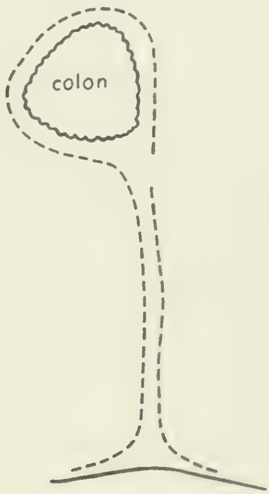

FIG. 2.

Fig. 1.—Diagram showing band produced by double fold of peritoneum adherent to base of meso-colon. (Peritoneum = dotted line.)

Fig. 2.—Diagram showing same after band has been cut away and meso-colon allowed to straighten.

short-circuiting the large bowel, does result in a cure of the patient and the disappearance of all the symptoms.

As my contribution towards this discussion I shall bring forward a few cases of well-marked alimentary toxæmia which have been treated by operation. I do not propose to recount a long series of abbreviated notes of cases as is often done under such circumstances, but only a few of the more interesting, as it seems to me that at present there is more to be learnt from the study of a few quite characteristic cases of which fairly full particulars are obtainable than from a number of cases many of which are incomplete, and some of which are complicated by other conditions. I have, therefore, deliberately selected some of the most interesting cases from my note-books instead of taking them in sequence. I think we often learn most about a disease from the extreme cases, and as the following case is the most severe example of the condition that I have ever seen, I venture to bring it before you; for although it cannot be claimed to be successful in any sense, I think it is instructive (Case I).

Some of the most interesting and important cases of auto-toxæmia are those in which there are associated joint lesions of the nature of semi-acute or chronic arthritis. I believe that many of the cases of crippling arthritis that we see from time to time are due to poisons formed in the large bowel. I have seen several cases in which arthritis, both of the chronic and semi-acute variety, was associated with stasis and toxæmia. In several of these there was definite lipping of the cartilages and a tendency to destruction of the joints. There is, I think, every probability that by operating upon some of these cases at an early stage we shall be able in the future to arrest the disease before serious destruction of the joints has taken place. There is, however, much more work to be done on this important subject before we can make any authoritative statements.

As regards the choice of operation, this must depend on the cause of the stasis, and everything possible should be done to arrive at a correct diagnosis. As Case III shows, it is very easy to miss the cause of the trouble. This lady had had her abdomen opened on two previous occasions by first-rate surgeons without the lesion being found. It is first necessary to exhaust every means at our disposal with a view to ascertaining the cause of the stasis. The sigmoidoscope has in my experience been of the most use in this respect, and has afforded absolutely invaluable information in numerous instances. X-ray examination after a bismuth meal or enema, and examination under an anæsthetic are also valuable means of arriving at a correct diagnosis. Examination of the stools has, in my experience, proved of very little value, and often most misleading. The amount of delay in the passage of the food residue is, I believe, best tested by a charcoal lozenge while the patient is on ordinary diet, the stools being watched for the first appearance of the charcoal. I think this will give quite as valuable information as an X-ray test meal. I attach no importance to the point in the bowel at which delay appears to occur with the latter method, the appearances of the bowel as seen with the X-rays being alone of value.

When there is a definite obstructing lesion, such as a band of adhesions, a stricture, &c., this should be removed by appropriate means. When there is no obstructive lesion, our choice lies between appendicostomy, which will allow the large bowel to be kept washed out and free from accumulation until it has recovered its proper function, and short-circuiting with or without removal of the colon. In my experience appendicostomy with daily lavage of the colon has given quite excellent results. A good example of this is Case III, and others will be found in the appended list. It has the merit of being almost free from risk, and of not leaving the patient with a condition which may cause subsequent trouble, and of not depriving him of any portion of his alimentary tract. For these reasons it should, I think, be the operation of choice, and the more serious operation of short-circuiting should be reserved for those cases in which appendicostomy fails to give relief, or in which there is serious reason to suppose that an appendicostomy alone will not relieve the patient. I have personally a serious objection to a short-circuiting operation which leaves the patient with a large bowel blind at one end. If the operation is performed by implanting the ileum into the sigmoid or rectum, I prefer to perform an appendicostomy at the same time in order to allow of the large bowel being kept empty ; or else to perform ileo-sigmoidostomy by lateral anastomosis, but with a sufficiently large opening to ensure quite an easy passage from the ileum into the rectum, the opening being at least 3 in. long.

With regard to the question of colectomy, I have never personally seen a case in which I considered this operation justifiable, where the patient was not already so ill as to negative the operation being performed.

I think it is a mistake to treat all cases alike by a single operation, irrespective of the cause of the stasis. The proper method of dealing with these cases, in my opinion, is first to ascertain the cause of the stasis, and, where possible, to deal directly with that; and only to perform appendicostomy or short-circuiting in those cases where the cause of the stasis is a general atony of the bowel, or where one cannot directly treat the cause of the delay successfully. My own experience of the results of short-circuiting has been rather disappointing, several of the patients having failed to be completely cured. By far the best results that I have obtained have been in cases where I have removed obstructing bands or performed appendicostomy.

There is an interesting point in connexion with these cases which is worthy of mention. I have noticed that acetonuria, or delayed chloroform poisoning, is more often seen in these cases after operation than in cases where alimentary toxæmia is not present. I have had no less than three such cases, one of them fatal, and I think it is advisable to administer dextrose in all such cases before operation.

In conclusion, I should like to say a word as to the after-treatment. I consider this of the utmost importance. Quite often where an operation has been performed the patient is expected to get well without any attention beyond that necessary to ensure proper healing of the wound. My experience has been that these cases require most careful supervision during what I may describe as the convalescent stages. They are usually neurasthenic and have lost confidence in themselves; at the slightest sign of recurrence of the old symptoms they get depressed. They often expect, as soon as they feel better to be able to do anything and eat anything. I think they should always be kept under most careful medical supervision for three months, and longer than this if their condition of health is not normal. In many cases I have found the greatest benefit from cutting the patient off all nitrogenous food for a time. The exact reason why a non-nitrogenous diet suits these cases best I must leave to my medical colleagues, but I have certainly seen excellent results from the adoption of this diet in the after-treatment of cases of alimentary toxæmia which had been operated upon. Exercise and massage for long periods are often indicated also. In a successful case the appendicostomy can be closed, but plenty of time should be allowed to elapse before this is done.

LIST OF CASES.

Case I.—A man, aged 47, had been very seriously ill for two years with constipation and occasional attacks of diarrhœa. He had lost flesh to such an extent that he was a mere skeleton. His skin was of a dark grey colour, and had a most unpleasant smell which nothing would remove. The subcutaneous fat had entirely disappeared, and there was considerable cutaneous pigmentation. The abdomen was greatly distended. There was never any proper action of the bowels, and medicines and enemata gave no real relief. There was no blood in the stools, but some mucus, and the urine contained indican. He had a subnormal temperature and a slow pulse. Operation: The whole of his intestines were much dilated and extremely thin, but no other lesion was found. Appendicostomy was performed in the hope of clearing the toxins from the colon. The wound refused to heal at all, though it remained aseptic, and in spite of treatment the dilatation of the bowel persisted. The bowels at last refused to act, even when the colon was filled with water, and although the small bowel was opened, he died, apparently from complete inactivity of the intestines and extreme toxæmia some three weeks later.

Case II.—A gentleman, aged 50, whom I saw in July, 1910, had been suffering from chronic constipation for a considerable period. This had steadily got worse in spite of treatment. He had been losing weight, was much wasted, and had an earthy-coloured skin which was much shrunken and very dry. He appeared prematurely old and was a complete invalid. He had all the symptoms of auto-intoxication, and his arteries were hard and easily rolled under the fingers. He looked so ill that it seemed unlikely that he would live more than a few months. He was under excellent physicians who had tried all the recognized forms of treatment, including massage and dietary, and the proper administration of enemata. After consultation appendicostomy was performed in the hope that he would recover if the bowel was kept properly washed out. He made an exceedingly rapid recovery, was about again in a few weeks, and began to put on weight fast. He gave up eating meat in any form. The appendicostomy opening was closed a year later. Eighteen months after the operation he was in better health and weighed more than ever before in his life, and at the present day he is one of the healthiest men for his age that I know.

Case III.—A lady, aged 26, was sent to me with a history of increasing constipation. She had had severe abdominal pain for which morphia had to be given, and bleeding from the bowel. There was a seven years' history; but the patient had been much worse for the last two years, and had become a complete invalid. She had been for long periods in nursing homes under excellent physicians, and every kind of treatment had been used. Right-sided colotomy had been performed eighteen months previously, and an exploratory laparotomy a year before that. She was, however, no better, and only weighed 6 st. 3 lb. Her temperature was always raised at night, and for nine months it had varied between 102 and 103° F., with rigors every two or three days. She passed blood from the rectum occasionally. The blood examination showed red corpuscles, 3,830,000; white, 10,000; haemoglobin, 35 per cent.; polymorphonuclear leucocytes, 65·5; eosinophiles, 5. She had been treated with both toxins and serums without effect. A sigmoidoscope examination revealed a stricture due to pericolitis in the sigmoid flexure. Operation: A hyperplastic tubercular stricture was removed from the middle of the sigmoid.

Resection and end-to-end anastomosis was performed. There was a complete recovery. The colotomy was closed a year later. The patient is now quite well and weighs 11 st.

Case IV.—A lady, aged 50, was brought to me on account of intractable constipation, with frequent fæcal impaction, only relieved with difficulty. She had the usual symptoms of toxæmia. She had been treated at various spas, and by her own doctor for a considerable time. Sigmoidoscopy revealed kinking of the colon, and there was a history of a previous operation for ovarian cyst. Extensive adhesions were found to the abdominal wall, and between the sigmoid flexure and left iliac fossa. These were divided and the bowel freed. Her doctor writes eighteen months later to say that she is in good health and much benefited by the operation.

Case V.—A lady, aged 42, had suffered from constipation almost all her life, which of recent years had become much more severe. She had an earthy-coloured skin, was extremely thin, suffered constantly from pain in the abdomen, headaches and sleeplessness, and was neurasthenic. She was a complete invalid, and had threatened to commit suicide. Her bowels were never opened except after repeated large enemata. On my advice an appendicostomy was performed. This gave some relief for a time, but the patient still remained far from well. A year later I performed ileo-sigmoidostomy, and although it would not be right to say she is cured, she is certainly relieved of the more serious of her symptoms, and there are hopes of her ultimate complete recovery.

Case VI.—A lady, aged 32, had suffered from severe constipation for twelve years. At the time she was brought to me she had become very much worse, and no drugs would give her relief. Enemata often failed to act and had to be repeated two or three times. There was chronic abdominal pain, loss of weight, mucus in the stools, headache, nervousness, loss of appetite, and sleeplessness. Appendicostomy was performed. This gave great relief, and the patient put on weight and got practically well. All the symptoms of auto-intoxication disappeared. Five years later she still had to use the appendicostomy opening, but enjoyed fairly good health and was of normal weight.

Case VII.—A lady, aged 31, was sent to me from Australia. She had been an almost complete invalid for ten years. There was severe constipation, alternating with occasional attacks of diarrhœa, abdominal pain, tenesmus, loss of weight, and complete inability to lead an active life. She had been treated by massage, aperients, diet, &c., for long periods. A diagnosis of retroflexed uterus and adhesions of the pelvic colon was made, after consultation with a gynæcologist, and it was decided to operate. I found a sharp kink in the middle of the pelvic colon due to adhesions, and a retroflexed uterus blocking the pelvic outlet. The adhesions were divided and the uterus fixed in the normal position. The patient got perfectly well and has written since from Australia to say that she now leads an ordinary active life.

Case VIII.—A servant girl, aged 19, had suffered from severe constipation since childhood. For the last three years she had been so bad that she was unable to earn her living as a domestic servant. No aperients were of any use, and enemata frequently failed to give her relief. She had marked symptoms of auto-intoxication. Appendicostomy was performed, and the patient instructed how to keep her colon washed out. The symptoms entirely disappeared, and the bowels acted quite regularly as the result of daily washing out through the opening. In spite of repeated attempts it was, however, impossible to get the bowels to act without utilizing the appendicostomy opening, and three years later there had been no improvement in this respect. The patient was, however, in good health and able to earn her living.

Case IX.—A man, aged 42, was sent to me at the hospital complaining of severe and increasing constipation, accompanied by abdominal pain, wasting, headache, and complete inability to lead an active life. The stools were apparently normal, but both aperients and enemata failed to give relief with any certainty. On opening his abdomen a stricture due to pericolitis, nearly 4 in. long, was discovered in the sigmoid flexure, this portion of bowel being adherent to everything in the neighbourhood. With considerable difficulty it was resected and a temporary colotomy performed, it being intended to close the colotomy at a later period. The patient made a complete recovery and was so pleased with the improvement in his health, which had resulted from the regular action of his bowels through the colotomy opening, that he has ever since refused to have a second operation for its closure.

Case X.—A gentleman, aged 50, was seen by me in 1910. He had suffered from severe constipation for the last four or five years, which had gradually been getting worse, at times almost amounting to obstruction. He had constant pain in the abdomen, headaches, sleeplessness. His appendix had been removed without, however, giving him relief. His bowels were only relieved by frequent enemata ; at times he passed large mucous casts. He had been under medical supervision for the last two years and had been treated by massage, &c. There was tenderness on the right side of the abdomen, and the pain was referred to this position. On operating, the ascending colon and the hepatic flexure were found to be involved in adhesions. These were carefully divided and the bowel freed. The patient developed pneumonia after the operation, but recovered. The bowels began to act regularly immediately after the operation and have done so ever since. At the present time, three years later, he has had no recurrence of the original symptoms, but remains in perfectly good health.

Case XI.—A lady, aged 31, had suffered from severe constipation all her life, and had never known what it was to feel well. She was much below the normal weight, suffered severely from headaches, and always had discomfort in the abdomen. The bowels only acted as the result of large and repeated enemata. A sigmoidoscopic examination revealed kinking of the pelvic colon, apparently due to adhesions. I advised that a thorough course of medical treatment should be tried, and in the event of that failing she should be operated upon. Five months later the patient had become worse, and the bowels refused to act unless enemata were administered by a nurse and followed by massage. It was accordingly decided to operate. A tough band of adhesions completely kinking the pelvic colon was discovered and divided. The patient was afterwards treated by massage for two months, and made a complete recovery. I saw her three years after the operation, and she said that she enjoyed excellent health, the bowels gave her no trouble, and she was able to lead a normal life.

Case XII.—A man, aged 25, was sent to me with a history of severe constipation for some years. There was great difficulty in obtaining relief, even with drastic aperients. He always felt ill and depressed, and was unable to do his work, which was that of teaching, properly. There were well-marked symptoms of toxæmia. After consultation it was decided to try medical treatment for a year. This was very efficiently carried out, but at the end of that period there was no material improvement in his condition. An operation was then performed, and marked kinking of the pelvic colon causing a partial obstruction was discovered. This was divided and the bowel freed. The patient made a good recovery, and his bowels acted normally without any artificial aid. He returned to his work, and when last heard of, five years later, was in normal health.

Case XIII.—A lady, aged 51, when first seen by me had been practically bedridden for five years, and during the whole of that time had been under medical supervision. She complained of chronic pain in the abdomen and back, constant headache, giddiness, sleeplessness, loss of appetite, and complete inability to lead an active life. She was markedly neurasthenic. She suffered from constipation and had constantly to take aperients. Her skin was slightly discoloured and had an unpleasant odour, and her relatives said that there was always an unpleasant smell in her room. There was no loss of weight. There was commencing arthritis in the lumbar and sacral joints with some lipping of the cartilages, which was clearly seen in X-ray photographs. She had seen several physicians and had been treated by Plombières douches, massage, vaccines, and other medical measures without benefit. I performed appendicostomy, and the bowel was kept washed out daily. After the operation there was very marked improvement. Her complexion became normal, the odour of the skin disappeared, and all the other symptoms slowly cleared up. She reports herself from time to time, and when last heard of was in good health and able to get about in a normal manner. She still continues to make use of the appendicostomy opening, but this causes her no inconvenience.

Case XIV.—The patient was a young man, aged 23, of poor physique, very thin, and weighing nearly 2 st. under his proper weight. He had suffered severely from constipation for two or three years, the bowels only being relieved by large doses of medicine and enemata. He suffered from severe headaches, giddiness, lassitude, and pain in the abdomen. He was markedly neurasthenic. His skin was harsh and dry and his complexion sallow. He had been treated by massage, exercises, douching, and had been under excellent medical advice. A sigmoidoscopic examination demonstrated the presence of adhesions tying down the sigmoid flexure and causing partial obstruction. At the operation a band of adhesions causing a sharp kink in the middle of the pelvic colon was discovered and remedied. As a result of the operation the patient's bowels acted normally and the more obvious symptoms immediately disappeared. The patient slowly returned to a normal condition, but a sufficient time has not yet elapsed to enable one to state what the ultimate result will be.

Professor ARTHUR KEITH, F.R.S. : I appear before you this evening as a representative of that humble and somewhat neglected group of men whose business it is to impart a sound knowledge of the structure of the human body to medical students and medical practitioners. It seems to me that the present generation of anatomists have appeared at an unfortunate time when medical men in general and surgeons in particular have lost faith in human and comparative anatomy as a source of help in enabling them to solve the practical problems of applied medicine. I want, therefore, to place before you the anatomist's point of view—so far as it bears on the present discussion. During the last twenty years our teaching and our research have been greatly influenced by the various movements which have cumulated in the present discussion. In reality this discussion centres round the nature and function of the great intestine. I will deal first with the formation of peritoneal adhesions—a matter which has and which is receiving a great deal of attention from anatomists. We are at once brought face to face with one of the most striking of all the developmental changes seen during the growth of the fœtus. There can be no doubt that man comes of a stock in which the mesentery and bowel were originally arranged in a primitive and simple manner, for in the second month of development the human intestine and its mesentery have the lineal arrangement seen in the lowest mammals. Then during the third, fourth and fifth months of fœtal life a profuse adhesive process sets in—a regulated embryological peritonitis—which leads to the cohesion of mesenteries and viscera to the posterior wall of the abdomen—an adaption as we suppose to the upright posture, for it is only in those upright forms that we find these adhesive changes take place. After the fifth month the process of adhesion proceeds more slowly, and is completed about the time a child learns to walk. The extent of the process is extremely variable. How variable it is one can readily see by consulting recent papers by

Dr. Douglas Reid, the monograph by Dr. Henry M. W. Gray and Dr. W. Anderson, or the fine treatise on the appendix by Kelly and Hurdon. In about one newly born child out of every ten the process of adhesion will be found to have bound the mesentery of the lower part of the ileum to the pelvic fossa, giving the condition which is associated with ileal kinking. The extent to which adhesions are produced between the cæcum and ascending colon varies, but a condition which gives rise to a "Jacksonian membrane" is not rare; wide adhesions at the region of the hepatic and splenic flexures are always present—all these adhesions are parts of a normal embryological and useful process. We recognize, however, that all peritoneal adhesions in the ileo-cæcal region are not embryological and normal; one sees occasionally in adult bodies a degree of adhesion never seen in the child at birth, and anatomists recognize that some of the adhesions are the result of pathological processes. The majority of the adhesions, often regarded as pathological in Nature, are in reality mere expressions of a normal and healthy fœtal process.

The question of ptosis or dropping of the viscera is also one which has interested anatomists. We had recognized the frequency of this condition before we were aware that Glénard had scheduled visceroptosis as a distinct pathological entity, and long before we succeeded in convincing our colleagues the clinicians of the frequency and the importance of the condition. It was a fortunate circumstance that at that time the discovery of Röntgen placed a new means of inquiry in the hands of anatomists. A study of the living diaphragm convinced us that the essential feature of the abdominal viscera is not their fixity but their mobility; they were so attached that they could move freely with the respiratory tide. It became apparent to all of us that mesenteries and visceral ligaments only come into action when the limits of normal movement are reached; the musculature of the abdominal wall is the essential mechanism for supporting the viscera. The pathology of visceroptosis is therefore to be sought in a better understanding of the nerve relationships which exist between the viscera and their supporting walls. The researches of Sherrington, Hill, Mackenzie, Elliott and many others show us how closely the tone and contraction of the abdominal parietes are related to the posture of the body and to the condition of the viscera. We have not yet solved the problem of how the tone and contraction of the musculature of the body wall of the alimentary canal are co-ordinated, but we have sufficient evidence to lead us to suspect they are regulated by a common nerve mechanism. It is probable that this mechanism may be acted on and deranged by pathological products generated in the intestine and that visceroptosis is one of the manifestations of alimentary toxæmia.

I now come to the essential question round which the present discussion has revolved; what is the functional value of the human great intestine? At the beginning of the present century a sharp divergence of opinion set in. In the years 1902 and 1903 three men—an anatomist, Barclay Smith, a bacteriologist, Metchnikoff, and a surgeon, Arbuthnot Lane — came independently and by a different train of reasoning to the same conclusion—viz., so far as man is concerned the great intestine is not only a useless but a pernicious structure. That is a very significant fact; in Paris, London and Berlin the condition of the great intestine was then forcing itself on the attention of thoughtful medical men. To anatomists who knew that the great intestine was an intrinsic part of every air-breathing vertebrate, that it reached a high degree of development and specialization in every mammal that included a vegetable element in its diet, that in all the animals immediately allied to man—his contemporaries and his very ancient predecessors, the great intestine was shaped, arranged and developed as in him, the conclusion that the human great bowel was a useless structure seemed a flat contradiction of every law applicable to the animal body. It is hard to believe that a great structure which has served that long chain of ancestors, carrying man's lineage through the secondary and tertiary periods of the earth's formation and assisting man to become the dominant and universal species of the world, should suddenly fail him. We seem drawn to the conclusion that it is not the organization of the great intestine that has failed, but that our modern dietary sets a task for which it is not adapted. In civilized modern communities the great bowel has to manipulate a dietary such as was never before prescribed to it at any stage of its long evolutionary history. If an engine runs unsatisfactorily it may not be from a fault in its mechanism, but from a defect in the fuel. Those who regard the great bowel as a useless structure blame the engine; for my part I stand by those who blame the fuel.

Let us look at the evidence on which the useless and pernicious nature of the great bowel is based. The great bowel can be excised and health maintained; that proves it is not an essential structure. If diseased, excision may lead to improved health; that proves no colon is better than a diseased one. What must be proved, however, before we regard the colon as a useless structure is that a man without a colon is in a better state than the man with a healthy colon. It will be time enough to relegate the great bowel to the list of useless structures when that much is proved.

This problem of the great bowel is really a child of our ignorance; we have only a vague knowledge of its rôle in the economy of the animal body. If we keep before us the history of progress in medicine it is not likely that anyone will presume, because we do not know the use of the colon, that it has no function and is a useless structure. Every year sees some structural part formerly placed with the useless or vestigial structures removed from that list. Only a few years ago, Pavlov and Starling introduced us to the elaborate mechanism which controls the exit of the contents from the stomach to the duodenum; it was certainly their investigations which led me to look for a similar mechanism at the junction of the ileum and colon.

Elliott demonstrated that such a mechanism existed at the time he was carrying out research on the movements of the great intestine. I feel certain that Dr. Hertz is right when he attributes the effects, which Mr. Arbuthnot Lane ascribes as the result of a kinking at the terminal part of the ileum, to a derangement of the ileo-cæcal sphincteric mechanism. Every step forwards in our knowledge cuts the ground from under those who take a purely mechanical view of the action of the great intestine. Evidence I have accumulated lately justifies me in supposing that the mechanism for regulating the passage of food from the ileum to the cæcum of man is more elaborate than we supposed. Besides the muscular fibres which are situated directly at the ileo-cæcal orifice and which evidently are designed to secure that orifice against reflux from the cæcum there is also another for regulating, as Dr. Hertz suggests, the passage of the contents of the ileum to the cæcum. The musculature of the terminal part of the ileum for an extent of nearly 4 in. (roughly 10 cm.) above the ileo-cæcal junction is endowed with a special tonic function; it serves as a sphincter for the terminal part of the ileum.

I should also like to take this opportunity of emphasizing the need of having every diseased colon removed by operation systematically and minutely investigated. My friend Professor J. W. Smith, of Manchester, has recently given me an opportunity of examining a very complete specimen removed by operation. There was mucous colitis throughout the whole of that part of the great intestine which was excised, with abundance of intestinal sand scattered along the lumen. A typical ileal kink was present 4 in. above the ileo-cæcal orifice. The musculature of that part of the ileum was strongly contracted. The terminal 4 in. of ileum were bound down to the pelvic fossa by a peritoneal adhesion exactly similar to that seen in a proportion of normal newly born children. There was nothing in the nature of the iliac adhesion to suggest that it differed in origin and nature from the natural bond seen so often in children.

Mr. GEORGE ROWELL : My remarks will be confined to the condition which Mr. Lane has named "chronic intestinal stasis." Put briefly, the main conclusion of Mr. Lane's discoveries may be stated thus: That civilization, particularly that of modern times, causes delay in the passage onwards of the contents of the alimentary tract, which often leads to the formation of new structures that accentuate the delay and so facilitate the production and absorption of harmful substances; and that the condition thus brought about may have such profound and extensive results as to affect the whole range of our knowledge of the causation and treatment of disease. Among the many subjects included in this discussion on Alimentary Toxæmia, it would seem to be of prime importance to examine and ascertain how far facts support this revolutionary proposition. The regard in which the theory is held is changing rapidly, and at the present time the position is of much interest, for every degree of agreement with Mr. Lane is now to be found in the profession. Speaking generally, it would appear that many of those who have given the most attention to the conditions which are actually to be found inside the abdomen are the most in accord with Mr. Lane's views. It is because I have seen a large proportion of his surgical work on cases of chronic intestinal stasis from its beginning that I venture to take part in this debate.

No one who has seen Dr. C. Mayo's photographs of the ileal kink can doubt the existence of the characteristic band. I have seen this demonstrated to the onlookers at operations scores of times in the most unmistakable manner. I have also seen the fastened-up appendix, crossing and obstructing the end of the ileum, similarly demonstrated on numerous occasions. Whether the new structures causing these two forms of abnormality are inflammatory or not in origin does not matter with reference to the results produced. In both cases it is easy to demonstrate that there is a fixed point, and it is obvious on examination that the sagging of the bowel in the vertical position of the trunk must in each case cause some obstruction. One constantly sees long lengths of intestine drawn up out of the pelvis in these operations, of which the only fixed attachment is the point alluded to. What the amount of obstruction actually is would also seem to be not necessarily a matter of moment, the important point being that cases exhibiting these structures display in a definite manner the symptoms described by Mr. Lane as those of chronic intestinal stasis. Again, the exact nature of the particular poison in any case is also a minor point so far as diagnosis and treatment is concerned, the weighty consideration here being that when the obstruction is relieved by operation the symptoms disappear.

Reverting to the question of the origin of the bands, the more familiar one is with them the more one comes to look upon them as formed in accordance with the three laws enunciated in Mr. Lane's paper. The exact understanding as to what is inflammatory and what is not is still vague, but if you call these structures inflammatory you will surely have to include the growth of bone on the shoemaker's atlas and in the fossæ of the coal-trimmer's humerus in the same category.

I have seen the bands which have developed in connexion with the duodenum and the sigmoid demonstrated many times in the clearest possible manner, and I have constantly seen the duodenum dilated, sometimes very dilated, in these cases. In many it has actually presented through the abdominal wound. In operations undertaken for duodenal ulcer I have several times seen ileal obstruction examined for and found. The adhesions described at the splenic and hepatic flexures and those supporting the stomach are also quite familiar to onlookers at Mr. Lane's operations. Most of the operations for the relief of chronic intestinal stasis at which I have been present during the last few years have been previously examined by the X-rays and reported upon by Dr. Jordan. It is nearly always the case that the report has predicted accurately the condition that has been found. Indeed, one of the most striking things that I know in surgery is the precision with which, when assisted by the X-rays, the exact causation and degree of stasis can be diagnosed before operation. In my experience the X-rays sometimes show less than is found at operation; a kink is occasionally missed. Certainly I have never seen a dilated duodenum diagnosed by the use of the X-rays that could not be demonstrated at the operation.

As showing the importance of the ileal band is a group of cases of which I have seen many examples; patients in whom pain has persisted after the appendix has been removed. In these cases I have seen the ileal band divided and immediate relief result, although one understands that the relief may not be permanent without a short-circuiting. In a case that I saw recently, a young lady who had marked symptoms of chronic intestinal stasis together with pain over the appendix, was treated by paraffin for six months, with the result that the whole of the marked objective symptoms were cured, but the pain remained. An operation was undertaken for the pain, and an appendix tightly held up and sharply kinked was found and removed.

The general effects of chronic intestinal stasis are claimed to include the impairment of every tissue. It is not easy to see the connexion; if it were, no doubt this would have been discovered long ago. Take, for example, thyroid enlargement. If it be true that the connexion exists, it follows that the relief of stasis ought to diminish the size of the thyroid. Besides the interesting case given in Mr. Lane's introduction, I have on two occasions seen an enlarged thyroid diminish very rapidly after short-circuiting. In the case of a very athletic young lady of which I have knowledge, who had a large thyroid and some symptoms of chronic intestinal stasis, after a course of paraffin given freely three times a day for ten days, the thyroid had diminished to less than half its former size, and the patient felt perfectly well. In Dr. Hale White's opening paper he adduces a case of exophthalmic goitre which disappeared during an attack of typhoid. The rest in bed, the milk diet, and the diarrhœa during the illness obviously must have kept the patient without stasis for some weeks, so that instead of providing an argument against the theory that exophthalmic goitre is associated with chronic intestinal stasis, this case would seem to be in perfect accord with that belief. The cure of duodenal ulcer by rest in bed has a similar significance.

With regard to cancer, I have often seen the symptoms of long-standing chronic intestinal stasis demonstrated in cases of cancer of the breast, and repeatedly in cases of cancer of the bowel, disease of the gall-bladder and of the pancreas, the ileal kink has been clearly shown.

Pyorrhœa is a condition held by Mr. Lane to be a result of chronic intestinal stasis, though I do not suppose that he would deny the possibility of a direct infection, as, for example, from pet dogs. As I see cases of pyorrhœa almost daily, I would like to remark that I have seen complete cures by vaccines as well as by scrupulous local treatment, and that I know of many cases in which the cure has been much aided by paraffin internally. Two of my dental friends of large experience in this disease make an almost routine practice of prescribing paraffin for these cases, and one of them has told me that he does not know whether the paraffin or the local treatment has the largest share in effecting a rapid cure. Certainly my experience is that it is quite unnecessary to remove all the affected teeth in pyorrhœa.

A large number of facts are being brought to light in various branches of medical knowledge which when examined are found to fit in with, and to lend support to, Mr. Lane's theories. The question of infant feeding may be alluded to as an example. In 1901 Dr. Eric Pritchard came to the conclusion that disease in infants, and much in after life, is largely due to overfeeding during the first year. This, he held, causes over-development in the length of the bowel, such overgrowth often extending to many feet, for which there is no adequate room, and therefore peristalsis is impeded, and delay in the passage of the contents of the intestine results. For the prevention and treatment of this condition he has given paraffin very largely, and, agreeing with Mr. Lane, is convinced that the constant exhibition of paraffin has a marked effect in both the prevention and the cure of tuberculosis.[1]

The fact has been alluded to that I have repeatedly seen the disappearance of the symptoms of stasis, more or less rapidly according to the degree of disease present, after the operation of short-circuiting, with and without colectomy. In many of the worst cases the results after some years are even more remarkable. I have seen a strong young man in perfect health, who now plays football, when a few years ago he was desperately ill with an extreme degree of stasis for which colectomy was performed; and there are many cases, equally striking, within my knowledge.

With reference to the points of interest in connexion with anæsthesia in sufferers from chronic intestinal stasis I have already given the results of my experience,[2] and as this matter is of small general interest I will only here call attention to the fact that by the use of the method that has been described the risk of the anæsthetic is practically a negligible quantity, and shock and after-sickness are commonly absent. I have never seen acetonuria, probably because I never use chloroform in these patients.

[1] *Practitioner*, 1912, xc. p. 294.

[2] *Lancet*, 1913, i, p. 32.

To conclude, I would submit, for what it is worth, with great respect to those whose work lies in the diagnosis and treatment of disease, that the facts one has been able to observe, so far as they have gone, completely support Mr. Lane's views.

Dr. LANGDON BROWN: Whether digestion can be carried on aseptically is a purely academic question, since the opportunity never arises. Although the infant starts with a sterile alimentary canal it speedily acquires bacteria therein. In health there is a state of equilibrium established between powers of attack by the microbe and the defensive powers of the body. At the two extremes of life the defensive powers are diminished, and I have had a case in an infant 2 days old and another in a man aged 79 of violent and fatal septicæmia due to *Bacillus coli* without any local lesions. I have met with the same thing in a child aged 3 due to the *Bacillus aerogenes capsulatus*. But normally a balance is struck between the attack and defence in this struggle for existence between the man and the microbe, and I hold that, apart from ingestions of definite poisons, alimentary toxæmia only results when either (1) abnormal intestinal flora become established, which produce specific toxic substances, or (2) the normal flora acquire an abnormal habitat—e.g., ascending the small intestine and invading the bile or pancreatic duct. As an example of an unusual habitat, I may mention a young man now under my care who suffers from rhinitis with offensive crusts, in which the predominant organism is the *Bacillus coli*.

Proteins putrefy, carbohydrates ferment, and to some extent these processes are antagonistic. Of the various groups in the protein molecule, the aromatic bodies, such as tyrosin and tryptophane, and the sulphur-containing bodies, such as cystin, are the main sources of putrefaction. The normal liver can deal with these and allied substances by combining them with glycuronic acid or with sulphates. Whether such substances are able to exert any toxic influence or no depends on whether they have been rendered inert in this way. A case of carbolic acid poisoning, reported by Garrod, brings this out clearly. He examined the urine of a woman who for many years had applied a carbolic acid dressing to an ulcer on the leg until she had produced ochronosis. He reported that she was on the verge of carboluria, as events proved, for soon after she had a sharp attack. His opinion was based on the observation that almost all the sulphates in the urine were in the form of ethereal sulphates, so that her powers of neutralizing the toxic effects of phenol were taxed almost to the full. A little more and she was over the brink. I would suggest that we have here an explanation of the comparative failure of the phenol compounds as intestinal antiseptics. By combining with the sulphates they deprive the body of the power of rendering harmless those putrefactive substances of which they cannot altogether prevent the formation. And a further suggestion arises that the value of sulphates in the treatment of certain intestinal diseases is not limited to their aperient action, but that in addition they have an antitoxic action. Thus the ratio $\frac{\text{ethereal sulphates}}{\text{simple sulphates}}$, which is normally 1 in 10, but may rise in toxæmias to 1 in 6 or higher, gives us a surer indication of the existence or approach of an intoxication by aromatic bodies, than we can possibly obtain by testing in the ordinary way for indican, which is merely one ethereal sulphate that has attracted most attention because of its striking reactions. In common with most other observers I have noted a great lack of agreement between symptoms of alimentary toxæmia and the amount of indicanuria. But I did not realize, until it was recently pointed out to me by Mr. Mackenzie Wallis, how unstable a substance indican is. It must be looked for as soon as the urine is passed, and it is hopeless to expect accurate results from the examination of a twenty-four-hour specimen. Fresh observations, now that this is known, may enable us to decide what diagnostic value indicanuria has. But in any case it merely represents that part of the indol which has been rendered inert.

I will illustrate my first point that toxæmia results from the presence of abnormal bacteria by reference to toxic cyanosis due to sulphæmoglobinæmia. When oxyhæmoglobin is reduced it readily combines with sulphuretted hydrogen to form sulphæmoglobin. As sulphuretted hydrogen is always being formed in the bowel, and as it can be injected there in comparatively large amount without harm, it is clear that unless there were some additional factor required the condition would be universal. In 1908 I pointed this out, and urged that there must be an abnormal bacterial agent at work. This has apparently now been found by Mackenzie Wallis, who isolated from the saliva of all the cases of sulphæmoglobinæmia he examined a nitrifying bacillus which formed a reducing substance from tryptophane and other aromatic bodies. This reducing agent passes into the blood-stream. Once oxyhæmoglobin has been reduced in this way, the formation of sulphæmoglobin follows as a matter of course. In another toxic alteration of the hæmoglobin molecule, paroxysmal hæmatoporphyrinuria, a similar train of events is probable. Laidlaw showed that hæmatoporphyrin was formed from blood pigment much more easily after reduction. Apart from sulphonal or trional poisoning hæmatoporphyrinuria occurs sometimes without any definite symptoms, but occasionally with vomiting, thirst, anorexia, abdominal pain, and profound prostration. Ranking and Pardington described two such cases occurring at the same time in the same house. I had a patient in whom three attacks of this character had led to the suspicion of intestinal obstruction, but my colleague, Mr. Peter Daniel, noted the curious appearance of the urine and handed her over to me for investigation. It has also been recorded in hydroa æstivalis and in periodic vomiting with acetonuria. So that the association of paroxysmal hæmatoporphyrinuria with an intestinal toxæmia due to some reducing substance is practically certain, and the analogy with sulphæmoglobinæmia renders an abnormal bacterial origin probable.

I have occupied so much of my time in dealing with these rare disturbances of pigment metabolism because I think that as the changes are manifest and comparatively simple, their continued study may throw light on vaguer conditions and help us to determine with more accuracy the criteria by which we can define alimentary toxæmia.

The diamines discovered by Barger and Dale to arise from the decomposition of histidine, another product of protein breakdown, afford another promising field of study. There is a very widespread belief that alimentary toxæmia is an important cause of the rise of blood-pressure in later life, and here we have an example of the production of definite pressor substances by intestinal putrefaction. But note that here, too, there is an abnormal intestinal microbe at work, the *Bacillus aminophilus intestinalis*, which supports my general contention.

The reaction of the body against comparatively simple, chemical poisons is quite different from that adopted against foreign proteins and toxins. For these simple, chemical poisons do not stimulate the production of antibodies. They are neutralized by substances normally present in the body, which are few in number, such as proteins, bile acids, glycuronic acid, sulphates, and possibly glycogen. These have an affinity for certain chemical substances, some of which happen to be poisons. The chemical processes are also few and simple, such as oxidation, reduction, hydration, dehydration, and methylation. An important deduction from this is that there is no acquired immunity from such poisons as they do not lead to the formation of antibodies. Such immunity as we acquire is therefore limited to the re-establishment of a normal intestinal flora, whereby the production of these poisons is prevented or diminished.

Just a word as to oral sepsis, on which a good deal has already been said. I have been much struck by the part pyorrhœa may play in the production of atonic dilatation of the stomach, and the rapidity with which the stomach may regain its tone when this is rectified. It is commonly said that atonic dilatation of the stomach is accompanied by diminution, or even absence, of HCl in the gastric juice. But I have met with a number of cases in which the test meal showed a normal and often an actually increased amount of HCl. The acid of the gastric juice is bactericidal, and it seems to me that the development of hyperchlorhydria is, sometimes at least, a compensatory measure to attempt to check bacterial infection. Like all pathological attempts at repair, it is accompanied by obvious inconveniences. But if this is so, it follows that to treat hyperchlorhydria simply by alkalies without attempting to fathom its cause is to side with the microbe rather than with the patient.

It has recently been suggested that an important factor in determining whether pyorrhœa sets up rheumatoid arthritis or not is the amount of acid in the gastric juice, and that if it is sufficient to have a bactericidal effect it will protect against this result. Accordingly one observer gave nitrohydrochloric acid, and claimed a marked success. Considering that the dose employed, 5 minims, would only confer an acidity of 0·005 per cent. on a pint of gastric contents, I find it very difficult to attribute the improvement to the drug. Not long ago I saw a young woman, aged 32, in whom marked rheumatoid arthritis was combined with a high degree of hyperchlorhydria and gastric ulceration for which gastro-jejunostomy had to be performed. This combination is familiar to those practising at spas where arthritics congregate. There are other channels besides the stomach by which the toxins of pyorrhœa can reach the joints, and I fear this ingenious hypothesis falls to the ground.

Finally, two points as to treatment. I would suggest that the very limited success that has accompanied vaccine therapy in alimentary toxæmia, due to abnormal bacteria, can be understood when we realize that it is caused by the ingestion of the simple poisons they form and not to antigens, while the bacillus remains anatomically outside the body. In the other group of cases, due to the presence of normal intestinal flora in abnormal situations, vaccine therapy offers a better prospect of success. Secondly, free purgation cannot be a panacea for alimentary toxæmia since it may produce desquamation of the intestinal mucosa, thus depriving the body of a method of defence against absorption of toxins.

Mr. Edred M. Corner: The history of the progress of the infection of our alimentary canal with micro-organisms is very important and must be thoroughly understood by those who desire to appreciate the practical bearings of the subject of alimentary toxæmia. Briefly, the life-history of an hypothetical individual with regard to alimentary toxæmia may be set forth as follows: As with the great majority of others, whilst the fœtus is *in utero* the contents of the alimentary canal are sterile and the physiological processes of digestion are inactive, so there is no chemical change taking place within the alimentary canal, toxins cannot be produced, and any alimentary toxæmia is impossible. But with birth, or soon after, organisms are introduced into the alimentary canal, and our life-long intestinal education is begun! Being unused to the products of the action of micro-organisms, the baby's tissues suffer. This is seen in the very frequent gastro-intestinal irritation of the new-born, breast-fed or artificially fed; also perhaps in the secretion of fluid by the peritoneum in the abdomen, seen when a patent processus vaginalis is present as a hydrocele. Should the baby's resistances overcome its alimentary toxæmia, all will be well. But should the actions of the toxins prevail, the child will get rickets, a result of the action of what may be called alimentary toxins on the growing tissues. Of course, alimentary toxins, or toxins absorbed from the alimentary tract, are not the only ones that can produce such deleterious action on growing tissues. Others might be absorbed from the tonsillar region. Still, our profession regards the alimentary toxins as the most important by far in the production of rickets. Rickets is not the only result of severe alimentary toxæmia in childhood. Many other diseases might be mentioned, such as wasting, thrombosis, osteo-arthritis, and so forth. Indeed, by lowering the resistance of the tissues of the child, alimentary toxæmia may be said to render the entrance of organisms to the body more easy. Thus it will dispose to tuberculosis, mesenteric glands, and to an infinite number of diseases.

From this life-long intestinal education we learn to endure alimentary toxæmia until a certain amount of alimentary toxæmia is physiological to us and necessary to our well-being. Our powers of resistance to such toxins increase and increase until with mature years and equanimity we can overcome such a feast as the "City banquet;" the onset of diseases are disclosed to us such as tuberculosis and malignant diseases, and as with life our war with organisms begins so truly are the seeds of death sown within us.

As life progresses the tissues of the alimentary tract are affected directly and the rest of the body indirectly. The results of the organisms in the alimentary tract are known and form different chapters, so to speak, in our book of intestinal sepsis. Appendicitis is at once the most frequent and dangerous result of intestinal sepsis. Colitis, ulcerative colitis, are others. Later in life there are gastric and duodenal ulcers. All the affections mentioned at present affect primarily the mucous membrane of the bowel. But the sepsis affects more than the mucous membrane, as shown in the above and diarrhœa and intussusceptions. The action of the muscular coats may be interfered with, producing delay in the passage of the intestinal contents, constipation, or, as it has been called, "intestinal stasis." But, in addition to the inner and middle coats of the bowel, the peritoneal covering membrane may be irritated; in the young leading to the formation of a hydrocele, and in the older, adhesions. The results and treatment of the adhesions, and the kinks caused by them, have already been gone into by Mr. Arbuthnot Lane. In this place it must suffice that of my own experience I can tell you that some patients are vastly improved by such operations as short-circuiting or colectomy, with or without appendicostomy; others are neither better nor worse, whilst a small number are even made worse by any attempt at operation. In the excitement and heat of so large a discussion as this, points of practical issue are apt to be overlooked or buried in accumulation of theories and scientific observations. That some patients are vastly improved by operation is, I *know*, beyond doubt, and I *think* that improvement could not have been attained by any other means. Therefore I would strongly urge that for our future progress data be suggested by means of which we can differentiate those who will be improved by operation from those who will not be benefited. This is very important from both the patient's and the surgeon's point of view. Surgeons owe much of their failure to improve patients suffering from alimentary toxæmia to the fact that so much time has been lost before it is decided to resort to operative treatment. And this brings me to a further point which I would strongly insist upon. Alimentary toxæmia is a progressive disease, or perhaps better, the results of alimentary toxæmia are progressive; so that if the consideration of operative treatment is delayed more and older changes (adhesions, mesenteries, &c.) will be encountered, and the surgeon will have still less chance of being able to benefit the patient. His inability to do so is not his, the surgeon's, fault—poor man! His the unfortunate result of delay; perhaps unavoidable, perhaps avoidable. And it must never be forgotten that the intestinal tract is only one of many systems of excretion whose faults may give rise to auto-intoxication.

In conclusion, I would insist that the terms "intestinal stasis" and "alimentary toxæmia" are not synonymous. People may enjoy well-being, and perhaps health, whilst suffering from intestinal stasis, *if the intestinal contents dry and the micro-organisms consequently die.* But where the intestinal contents are *moist and organisms are abundant*, fermentation must occur, and alimentary toxæmia becomes sufficiently marked to require treatment. A course of waters and purgatives may accomplish the result required, provided no kinks or other variety of intestinal obstruction are present. Thus, when alimentary toxæmia is recognized clinically, I would suggest that treatment by means of diet and purgation be begun. Whilst it is in progress let an X-ray examination be made of the progress of the intestinal contents along the alimentary tract. If any obstruction exists, the surgeon must obviate it. But his work "merely makes the paths straight" and afterwards treatment by diet and medicines must be continued.

Mr. PETER DANIEL: The cause of gastro-intestinal toxæmia is bacterial infection: without infection there is neither food nor any other form of toxæmia other than poisons.

Stasis of the extremest degree occurs for years with no manifestation, while extreme toxæmia occurs with pronounced diarrhœa. Stasis, when it occurs, is either the result of congenital pathological lesions, or, in extra-uterine life, of inflammatory lesions of the mucosa always—with or without secondary affections of the peritoneum.

The greater number of intra-uterine fœtal lesions must be the result of adverse conditions in the mother; almost all such adverse conditions are inflammatory—i.e., bacterial—hence, infection of the mother is the chief cause of fœtal lesions; such lesions are often manifest as exaggerations of normal evolution. In extra-uterine life gastro-intestinal toxæmia is the result of direct infection of the child's own mucosa. All other factors are accessory. Bacteria in the intestine are neither necessary nor beneficial; they are just inevitable, but we can greatly reduce this inevitable infection and ward off virulent varieties of bacteria.

There are two types of children: (1) The comparatively aseptic—i.e., with minimum infection; (2) the relatively septic.

Aseptic children are the product of careful, hygienic mothers, who attend to their mammæ (and general health) during pregnancy; who devote great care to their nipples and children's mouths during suckling; who interdict teats, thumb-sucking, and dirty habits; who succeed in warding off colds, &c., thus preventing infection of tonsils and adenoids with the resulting mouth-breathing, gingivitis and vicious circle they form; also glands in the neck, &c. After weaning, similar care is devoted to the food utensils: the mouths, which are carefully cleansed after each meal; the milk carefully selected and looked after (cow's milk teems with bacteria), and so on. The regular and efficient evacuation of the bowel is secured, and cleanliness about the genitalia. Under such a regime only few and mild bacteria from the mother's skin and ducts gain entry to the mouth and stomach, so that possibly they may be destroyed by the gastric juice (such being normal); in any case, few contaminate the intestine, and few portals of entry for other germs have been created and "the soil tilled." Such a child does not have intestinal toxæmia.

The septic child is reared under reverse conditions—cracked nipples, eczematous skin, dirty ducts, or even with abscesses of the breast, with mother's milk teeming with bacteria, of necessity more virulent and numerous than normal. Later, dirty vessels, evil habits, absence of care in selecting and supervising the cow's milk leads to gastritis with excess of mucus, which protects and conveys the bacteria into the intestine; hence, gross infection of the intestinal tract.

Which of these children gets adenoids and tonsils with mouth-breathing, gingivitis, glands, bronchitis, and a whole gamut of diseases—æons before intestinal stasis occurs. In such infected intestine is laid the foundation for gall-bladder, appendix, pancreatic, and other lesions, which may go on to peritonitis—chronic with adhesions, or acute with pus formation—ulceration, mucous and other forms of colitis and cancer. Which of these gets thyroids, rheumatic affections, tuberculosis, in fact any secondary disease?

When teeth arrive, and in after-life, the mouth is still the great source of infection. Gingivitis, caries, all ill-devised dentures—and particularly those which are permanent fixtures—stoppings, crowns, and so on, and in particular the chronic osteitis of the jaws which finally arises (and is the true cause of trigeminal neuralgia), these are the laboratory for bacterial infection and reinfection of the whole gastro-intestinal tract, as they are themselves prolific sources of systemic toxæmia and infection. Space is restricted, but I have outlined the importance of the mouth. The nose, nasopharynx, accessory sinuses, middle ear, bronchitis, are also great sources of gastro-intestinal infection and toxæmia.

Cow's milk teems with bacteria of various varieties, including tubercle bacilli; the commonest variety of tuberculosis in infants is bovine, and if children acquire tubercle bacilli from cow's milk, what other types of bacteria may they not acquire of greater virulence, and possibly other species, than those acquired from a mother's milk and nipples? A very small difference in variety of intestinal flora produces marked general, as well as local, lesions (Herter and others). Thus, long ere the intestine is involved, tuberculosis, the zymotic fevers, and many other infections may gain entrance into the bodies of these septic children.

Stasis, of which so much is spoken, is one of the most evident gross manifestations of bacterial infection. When the intestinal mucosa is infected, especially at a sphincter, it leads to inactivity of the muscular walls of the bowel—to spasm of sphincter muscles and inactivity of the propelling muscles, in effect protective—hence, stasis occurs, a pseudo-paralysis.[1] When loss of epithelium, particularly if exposing the muscularis mucosæ, has taken place diarrhœa follows, certainly in the large intestine where the process may be watched visually. Hence, I do not believe stasis occurs in the small intestine apart from infection, and I believe the stasis originates most commonly in the cæcum, where the most profound infection of the mucosa occurs and involves the ileo-cæcal valve. Macewen, in his Huxley Lecture at Charing Cross Hospital, showed that the chemical reaction of the food reaching the cæcum (he said passing over the orifice of the appendix) determines whether the ileo-cæcal valve relaxes or remains closed. What is most liable to induce abnormal changes in the food in the ileum other than bacterial infection? Consequently the axiom that "Stasis is the result of infection" is supported by Macewen's direct experiments carried out through a cæcal fistula. The pylorus reacts in the same manner.

Stasis is an evidence of mucosal infection, and does exist in a wide-spread manner; it is, however, the toxins developed by the bacteria that result in general toxæmia, not the food toxins; or if food toxins do take part in the toxæmia, then they arise directly as a result of the action of specific bacteria upon the foods and upon all kinds of food, proteid and carbohydrate. The intestinal tract is an ideal test-tube for germs; it houses them, supplies them with food, removes their by-products, and constantly keeps the stock active and virulent by re-infection. When a patient has an abscess or cellulitis, we do not attribute the temperature, delirium and ill-health to food toxins, the end-products (practically fæces and urine of bacteria) of the bacteria suffice to explain them all. In the genito-urinary tract the same argument applies; although I quite believe the breaking up of both food and urine does result in toxins peculiar to each.

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[1] *Clin. Journ.*, 1913, xli, pp. 289-304.

Since children are born tuberculous and syphilitic, &c., it is obvious that an intestine in a condition of stasis is not essential for the entry of tubercle bacilli into the body, and thus into a joint. The lymph glands in the neck (secondary to mouth, &c., sepsis) and the bronchial mucosa are probably as prolific portals of entry of tubercle bacilli as the intestinal mucosa. If stasis exists, just in the same way as if the genito-urinary infection exists, they damage the body tissues—i.e., till the soil and conduce to successful invasion, say, of a joint.

BANDS.

As for the pathology of Mr. Lane's bands, Nature would not evolve "bands" below the intestine to prevent them falling down into the pelvis; if so she is an inferior architect. Since space is not afforded here to indicate the real explanation of these bands, and why they are below the intestine, I must simply state they are certainly inflammatory in origin, often congenital in point of time, and do not cause stasis *per se*, but only conduce to that condition or exaggerate it when inflammatory conditions develop in the mucosa.

Mr. Lane occupies a large portion of his paper with a consideration of the effects of injuries to bones or joints, which he explains on purely physical grounds, utterly ignoring that he himself shows that these very persons are subjects of toxæmia, and that the processes he describes are produced as much by the toxæmia acting upon damaged tissues as by the damage itself. If the breast can be so affected by intestinal toxæmia as to undergo fibrosis, &c., surely bones and joints may be equally affected, particularly after *damage* such as he describes, gross trauma, or over-work? Myositis ossificans not due to syphilis is certainly due to gastro-intestinal toxæmia, and if such changes can be produced in fibrous tissue, it is easy to understand the excessive formation of bone and the buttresses, &c. Traumatic myositis, such as follows elbow injuries, is enormously added to by undue massage and movements of the injured part—so much so that rest and absence of treatment appears the best method of cure. My experience of these two conditions is that I never see either of an advanced type in the absence of oritical sepsis. When he states that intestinal toxæmia affects all tissues of the body, does he mean to infer that the injured bone or joint tissues are exempt from such toxæmia? For similar reasons surely the systemic toxæmia *may* produce bands in the peritoneum if it is competent to produce fibrosis elsewhere.

The pathology given by Mr. Lane for ileal bands and kinks is condemned by the fact that they occur *in utero*. In the fœtus in whom bands exist, what was the condition of the mothers? Were they not in all probability the subjects of toxæmia—gastro-intestinal, genito-urinary, or bronchial? And is it conceivable that a profound degree of infection in the mother is without influence upon the fœtus, and particularly at an early period when the physiological changes in the peritoneal cavity are taking place? Such toxæmia is likely to lead to excess or failure in the physiological changes.

THE CAUSE OF ILEAL STASIS.

When the intestinal mucosa, especially at sphincteric areas, is infected, the result is interference with the chemistry and motility of the organ or part; this leads to delay in the passing onwards of the intestinal contents. From stomach pathology we know that infection results in disturbance in the chemistry of the organ and alteration in the behaviour of the sphincter. These two factors, then, explain entirely the phenomenon of stasis; visceroptosis may follow, and any mechanical disabilities may accentuate the stasis, yet when we consider the grave mechanical obstruction to the onward passage of the intestinal contents in irreducible herniæ, we must regard pure mechanical obstruction to the contents of the small intestine as doubtful, since we never find the Lane symptom-complex of mechanical obstruction in the worst varieties of irreducible hernia.

After gastro-jejunostomy for stomach ulcer in women whose whole viscera are prolapsed, so that the hepatic flexure is below the level of the iliac crest and (in one case) the splenic flexure also below this level with the cæcum in the true pelvis, I have seen bismuth food reach the splenic flexure within four hours. Yet these cases gave *all* the bismuth X-ray peculiarities of ileal stasis before the operation of gastro-jejunostomy.

THE SYMPTOMATOLOGY OF GASTRO-INTESTINAL TOXÆMIA.

The more we know of diseases of bacterial origin the more do they appear to be *septicæmic*. There are few strictly local diseases; they have local foci, but the bacteria constantly escape into the systemic circulation. There may or may not be systemic manifestations of such generalized infection—that depends upon accessory factors; often the accessory are considered the essential factors, and it is this error which so many clinicians make—they overlook the primary gastro-intestinal lesion. Consequently the symptoms of gastro-intestinal toxæmia are: (1) local, (2) systemic—i.e., septicæmic. The local are not under consideration now, and the systemic are so numerous and vary so much in degree and intensity that it is impossible to say more than that all the organs and tissues of the body are at some time or other made diseased by the systemic absorption of toxins and bacteria from the gastro-intestinal tract, as I have in my own experience verified (with very few exceptions). Thus the toxæmia may be manifest in any form in which septicæmia may evince itself. I believe it is the most frequent source of most of the ailments of everyday practice, and that oral sepsis is the commonest form it is derived from.

Dr. Morton Hewetson in his work on "Scurvy," in natives working in the mine at Wankie, South Rhodesia, proves conclusively that oral sepsis is the direct cause of scurvy with an exceedingly high mortality —that the sepsis originates in tribal customs which by removal or mutilation of the upper incisors and other teeth lead to gingivitis and the other sequelæ of oral sepsis.

The special pigmentation Mr. Lane lays stress upon is not special to gastro-intestinal toxæmia; it is one of the special septicæmic manifestations of this action upon perhaps the adrenals or some other organ, and it occurs in other septic states. Stasis may be associated with a most excellent complexion.

Much is made by physicians of the effects of liver disorders in preventing or modifying the absorption of toxins from the gastro-intestinal tract. The liver is the first organ to be damaged by stomach and intestinal toxæmia, its chief diseases are directly due to such absorption, and if the blood supply to the liver is considered it will be seen that the liver is for all practical purposes exempt from toxæmia from systemic infections. The hepatic artery is only about the size of the radial, so that the amount of toxic material conveyed by it is relatively small; true, the splenic artery conveys infected blood to the spleen and hence indirectly by its vein to the liver; but the spleen is surely competent to deal with such poisons if any organ is, and, therefore, it is possible that no toxins, but antitoxins, may be conveyed to the liver by the splenic vein. On the contrary, the portal vein, by its great size, the amount of mucosa it drains, must convey a great amount of toxic material, including bacteria, to the liver, and it is hardly to be wondered at if cirrhosis and the other common lesions of the liver arise.

We have all known life abstainers from alcohol who have died of pronounced cirrhosis; they have invariably been dyspeptics, and all I have known were the subjects of oral sepsis or were edentulous— an excellent proof of many years of such. Alcohol, being a toxin, helps to damage the mucosa and thus conduces indirectly to cirrhosis.

Do we know what the toxins of scarlet fever are? We do not doubt their evil effects. For years syphilis and malaria were treated empirically successfully, yet we knew not their cause nor toxin. Let us first recognize the ill-effects and combat them, and if we do this with regard to orificial sepsis, and leave off wondering what the toxins are, we should do immeasurably more good than we did with regard to syphilis and malaria, since at present orificial toxæmia is almost universal.

It is the bacteria which invade the mucosa or act in the mucus immediately adherent to it which are the chief agents in intestinal toxæmia; the results of cultivating from the fæces are bound to be misleading, we must obtain smears from the actual mucosæ.

THE TREATMENT OF GASTRO-INTESTINAL TOXÆMIA.

This simply means in the first place the radical cure of oral, nasal and the other sources of sepsis. In so far as stomach and intestinal sepsis is concerned recovery after the primary sources are dealt with is automatic. Prevention is most certainly possible if we begin with the infant; if, however, intestinal stasis be due to the upright posture prevention is impossible unless we revert to the primitive all-fours mode of progression. But since stasis is really secondary to infection the outlook is not so doleful.

If we find the source of infection is a curable lesion, as in gingivitis or antral abscess, then such cure followed by laxatives and the usual modes of building up strength, &c., usually results in a practical cure of the secondary infection lower in the track, unless the damage done is irreparable. If the source of infection is not curable then the gastro-intestinal toxæmia will persist; if the source is amenable to amelioration then some improvement may take place in the lower tract, but on account of the reinfection taking place this will be very slight. Thus adequate treatment of the primary lesion is essential, and herein lies much difference of opinion and shipwreck. One is commonly told, "The teeth, or what not, have been cured, yet my other symptoms are not cured." Such is often true, although usually adequate treatment has not been carried out: nevertheless, because the endocarditis which followed tonsillitis is not cured when the tonsil has subsided, we do not say "You must not treat the tonsil." I am convinced indeed that enucleation of tonsils in the early stage of rheumatic fever would save many an endocarditis. Such has been proved in acute nephritis.

As regards stasis, if it exists, I am convinced most of the evils of the stasis are cured if the oral or other primary source of infection is remedied, since Mr. Lane admits that the administration of paraffin alone will remove all the appearances of stasis. By all means help the bowel by every means in our power — massage, enemata, belts, drugs, &c.

If the appendix has become so involved that its recovery is impossible we must remove it. Likewise, if a gastric or duodenal ulcer exist it demands adequate treatment also. When the colon is ulcerated then I believe ileo-colostomy is a splendid operation, granted we can implant the ileum below the lesion in the colon. I am convinced that this operation will in future be utilized chiefly for colon diseases and not for so-called stasis. Yet I would warn surgeons that patients who submit to this operation are often rendered uncomfortable thereby, and if undertaken without sufficient reason will regret it. When they have sufficient cause then the resulting discomfort is in comparison negligible.

When the colon is gravely damaged, either an appendicostomy must be performed at the time of short-circuiting or the colon must be excised, as the ulceration has a deplorable tendency to progress. The patients usually are too ill to stand the whole operation, so ileo-colostomy with appendicostomy should be the first step, and when the health is improved colectomy may be permissible should the bleeding, &c., recur. I have done this operation in one sitting on several occasions, but adhesions in the cæcal and hepatic regions are almost inevitable, though much may be done by careful use of the lax peritoneum and portions of the omentum to cover the bared retroperitoneal tissue.

Mr. C. H. FAGGE: The purpose of my intervention in this discussion is to support the view for so many years advanced by Mr. Arbuthnot Lane, that in many cases of intestinal toxæmia mechanical factors in the shape of adhesions, bands, or false mesenteries produce kinks with intestinal stasis as their result, and that the operative treatment of these kinks is of the first importance. I have had the unique advantage, which I trust I appreciate, of working in the same wards as Mr. Lane during the past eight years, and during his occasional absence his cases have come under my care; I shall, however, as far as possible speak of opinions gained from my own operative experience, though I must, of course, admit that the principles on which my opinions are founded have been gathered from Mr. Lane's practice, and that I was led to take interest in the matter by the success which he has achieved in many cases which I have watched throughout.

I do not propose to found any argument or base any conclusions upon analysis of symptoms or X-ray evidence after giving bismuth. No one can have much experience of abdominal surgery without realizing that the conjuring with probabilities which is known as diagnosis is still largely founded on ignorance, for observation at operations has taught us that symptoms apparently suggestive of disease in one organ are often due to disease in another; even in the present day physicians are too prone to found theories of abdominal pathology on bedside or post-mortem evidence and to neglect daily opportunities of correcting their beliefs by witnessing abdominal explorations by their surgical colleagues; in no branch of medicine is Sir Berkeley Moynihan's plea for an investigation of the pathology of the living more necessary than in the study of intestinal stasis.

I propose to omit X-ray diagnosis because I am not competent to construe its findings, though I must admit that in this class of case the advice of an expert is invaluable and it would be ungenerous not to acknowledge that in nearly every case on which I have operated I have gained help from Dr. Jordan. In no single case has Dr. Jordan's X-ray diagnosis suggested more than I have found at operation, and if I were to grumble it would be that an occasional kink or bound-down ileum has escaped him.

I have omitted the above because I want my hearers to understand that my statements and my beliefs are all founded on what I have actually seen during the course of systematic laparotomies, and I may say that, except when circumstances were unfavourable, I have for several years carried out a thorough routine examination of the parts concerned in this question in every abdomen I have opened for any purpose.

It is fortunate that now the presence of ileal or other bands and kinks is not denied, for there must be many who, like myself, have sufficient personal experience to know that in certain types of case Lane's ileal kink is very commonly met with *if it is systematically* *looked for.* I have taken most interest in that class of case which is considered before operation to be either duodenal or gastric ulcer, cholecystitis from gall-stones, chronic appendicitis or ileal stasis, or a combination of any or all of these, and I have therefore paid most attention to those bands and kinks which affect the end of the small and the commencement of the large intestine, though of course the rest of the colon has shared with the other abdominal viscera in my routine examination. I am inclined to think that we may divide cases of intestinal stasis into those which involve the lower ileum and often simulate and probably induce gastric and duodenal disease and those which affect the flexures of the colon; the latter, though peculiarly amenable to surgical treatment by short-circuiting, do undoubtedly in many cases get along by the aid of medicinal treatment, and under the head of mucous colitis or constipation lead an active life provided they use purgatives or enemata regularly; in advanced cases both types are found in combination. It is interesting historically to note that in his earlier papers Mr. Lane regarded the large intestine to be the chief source of trouble, taking much the same view of its iniquities as we heard expressed last week in a paper from the medical point of view, and it is only in his publications of the last four or five years that Mr. Lane has ascribed the changes in the large to earlier defects in the small bowel.

The harsh antagonism to Mr. Lane's views has now shifted its ground, and it is most interesting to watch the transition from a denial of the actual occurrence of these bands either to a criticism that the evolutionary view of their causation is inaccurate or to an opinion that if their occasional presence be admitted no symptoms can possibly result therefrom. I share with others a difficulty in comprehending and in accepting in their entirety all Mr. Lane's views as to the pathological conditions which he regards as resulting from ileal stasis, but after many opportunities of actually seeing that his diagnoses before operation were found to be substantiated at the operations, I have verified the fundamental observations in so many of my own operations; I am, therefore, logically bound to admit that when the basis of his work entirely coincides with one's personal observations on the living, there is every probability that Mr. Lane's far-reaching suggestions of ætiology and practice may also in course of time prove to be correct.

It is unfortunate that at first the wide question of stasis producing toxæmia was regarded as synonymous with constipation, though this is often but slightly marked in the most toxic patients; it is also to be deplored that Mr. Lane in his earliest papers of 1902 and 1903 did not attach definite names to the abnormal structures he discovered in these patients for those bands which support the cæcum and ascending colon, as now widely known as Jackson's veil, who first described them in 1908, while Lane described and actually figured them in his second paper on this subject.[1]

Discussing now the ileal kink, it may occur in two forms: Firstly, as a new flat band passing from the right pelvic margin or from the right (originally the left) surface of the mesentery to the adjacent aspect of the ileal circumference and gradually creeping round it until it has reached and bound down the anti-mesenteric border of the bowel. This band typically occurs alone, the appendix is quite free and probably normal to the naked eye, the proximal ileum ascending from the pelvis is dilated; no better depiction of this condition can be imagined than that of Dr. C. Mayo, which Mr. Lane has reproduced in his opening paper.[2] I have now seen this type of band and kink in such a large percentage of my cases of duodenal ulcer (I am sure I am not overstating it at one in three) that I cannot believe that this association is accidental; I have usually been content to free the ileum by dividing the band and to do a posterior gastro-jejunostomy as I did in the last case I operated on. Even if one upholds the view that the ileal kink *causes* the duodenal ulcer it does not appear to me illogical to relieve the end-result by the drainage brought about by a posterior gastro-jejunostomy, which is in itself, in my experience, one of the most satisfactory operations of surgery. In another recent case I operated on a moribund man for a perforated duodenal ulcer, and at the autopsy the kink was found and demonstrated by one of my medical colleagues, and by our resident surgical officer. As yet I have no fixed opinion as to the right treatment of this type of kink: when slightly developed division of the band gives at least temporary relief, but a raw surface has necessarily been produced, and at present we have no sure method by which we can prevent adhesions forming over this area; paraffin and an abdominal support often give much assistance. Then again, extensive freeing of this band, especially in men, produces considerable disturbance for a few days, probably due to paralytic distension, so that when in doubt as to the safety of dividing a well-marked band it is wiser at once to short-circuit the ileum into the pelvic colon. My small experience coincides with Mr. Lane's, that ileo-colostomy is less dangerous than extensive separation of bands involving the ileum.

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[1] *Lancet*, 1903, i, p. 153.

[2] *Proceedings*, Discussion on Alimentary Toxæmia, p. 75, fig. 34.

In my series of cases, this isolated band controlling the lower ileum is commoner than the second type in which a similar kink is produced by a twisted, narrowed, or kinked appendix adherent near its base to the mesentery and eventually involving the circumference of the ileum (appendix forming inner limb of parallelogram of Lane); in this class are the cases of stasis which are much benefited by simple removal of the appendix. Dr. C. Mayo's figure, referred to above, well shows another way in which I have often seen the appendix involved as first described by Mr. Lane, viz., by being caught and dragged upwards and outwards by the lowest band passing from the parietal peritoneum on to the ascending colon and cæcum (bands representing the outer limb of the parallelogram of forces of Lane or Jackson's veil); in this type, too, removal of the appendix is beneficial in that appendicular colic or acute inflammation is prevented, but this fails to relieve the greater part of the mischief, which is colonic stasis. I have seen several cases bearing out this contention, for which ileo-colostomy alone appears in my opinion to be effectual.

The duodeno-jejunal band, which according to Lane's view causes an abrupt kink at that flexure, was, I believe, first described by Dr. W. J. Mayo[1] under the title "Relation of the Meso-colic Band to Gastro-enterostomy." It is interesting to note that so acute a mind did not suspect any causal relationship between this band and the gastric or duodenal ulcer, for which, presumably, gastro-jejunostomy was required. I gather that Dr. Mayo regards this, with the band suspending the duodenum to the gall-bladder and Lane's ileal band, as congenital in origin.

In the course of this discussion we have been told that dilatation of the duodenum or duodenal kink does not accompany duodenal ulcer. I should like to inquire on what evidence this statement is made, as apparently we are in this instance to accept evidence derived from a bismuth X-ray, whereas in the same paper a bismuth X-ray of the ileal kink was stated to be unreliable. Only last week a man was sent to me at Guy's Hospital as a case of duodenal ulcer; this was agreed to by one of my medical colleagues and myself as the probable diagnosis after a test meal, yet operation showed no ulcer but a dilated duodenum and a marked cholecysto-duodenal band was associated with an extensive ileal kink caused by a kinked appendix controlling the ileum. I have seen this dilatation of the duodenum at several operations, just as I have under the same circumstances seen the ileum dilated behind one of the forms of ileal kink.

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[1] *Ann. of Surg.*, Philad., 1908, xlvii, p. 1.

There is less discussion about the bands which form in relation to the colon; Wilms[1] described as a congenital variation the "cæcum mobile," which Jackson[2] believes to be secondary to obstruction from membranous pericolitis (Jackson's veil). I have seen these bands, as I have said first described by Lane, on many occasions passing down on the outer side of the ascending colon, and have usually found the cæcum dilated and prolapsed; I have less often met with a band at the splenic flexure, but in most of the cases in which the ascending colon has been involved the right half of the transverse colon has been drawn down parallel to it.

The close association of these conditions would seem to demand a common line of treatment; I do not feel drawn towards the cæcopexy of Wilms, nor did the operation of cæcoplication which I saw carried out several times last year in the Mayo Clinic appeal to me as sound, because it can only favour the further descent of the cæcum into the pelvis, and does not deal with the bands involving the colon. For this condition short-circuiting of the ileum into the pelvic colon appears to me ideal, and to have a brilliant future in cases where medical treatment has failed; if I rightly gather the gist of the last few sentences of Dr. Hertz's lucid contribution to this discussion this view will be a basis of treatment on which medical and surgical opinion will eventually agree; colectomy will sometimes be required in severe cases or as a secondary operation to short-circuiting for persistent pain due to reflux into the descending colon.

Now the alternative theories to the evolutionary one of Lane is that these bands are congenital (developmental) or inflammatory in origin, and these alternatives are ably discussed in Jackson's paper, to which I have already referred. Can the supporters of the congenital theory point to other instances in the structure of the body in which the abnormality regarded as congenital is hypernormal, as such bands must be? Instances of hyponormal congenital abnormalities (arrests of development) are common enough, but I cannot think of any instance of the converse; yet Dr. W. J. Mayo in a recent paper on the subject[1] writes: "Lane's kink is undoubtedly also of developmental origin." I have seen nothing in a fair number of laparotomies in infants to support this view. Again, I cannot believe that bands (*a*) localized as they often are to the terminal ileum with the appendix free and normal to the naked eye, and (*b*) always passing in the same direction as those which pass down and inwards to the ascending colon, (*c*) limited to one aspect of the intestinal circumference, can have an inflammatory origin; I would even go so far as to regard as non-inflammatory some bands in which the appendix was involved, meaning that I have failed to find evidence that they were due to a subacute or chronic infection spreading from that organ. One has only to examine carefully several of these bands to realize that they are quite unlike ordinary inflammatory adhesions in appearance. My experience has absolutely convinced me that these bands and kinks produce pathological states as evidenced by abdominal pain, tenderness, loss of weight, tone and activity, flatulence, constipation, &c.; and from my results in a small number of cases treated, since 1908, by separation of bands with or without appendicectomy, ileo-colostomy or colectomy without mortality, I have sufficient confidence to state as my firm belief that stasis is generally primarily due to a pathological condition of the lower ileum, evolutionary in origin, and that in well-marked cases surgical treatment is required, for I have seen marked immediate benefit result to these patients as evidenced by loss of toxæmia and pain, gain in weight and of appetite, and by a return of ability to lead and enjoy an active life.

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[1] *Archiv. f. Klin. Chir.*, Berl., 1903, lxix, p. 795.

[2] *Ann. of Surg.*, 1913, lvii, pp. 374-401.

Mr. H. W. CARSON: I propose to confine my remarks in this discussion to the form of intestinal toxæmia that results from intestinal stasis. There is no necessity to recapitulate the effects produced by this toxæmia on the general health, or on special organs, for these have been referred to by many speakers. In passing, however, I wish to refer to two disorders, the cure of which almost depends upon the correction of intestinal toxæmia. The first is parenchymatous goitre, the treatment of which on these lines has been so ably advocated by Major McCarrison, and the second is subacute or chronic mastitis, such as is seen in young unmarried women. The results of treatment of this condition, which have always been unsatisfactory, have been much more encouraging in my hands since treatment has been directed to the cure of intestinal auto-intoxication.

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[1] *Amer. Journ. Med. Sci.*, Philad., 1913, cxlv, p. 157.

One of the first points to discuss is the question of causation. Accepting, as one must, that toxins are normally formed in the intestine, and agreeing that in health the protective powers of the body are able to neutralize the toxins normally formed, it follows that for intestinal toxæmia to occu reither the toxins must be formed in excess or there must be some abnormality in the elimination of those toxins, or both. It is accepted, I believe, that delay in the passage of the intestinal contents leads to increased bacterial activity and increased absorption of toxins, so that it is not unreasonable to suspect abnormalities in the intestinal tract causing delay in cases of intestinal toxæmia. It is my experience that in this form of intestinal toxæmia certain varieties of the normal intestinal flora are in excess, and occasionally, though rarely, new varieties are present, and in addition some abnormality of the intestinal tract, such as ptosis or interference with its peristaltic action by kinks or adhesions, is superadded. It is my view that the bacterial excess is not fortuitous, but is dependent upon the stasis of the intestinal contents. I consider that the course of events is as follows: First, constipation, which may have originated in many ways—for example, by improper feeding, insufficient exercise, irregular habits of evacuation, and so on. This results in over-loading of the large intestine, and, owing to the force of gravity in the erect position, which is maintained practically for sixteen hours out of the twenty-four, there is a great strain upon the cæcum and ascending colon, a lesser strain on the transverse, and little, if any, on the descending colon, though at the beginning of the sigmoid flexure there may be evidence of strain. I have no doubt that every surgeon has handled a cæcum full of fæcal matter, and no one who has not done so can realize how heavy it is. The effect of this dead weight is to cause prolapse of the cæcum, and the over-distension of the cæcum causes delay in the emptying of the terminal cells of the ileum. Nature's effort to overcome this tendency to prolapse of the cæcum leads to the formation of bands, which pass from the parietal peritoneum on the outer side of the ascending colon downwards and inwards, to be attached to the colon on its anterior surface as far inwards as the longitudinal band. At first they are transparent veils of no strength that can be easily stripped off, but later on they become so firm and so intimately attached that they not only cannot be stripped off but they kink the colon, sometimes indeed doubling the colon on itself, or fusing the ascending and transverse limbs of the hepatic flexure together. These adhesions, which soon interfere with normal peristalsis, later on may cause actual organic obstruction. A similar band, or series of bands, is often seen at the beginning of the sigmoid flexure, the object being to support the sigmoid, which, owing to its long mesentery, may easily prolapse into the pelvis if constantly weighed down with retained fæcal matter. In the same way a definitely limited, fan-shaped band may form, always in the same position, on the lower surface of the mesentery of the ileum near the ileo-cæcal junction, its object being to afford support to the terminal coil of the ileum, which is constantly being dragged upon, at the point where its mesentery is shortest, by the weight of its contents, which cannot enter the already over-distended cæcum.

My belief, then, is that these bands are protective in the first instance and are not inflammatory at all, and if this is so it very profoundly affects one's treatment. My reasons for thinking that they are not inflammatory are these: The bands occupy definite positions, their thickest part is the part farthest away from the gut, proving that they do not originate on the gut as one would expect if they resulted from infection through the intestinal wall, and the regularity of their structure is quite different from that of the ordinary inflammatory adhesions.

I had the opportunity of operating on a case just before Easter, in which these bands were present in a typical formation while inflammatory adhesions were present on the sigmoid and pelvic colon resulting from sigmoiditis, and the difference between these two formations was very marked. I recognize, of course, the occurrence of adhesions round the lower end of the ileum as a result of appendicitis, but this is different in appearance and distribution from the ileal kink resulting from stasis, and, indeed, in an ordinary case of intestinal stasis the appendix is normal, and free from adhesions. The effect of the ptosis of the large intestine and of the pericolonic adhesions is a slowing of the passage of the contents of the colon and the damming back of the contents of the lower ileum. Examination with the X-rays after a bismuth meal shows that there is no delay in the passage of the first part of the meal through the small intestine to the cæcum, but there is delay in emptying the ileum, showing, I think, that the ileal kink does not cause delay, but that delay is caused by the inability of the whole of the contents of the ileum to enter the cæcum which is already full on account of stasis. But I am convinced that the ileal kink does give rise to one symptom, and that is pain, for I have found on several occasions, where an ileal kink is present without pericolonic adhesions, that pain is a more prominent feature than constipation. Absorption of toxins may then take place either from the large intestine or from the lower part of the small intestine; which takes the more important part is a matter merely for academic discussion, if we agree that from the point of view of treatment the important factor is the preliminary stasis in the cæcum and ascending colon. The effect of this stasis and deficient peristalsis on the colon itself must be profound, and I cannot help being convinced that mucous colitis, for instance, is not a primary disorder, but is secondary to stasis in the large intestine.

Following this line of reasoning, it is apparent that treatment must have for its object the relief of constipation, and the prevention of ptosis of the intestine. That this can be done in early cases by medical treatment I have no doubt. This should take the form of dieting to reduce fermentation, and to make the indigestible residue as small as possible: the administration of aperients or of liquid paraffin, associated with abdominal massage to promote the ready passage of the intestinal contents; and, if ptosis has occurred, to apply support in some form or other, as by means of a Curtis belt. Intestinal antiseptics and vaccines are also of some value, though that is limited. But if ptosis is marked, and especially if adhesions are present, I am convinced that medicinal treatment must be attended by many failures.

In what cases, then, should one recommend surgical measures? There are two conditions which justify operation: Firstly, failure to obtain any relief, or only temporary relief, by thorough medical treatment; secondly, when the presence of adhesions can be demonstrated. There is one symptom above all others which is suggestive of adhesions —namely, pain of a diffuse character, but mainly on the right side of the abdomen, which is relieved by lying down. The pain is generally described as a drawing, or dragging, pain, which tends to radiate upwards from the right iliac fossa. It is occasionally associated with intestinal distension and spasmodic gas pains. But the method of examination of most value is an X-ray examination after a bismuth meal. It is possible by this means to demonstrate without question delay in the terminal coils of the ileum, ptosis of the cæcum and transverse colon, and marked kinks or distortions of the colon. I do not believe it is possible at present to demonstrate an ileal kink with certainty in all cases, but in some cases the kink is shown very clearly. I am of opinion that if in a case of intestinal toxæmia one can demonstrate ptosis of the cæcum with stasis of its contents and kinking or other malposition of the colon, one is justified (especially if the typical pain is present) in recommending operation.

With regard to choice of operation, one must be guided entirely by the condition found on opening the abdomen. The choice lies between freeing the gut by dividing adhesions and covering in the resulting raw surfaces to prevent re-formation, and short-circuiting the large intestine by dividing the ileum and implanting the proximal end into the pelvic colon. It is impossible to lay down definite rules as to the operation of choice, but in view of my belief that the adhesions are designed in the first place to prevent ptosis of the cæcum, I am convinced that if the operation of dividing adhesions be decided upon, it is absolutely necessary to anchor the cæcum by sutures into the right iliac fossa, or the operation will merely increase the ptosis of the cæcum and aggravate rather than relieve the condition. It is very important also, whatever operation is done, to continue medical treatment for at least six months.

Mr. K. W. Goadby: A good deal has already been said of oral suppuration as a cause of alimentary toxæmia. Much stress has been laid upon "swallowing pus," as if this fluid in itself was the actual cause of intestinal disturbance. It appears, however, more reasonable and more in accord with other chronic pathological processes, to regard local absorption and infection through the actual site of inflammation as the channel of invasion; particularly must this obtain in pyorrhœa alveolaris, where the primary effect is lowering of general body resistance, allowing a secondary invasion of the mucosa of the alimentary tract by the swallowed organisms, thereby producing the curious complex of symptoms frequently associated with pyorrhœa alveolaris.

In the following table I have summarized a series of 150 consecutive and unselected cases of suppuration of the alveolar process of the jaws (pyorrhœa alveolaris); 36 of the cases—i.e., 24 per cent.—had no general symptoms whatever, notwithstanding the constant swallowing of pus for a period of at least two years; 64, or 42 per cent., had

Table showing Chief Symptoms of Disease associated with Pyorrhœa Alveolaris in 150 Consecutive Unselected Cases.

		Previously treated	Not previously treated	Gastro-intestinal symptoms		Secondary anæmia		Rheumatism		Neurasthenia		Unaffected
		(*a*)	(*b*)	(*a*)	(*b*)	(*a*)	(*b*)	(*a*)	(*b*)	(*a*)	(*b*)	
Class A.—General pus formation from sockets of all teeth; affection of at least two years to patient's own knowledge	42	31	11	17	7	15	6	16	5	14	3	7
Class B.—Localized but definite pus formation, many teeth unaffected; affection at least two years to patient's own knowledge	82	39	43	17	13	16	19	26	25	8	9	21
Class C.—Rarefying osteitis, interdental pus, microscopical diagnosis only	26	—	26		10	—	10	—	12	—	10	8
Total { Male 60 Female 90 }	150	70	80	64		66		84		44		36
Percentage	100	46·5	53·5	42		44		56		29		24

Post nasal catarrh	20
Antrum suppuration...	1
Appendicitis	4
Youngest	16
Oldest .	78

definite symptoms of gastro-intestinal affection, indigestion, flatulence, constipation alternating with diarrhœa, &c. Again, 56 per cent. of all the cases had rheumatic symptoms, peri-articular arthritis, arthritis deformans, or fibrositis and muscular rheumatism. In none of the 150 cases was gastritis an acute symptom, and no history of gastric or duodenal ulceration was obtained in any case, the main symptoms always pointing to affection of the large bowel, rather than the upper portions of the intestinal canal. Anæmia of secondary or toxic type was a feature in 66 cases; the anæmia characterized by diminished hæmoglobin, red cell and polymorphonuclear counts, with increased lymphocyte count.

It would appear, therefore, that pyorrhœa alveolaris, as well as suppurative processes of the accessory nasal cavities, are more often associated with intestinal than gastric symptoms, that other generalized affections, such as rheumatism, anæmia, secondary toxic neurasthenia, are quite as common, if not more common, than the intestinal affections; further, rheumatism and anæmia are often unaccompanied by intestinal symptoms. Direct gastro-intestinal affection by swallowed products which must pass through the protective fluids of the stomach and duodenum is insufficient to explain the remote pathological changes produced by oral and nasal sinus suppuration. Secondary anæmias, arthritis deformans, and other rheumatic syndromes proceed from blood infection by chronic suppuration in other regions of the body, and the constant deep involvement of the bony structures in pyorrhœa, the close association of the injected areas with the blood-stream and the frequent exacerbation of anæmia, arthritis, diarrhœa, &c., on the injection of autogenous vaccine or by operative interference with the infected tissues before immunization has been carried out, all point to blood-stream infection as the most important and common road of infection.

The essential pathological change in pyorrhœa alveolaris is rarefying osteitis of the alveolar process, together with the body of the bone as well. The alveolus becomes replaced by succulent granulation tissue in which are numerous bacteria. If the total tissue involved be computed, it will be found in many cases to comprise about one-sixth of the whole jaw, thereby allowing of a wide area of absorption in intimate relation to the blood-stream. In the upper jaw the affection frequently extends into the maxillary antrum. When interdental pockets are formed, pus is produced, but not otherwise; the pus contains an enormous number of bacteria, the greatest number adventitious species, acting not upon the living tissues, but upon the epithelial debris, entangled food, altered pus cells, &c.; most of these organisms are anaerobic and serophile. The *Crenothrix buccalis* accounts for a large number of the morphological forms seen. Under this decomposing mass the true active agents are found, much as is the case in chronic putrid bronchitis, bronchiectasis, &c. Certain of these organisms may be found in the fæces in severe cases as well as in the urine.

So far I have considered the affections classed under the term pyorrhœa alveolaris in relation to gastro-intestinal toxæmia and infection, but the converse—namely, the effect on the tissues of the mouth engendered by primary diseases elsewhere—must be referred to; just as it is certain that infection of the mouth may determine infection in other parts of the body, so intestinal and blood affections directly influence the oral cavity. Ulcerative stomatitis commonly affects ill-fed and ill-nourished children; previous intestinal disease is invariably present. In scurvy the mouth symptoms are preceded by the blood changes, in sprue oral ulceration may precede, but often follows intestinal inflammation, while in certain specific blood diseases, notably lymphatic leukæmia, acute generalized hypertrophy of the gums is a frequent symptom. Further, as Lane has pointed out, colon stasis may have as a symptom atrophic and hypertrophic gums. Even in secondary anæmias, and many are of intestinal origin, the gingival margins become atrophic, the tissue flabby and easily infected by various micro-organisms; but whatever the predisposing causes, the liberating cause of the local gum disease is invariably bacterial infection. It is, therefore, of the first importance to enlarge our view and obtain a proper perspective of the correlation of disease of the upper parts of the alimentary tract to disease in other parts of the same canal.

In chronic industrial lead absorption I have shown that the metal absorbed by the lungs is excreted into the large intestine, and in most instances the presence of a blue line on the gums is a concomitant feature; I have observed, however, that when under the influence of treatment the accumulated metal is undergoing progressive excretion into the large bowel, a blue line previously absent often makes its appearance along the gums.

Injections of toxins, filtered broth cultures of mouth bacteria, into animals frequently lead to hyperæmia of the mesentery, especially of the large intestine, suggesting that excretion into the bowel is taking place along the channels which I have experimentally shown are those through which lead is eliminated. By feeding guinea-pigs (twenty-four) upon groats only with a very small proportion of bread and milk at first, and afterwards with groats alone, I succeeded in producing all the signs of pyorrhœa alveolaris. All the teeth became loose, and could be moved with the finger; some actually altered their positions. Even if cabbage was given at this stage the animals did not recover, but died. The post-mortem signs were those of generalized hæmorrhage, including the large and small intestines. Some of the animals showed suppuration around the teeth, but there was no hypertrophy of the gums. In dogs and cats the fixation of a gold band around one of the teeth, well under the gum, produced local alveolar infection. Further, I find peptic digestion *in vitro* of broth cultures made from the pus of pyorrhœa alveolaris reduces the virulence, although in the case of *Bacillus pyocyaneus* I have successfully immunized animals with the peptic digest of killed agar cultivations.

To epitomize, affections of the portals of the alimentary canal produce secondary changes in the whole organism, not so much by the passage of their products into the stomach as by the entrance of bacteria and their toxins into the blood-stream in the affected areas, while, conversely, disease processes of the lower parts of the alimentary canal or the blood may affect the oral tissues.

In the treatment of such disease as chronic oral or nasal suppuration, both local and general measures are essential—local, to remove the affected areas; general, to eliminate the infecting agents. Further, if the best results are to be obtained, rational and scientific steps must be taken to increase the vitality of the damaged tissues before operative interference is proceeded with. That is to say, the general and local resistance of the tissues should be increased by means of autogenous vaccine prepared from the infecting organisms controlled by careful observation and examination, and followed by surgical treatment upon ordinary surgical lines. In those cases where there are no generalized symptoms, where the localized infection has not overcome the resistance of the body generally, when the constitution of the blood shows no wide divergence from the normal, immunization is not essential, but is useful; but if operative interference is contemplated in the presence of secondary anæmia and other signs of general diffusion of the disease, vaccine therapy is absolutely necessary to ensure success.

It has been stated by Colyer that he has never seen a case of pyorrhœa cured by vaccines, but I gather from his remarks that if a case of pyorrhœa is treated and recovers, it is thereby shown not to be pyorrhœa, whereas if it does not recover then the treatment is of no avail in any case.

Very considerable misconception exists as to the use of vaccines. They are not to be used to the exclusion of other methods of a surgical nature, but, as I have outlined, must be used in an intelligent manner, and in conjunction with and in addition to local measures. I entirely and emphatically dissent from the proposition that the only remedy for pyorrhœa is extraction, and I regard as a grave confession of failure the statement that it is impossible to produce efficient drainage in the pockets or interdental spaces, but if immunization be properly carried out there is no difficulty whatsoever in resecting the infected areas when large or removing them with the actual cautery when small. Where the individual teeth are so far damaged that more than a third of the attachment has disappeared, the matter is somewhat different and extraction may then be necessary, but the mere removal of the teeth does not invariably remove the disease in the bone, the gums constantly remain sore, intolerant of the artificial substitutes, the arthritis continues to progress, and in many cases the anæmia persists. Radiograms of such cases show small but definite areas of localized rarefaction in the jaw from which I have many times succeeded in obtaining cultivations of the organisms causing the general symptoms. Such cases are far commoner than those who advocate general extraction will admit, in fact their existence is often denied, but I have under my care at the present moment eight such cases.

The extraction of all the teeth in a young adult suffering from a mild degree of pyorrhœa, with perhaps a quarter of the alveolar margin of the tooth-sockets lost, a by no means uncommon circumstance, appears to me, after a careful consideration of the pathology of the disease, an unwarrantable and barbarous proceeding, and hardly in keeping with recent advance in modern surgery. I have only recently seen two cases in which the affection had progressed to the extent of destruction of a third of the tooth-sockets, which I had treated some two years ago with immunization and local surgical measures. Both of these cases had recently been attended by dental surgeons, one in Switzerland and one in this country. Both of these informed their patients, who had requested special precautions on account of previous gum disease, that there was no sign of pyorrhœa, nor could they have ever had it, as it was never cured and they were; a more unbiassed opinion is hardly possible.

Mr. E. C. HUGHES: If one examines the peritoneal cavity of adults, either during operation or in the post-mortem room, one is continually coming across peritoneal adhesions and bands which are not present at birth, and this without any sign of inflammation in the various viscera contained in that cavity. These bands are present at certain situations and for the most part in the neighbourhood of the junction of a fixed and a movable part of the intestine—i.e., pylorus and duodenum, duodenum and jejunum, ileum and cæcum, hepatic flexure and transverse colon, transverse colon and splenic flexure, and the iliac and pelvic colons. These bands vary in size and may be only narrow adhesions or may be developed into definite sheets.

Mr. Lane has taught for a long time that these bands are evolutionary in origin; that is to say, they are developed to meet the downward drag of the intestines due to the upright position, and my own observations convince me that this is the correct explanation. These bands in the neighbourhood of the termination of the small intestine and the beginning of the larger are two in number, excluding for the moment the changes in connexion with the appendix. The band in connexion with the small intestine is that responsible for the ileal kink, and anyone who will look at the peritoneum at this part in a series of cases will see the following changes: At first, on the under surface of the mesentery near its termination, an opacity develops which is a thickening of the peritoneum; this gradually extends up to the ileum to its anti-mesenteric border about 3 to 4 in. from its termination, rotating it as it does so; at the same time it draws the ileum towards the posterior abdominal wall, until, in an extreme case, it is pinned down against the brim of the pelvis. The fully developed kink and band then show three chief characteristics:—

(1) It is 3 to 4 in. away from the termination of the ileum, never actually at its termination.

(2) The band is attached to its anti-mesenteric border, rotating it.

(3) It is only the under surface of the mesentery which is affected.

Whenever this condition is met with it has these characteristics. Can it possibly be the result of an inflammation of the appendix? Surely if it were so there would be some variation in the characteristics; it would be expected to occur at the termination of the small intestine, to affect both surfaces of the mesentery, and it would not constantly affect the anti-mesenteric border. Further inflammation in the peritoneal cavity is so variable in its results that it is unthinkable that it should be followed in 100 per cent. of cases by the same result. The band, too, in connexion with the large intestine is constant in position, varying only in the degree of its development. It may be in the shape of separate peritoneal adhesions or it may be in the form of a definite sheet, and in many cases the sheet is so definite that the finger can be passed beneath it so as to be between it and the normal reflection of peritoneum from the ascending colon on to the posterior abdominal wall. This band passes outwards towards the junction of the posterior and lateral abdominal walls and traced inwards it passes over the ascending colon. When present it does not vary; it never passes in any other direction, it is never attached to the anterior abdominal wall. It is not possible to believe that inflammation here will always end in a constant result.

There is no doubt but that the ileal band and the colon band are evolutionary, and are developed to prevent these parts of the intestine from dropping down into the pelvis. It is said that these bands are infrequent in their occurrence. That is not so. They are extremely common. My experience is a comparatively short one, but I am constantly meeting with cases showing these bands and the others described by Mr. Lane.

The upright attitude is followed by important changes in the position of the appendix. The normal appendix is a freely movable piece of intestine with a mesentery of its own, of greater or less length. In adults, as far as my experience goes, it is more common than not to meet with an appendix abnormally situated. The abnormality takes one of two directions. One or other of the evolutionary bands picks up the appendix either in whole or part, so that its mobility is impaired by its attachment to the band. The other abnormal condition, which is probably the commoner, is the adhesion of the appendix to the under surface of the mesentery of the small intestine. In this condition the course of events is as follows: The proximal part of the appendix becomes attached to the under surface of the mesentery, and gradually more and more becomes attached in an upward and inward direction, until the whole organ is adherent, or the greater part is adherent with the terminal inch free. In this condition the appendix is acting as a ligament to the cæcum, suspending it in order to prevent its downward displacement.

It is said that these bands, whatever their origin, are of no importance. Consider their effect on the appendix. This organ, being adherent in whole or part, has its mobility impaired, with the result that its ability to discharge its secretions into the cæcum is lessened; and that this is the case is shown by the presence of fæculent contents in its interior, the normal free moving appendix always being empty.

A further stage is the accumulation of fæcal contents, its inspissation, and its conversion into the fæcal concretion of the appendix. This retention of fæcal material will lead to inflammation of the mucous membrane, since conditions are very favourable to the development of micro-organisms in an appendix, which cannot discharge its contents efficiently; and the intensity and extent of the inflammation will depend on the virulence of the micro-organisms, their quantity and quality, and the resisting power of the individual. An appendix adherent in part with a freely moving termination is liable to torsion or kinking taking place at the junction of the fixed and mobile parts. I believe that nearly all cases of acute appendicitis are primarily due to the fixation of the appendix in the two ways described.

With a totally adherent appendix and consequent retention of products the inflammation begins from the interior and spreads externally, producing practically a cellulitis, which may cause gangrene of the organ, or it may stop short of this and produce an empyema of the appendix. This type of appendicitis results in an inflammation of a considerable portion of the appendix, but there is nearly always a small portion of the cæcal end which is normal. With an appendix partially adherent, and with the terminal part free, inflammation is due to the deprivation of blood to the latter part owing to it and its mesentery kinking or rotating. In this case, if the blood supply is cut off for a sufficiently long time, gangrene of the extremity occurs, or if for not so long, on the kink or twist being corrected, the free part becomes intensely congested, and after a time becomes normal again. The symptoms of the former type are far more severe than those of the latter, because the appendix has been for some time practically a culture tube for bacteria, and contains an enormous number of virulent organisms; while in the latter case it is merely a gangrene of the terminal part occurring suddenly, and there has been no such opportunity for the bacteria to increase in numbers and virulence.

It is because of the impossibility of knowing which type of appendix is at fault that no one is able to foretell the result of an attack of acute appendicitis, and why surgeons at once operate to remove the appendix. Just as acute appendicitis is due to the abnormal anchoring of the appendix, so frequently is the condition of chronic appendicitis, or appendicular dyspepsia. In this disease there is discomfort or pain in the right iliac fossa, pain on pressure in this region, and the inflation of the colon with air, or displacement of gas into the cæcum by pressure over the pelvic colon, produces pain over the appendix region. The explanation of these signs depends on the adhesion of the appendix; the cæcum by increased pressure within forces an appendix, inflamed to a greater or less extent, against the iliac fossa, or increases the kink between the fixed and mobile parts, with the result that pain is produced, and removal of the organ by operation cures the local symptoms.

In addition to these local signs of disease, there are others simulating duodenal ulceration which are assigned to the diseased appendix, and there is no doubt that a number of such cases are cured by removal of the appendix, but it is also certain that a number of them are not benefited at all, and I think it likely that those who believe in appendicular dyspepsia, and remove the appendix for its cure, feel very doubtful as to the relief their patients will get when they come across and remove an apparently normal appendix.

The cases which will be relieved are those where the appendix is adherent to the mesentery of the small intestine, and where there is no ileal kink, and I think Mr. Lane's explanation is correct—viz., that the appendix in these conditions is acting as a ligament to the cæcum and as it passes upwards behind the termination of the ileum obstructs it, and causes a delay in the flow of the ileal contents into the cæcum.

Up to the present in my own cases I have not met with an extreme ileal kink, and I have been content to divide the band. Whether this is sufficient I am not yet sure. But it is incorrect treatment if the kink is an extreme one, as I have seen death from general peritonitis follow this treatment on at least two occasions.

I have seen a good deal of Mr. Lane's operative treatment by short-circuiting, and the benefit in a few days is most marked. The pigmentation around the eyes rapidly disappears and the complexion becomes good, and various symptoms of auto-intoxication disappear. I was much struck recently by a woman who had intolerable itching of the skin, and whose abdomen at the operation was covered with excoriations, the result of scratching. She expected that the nights succeeding the operation would be miserable on account of the irritation, but to her surprise she had no more itching and was able to sleep with comfort. I saw the case of Still's disease, which Mr. Lane has mentioned, and I have seen cases of rheumatoid arthritis, where the immediate benefit was marvellous. I am convinced that Mr. Lane's explanation of intestinal toxæmia is correct, but I am not yet sure how far medical treatment can benefit the condition.

The Royal Society of Medicine.

April 28, 1913.

Sir FRANCIS H. CHAMPNEYS, Bt., President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.[1]

DR. WILLIAM HUNTER, in reopening the discussion, said that the title selected for it was a veritable "Challenger" expedition sent out widely on the ocean of troubled waters of the abnormal processes of the alimentary tract and of the general systemic effects on the blood and organs connected therewith.

SCOPE OF THE DISCUSSION.

The subject matters included were of special interest to him. They practically covered the whole series of processes which had been the special subject of his interest and investigations during the past thirty years. Alimentary processes began in the mouth, were continued in the stomach and intestine, extended to the portal blood and might be said to end in the liver. His own studies had commenced in the liver, had passed back to the portal blood, thence to the intestine and stomach and had finally ended fifteen years ago at the mouth, in the great practical outcome of the final recognition and description of "Oral Sepsis" as a great cause of disease. In the course of these studies relating to the nature of at once the rarest and severest form of blood disease—"pernicious anæmia"—he had practically spent the greater part of his time in exploring in all their depths, superficial, middle and deep, the muddy waters of the intestinal processes now brought under review. As the first and most important outcome, from a physiological as well as pathological point of view, he was enabled after three years experimental and pathological work (1885-88) to trace back this great anæmic disease to destructive (hæmolytic) changes affecting it in the portal blood, induced by some special hæmolytic poison reaching it from the intestinal tract. These studies had founded and formed the basis of what had become to be termed "The Doctrine of the Enterogenous Origin of Severe Anæmias," or "The Toxic Hypothesis" of the origin of these anæmias associated with his name and work. But although termed a "doctrine" or "hypothesis," it was, so far as his own work was concerned, in no way any initial speculation or theory but an actual interpretation of the facts he had ascertained experimentally, viz., that the blood changes of the disease he was studying could only be produced by special poisons which he employed. They were "toxic" or "toxæmic"; but these conclusions and terms had only been in his hands a new staff to help him on to further investigations as to the site of the lesions and the source of the infection which produced this particular hæmolytic toxin. These studies pursued without intermission in all directions, both pathological, clinical and ætiological, for ten years led him eventually step by step from the intestine to the stomach, and finally to the tongue and to the discovery in these three organs of the infective lesions which always underlie this grave and utterly mysterious disease. They had also, however, had a far greater and far more important practical outcome. For they had also led him for the first time to recognize the far-reaching practical, ætiological and clinical importance of the glaring septic lesions in the mouth, termed by him "oral sepsis" as the greatest cause of infective disease in the body, and the chief cause of most of the disturbances in the alimentary tract, generally classed as toxæmic.

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[1] Adjourned from April 21.

In respect alike to the rarest, most mysterious, and without doubt the most severe toxæmic blood condition observable in disease, and in relation to the commonest, most obvious and without doubt the most prevalent, potent and neglected infection in the body—viz., oral sepsis—and the numerous and varied disturbances of the stomach and intestine connected therewith, his observations had always led him back to definite infective lesions in the mucosa as an invariable accompaniment of their symptoms. And the important conclusion he drew from this was, that whatever rôle toxæmia, apart from lesions of the gastro-intestinal mucosa, played either in health or disease (and it did occur) from an ætiological, pathological, clinical and practical point of view it was much more important to recognize that definite infective lesions, which were easily recognizable and in many cases could be easily dealt with, underlie nine-tenths of the disturbances of the alimentary tract which were termed "toxæmic." Such was the chief thesis which he would lay before them to-day as his contribution to the discussion and which he would support by demonstrating to them by means of the epidiascope the obvious lesions in the alimentary tract which underlie so much of its so-called toxæmia.

RESULTS OF THE DISCUSSION.

The result of the discussion has been a big haul containing all sorts of fish, both small and great, from the sprat of the rash produced by eating shell fish to the veritable whale of chronic constipation—fairly landed on the shore, colon and all, under the euphonic new name of "intestinal stasis" given to it by the surgeons in charge. After undergoing the surgical and anatomical dissection or vivisection to which it was now being subjected, he thought, or at least hoped, that for the future most of the other members of this great genus would be allowed as heretofore to cruise in the more friendly waters of medical practice, to be driven off when a nuisance by the kindlier measures heretofore adopted rather than by the ruthless slaughter proposed for them. This result was the more desirable and the more probable, since in his experience, and it was a wide one, of intestinal toxæmia and its effects, it was a notorious fact that the disturbances and effects connected with such processes were in 99 per cent. of cases associated with the opposite condition of bowel—viz., looseness and diarrhœa. The most important content of the haul was in his opinion the number and variety of specimens of the great genus of shark—of "oral streptococcal sepsis" which had been landed by various speakers—the genus which he had reported on some fifteen years ago, and frequently since as common in the waters of gastro-intestinal processes and the cause of so many of the disturbances to which they were liable. He proposed as his chief contribution to the day's discussion, to show by means of the epidiascope all sorts and sizes of this particular genus—which he would designate "the shark of the intestinal tract"—and of the multiple effects which it produced not only in that tract but elsewhere. He hoped he would thereby make it clear that to describe such gross and obviously septic lesions of the mouth as he now showed under the euphemistic and belittling title of "toxæmia," was as illuminating and as helpful as if one applied the term "toxæmic" to a foul septic knife or sponge.

To bring Oral Sepsis—as it happened for the first time—under discussion as only a form of "alimentary toxæmia" was as if some thirty years ago the whole subject of sepsis and antisepsis in surgery, of the importance of sepsis in wounds of the skin, had been for the first time drawn attention to in a general discussion under the title of "cutaneous toxæmia" grouped with rashes and all other affections of the skin.

For the chief historical feature of this discussion would in his judgment prove to be the continual reference made by speakers in all departments of medicine and surgery to the importance of Oral Sepsis in connexion with the diseases of the mouth, throat, stomach, intestine, and colon; of rheumatic diseases, eye diseases, ear diseases, skin diseases, blood diseases, and nerve diseases—the whole range of subjects in his experience in many cases most important manifestations and results of Oral Sepsis. Beyond all doubt this condition was the most potent and prevalent infective cause of disease in the body and deserving the attention of every branch of medical work, and no branch more specially than that of the surgery of the intestinal tract.

To have the name "Toxæmia" or "Toxæmic" attached to processes occurring in wounds obviously septic and easily accessible to treatment was, in his opinion, then, a great mistake which would do infinite damage to the whole subject of "Medical Sepsis"—namely, the importance of sepsis in causing medical diseases; to the great department of dental surgery, which was especially concerned with these disease processes in the mouth; and lastly to surgery in its interpretation and treatment of the conditions occurring in the gastro-intestinal tract.

CAUSES OF INTESTINAL TOXÆMIA IN HEALTH.

The sharpest and broadest distinctions existed and should be drawn between processes in the mouth and processes in the gastro-intestinal tract in relation to the subject of "intestinal toxæmia or auto-intoxication." The mouth processes were not toxic in their character in health. If they had been so, Nature, who was a careful dame knowing her own business best, would have provided for it by connecting up the mouth with the portal circulation. The toxicity of the gastro-intestinal processes, on the other hand, was a great physiological fact, as great as that of the circulation, respiration and renal excretion. Like these processes it was provided for by a special structural mechanism to deal with it—viz., a special portion of the circulation, the portal, for the immediate receipt of the multiple products of digestion, guarded at its entrance, as a first line of defence, by the extraordinary mass of active cells of epithelial and lymphoid nature, and the powerful muscular and nervous apparatus of the gastro-intestinal walls; and guarded at its other end by a still more powerful fortification, the liver, one of whose chief functions was the detoxication of the portal blood. The portal blood was always toxic even in health, the liver was the great anti-toxic and excretory organ of the portal circulation, just as the kidney was the great excretory organ of the general circulation. This toxicity was not an accident, nor did it depend on any one product of gastric or intestinal digestion. All attempts to connect it with any such product —e.g., indol—had in his opinion been futile and all such attempts were bound to be futile, and he entirely agreed with one of the earlier speakers (Dr. Ledingham) with regard to the inconclusive result of the work of French observers in this relation.

Evidences of Natural Toxicity of the Portal Blood.

Far more conclusive evidence of the natural toxicity of the portal blood was in his observation and judgment presented every day in the facts of disease—in the disturbances connected with inefficient activity or actual disease of the liver. These ranged in character and degree from the headache, giddiness and depressant mental effects of the simplest attack of "biliousness"—to the toxic phenomena of "Cholæmia" which so constantly were the termination of cases of severe liver disease—up to the fulminating toxic phenomena of post-chloroform poisoning and acute yellow atrophy. The most common evidences of portal toxicity were in his judgment those presented daily in surgical practice in the phenomena of the sickness, vomiting, headache and sense of illness following the administration of anæsthetics, more particularly after prolonged administration. These were not due to the poisonous action of the anæsthetic *per se* on the nervous system; nor yet —as was so often and fallaciously supposed—to the presence of food in the stomach or intestine. They were the direct result of the depressed activity of the liver cells induced by the anæsthetic, and the consequent imperfect detoxication of the portal blood passing through the liver. Under certain circumstances—fortunately rare in combination—this depression could be carried too far. The energy of the liver cell already depressed by disease and intensely aggravated by the *unsound clinical practice of withholding food unduly* from the patient prior to operation completely failed. It became in consequence the victim of its own digestive ferments and its own self-digestion, with resulting acute fatty degeneration and acute necrosis, as in the specimens he now showed. The portal blood passed unchanged into the general systemic blood, and we had presented the fulminating toxic disturbances of continuous sickness, vomiting, hæmatemesis, delirium and coma—characterizing post-chloroform poisoning—precisely the same phenomena producible experimentally (in Eck's fistula) by connecting up the portal vein directly with the inferior vena cava.

CAUSES OF PORTAL TOXICITY IN HEALTH AND DISEASE.

These were, in his judgment, of two classes :—

(1) *Non-Infective Causes.*—Those of a non-infective nature were the various products of digestion formed by the ferments of the salivary, gastric, pancreatic and intestinal digestion. The toxicity of many of these products, especially those of pancreatic digestion, where disintegration of proteids was carried farthest—was considerable and constant. But if it was confined to such products, intestinal toxæmia would play a limited part in disease. For the powers of the gastric and intestinal mucosa and submucosa and of the liver were ample enough to deal with all such products.

(2) *Infective Causes.*—Far more important as a cause of natural portal toxæmia were the various products formed by the action of bacteria on the food products.

The Coli Group.—In health the chief and most abundant were those of the *coli* group. These were the natural inhabitants of the intestinal tract. They were naturally intensely putrefactive in their action, carrying the natural disintegration of proteids much beyond the limits effected by pancreatic digestion. The degree of putrefaction varied according to the amount of proteid in the food on the one side and according to the abundance of *Bacillus coli* on the other. The provision made by nature for due regulation of the latter was, *first*, the daily removal of the mass of excrement—alike of food and of organism in the fæces—and, *secondly*, the provision of protective mucus which lined the whole gastro-intestinal tract, and of protective muscular movements which kept the whole gastro-intestinal contents in more or less continuous movement. In short, they were being continuously "moved on." Chronic constipation was therefore a highly unnatural condition and always a source of increased portal toxæmia. That it might exist in certain individuals as an almost permanent condition without apparently causing ill-health, was solely due to the power and protective action of the liver. It was not any evidence of the comparative harmlessness of constipation *per se*, but only an evidence that some individuals possessed the cæcum and colon of an ox with the liver of a pig, capable of doing any amount of detoxication.

The Lactic Acid Group. — The bacterial flora of the intestine included many other organisms, which contributed to the sum total of toxic and non-toxic products, formed from the food and absorbed into the portal blood. The most important of these from a health point of view were the lactic acid group—the group which specially, although by no means exclusively, were concerned with the carbohydrates of the food. The influence of this class of organism in modifying the character and lessening the degree of putrefactive changes in the intestine was in his opinion great and important. It had been investigated by him both by experiment and by clinical studies some twenty-five years ago, and recorded by him before the Royal Society (1890), the investigation being so far as he knew the first of the kind that had been made. He found in a series of parallel studies made with equal quantities of meat allowed to putrefy—under similar conditions—that the addition of sugar to the putrefying mass profoundly modified the character of the chemical changes. The amount of putrefactive diamines (cadaverine and putrescin) was reduced to a twentieth part of that formed when no sugar was added. He had subsequently (1889) made similar studies on the effect of carbohydrate diet on intestinal putrefaction both in man and in animals, with the same result, and had found even in the short period of two days that the substitution of a farinaceous for a meat diet reduced the amount of putrefactive (aromatic) sulphates of the urine to a third or a fourth of their previous amount—viz., from a total excretion of 0·1258 grm. *per diem* to 0·0396—changing the ratio of free sulphates to aromatic from 5½ : 1 to the strikingly low ratios of 17 : 1. He had also obtained and had since frequently observed similar results in cases of disease, a marked reduction in putrefactive sulphates or of indican (a much simpler guide) as the result of carbohydrate diet, or of the introduction of lactic acid organisms by means of sour milk into the intestine. The lactic acid group were the least harmful and most beneficent group of organisms in the alimentary tract. There were therefore, in his opinion, absolutely sound physiological, pathological and clinical grounds for the use of sour milk in conditions of intestinal putrefaction.

The Streptococcal Group.—The third group of organisms present in the alimentary tract in health was the streptococcal group. It was always present, but little obvious in health, playing probably nothing but a saprophytic part in the intestinal processes. But in the light of all his pathological, bacteriological and clinical experience, it played an altogether different and mightily important part in diseases. It was responsible more than all the other normal organisms of the alimentary tract for the various disturbances and lesions of that tract which underlie the clinical conditions generally termed "toxæmic." There was a great pathological reason for this, viz., that the streptococcal group, however few or harmless they might be, or appear to be, had always an intense capacity for local invasion, for causing local lesions and for persisting in such lesions after they were formed. In this respect their power was quite different from that of the organisms of the *coli* group. These latter had also powerful infective and inflammatory properties, and without doubt they were capable of producing congestions, inflammations, and ulcerations of the intestinal mucosa, or of powerfully contributing to such conditions produced by other infections—e.g., dysenteric, typhoid, tuberculous, &c. But they were extremely motile (while streptococci were notoriously, as in erysipelas, very creeping and invasive in their progress) and they only manifested their full infective power—in causing inflammation, ulceration, suppuration and necrosis—when they were shut off temporarily or permanently from the intestinal tract—e.g., in gall-bladder, appendix, or after perforations in the peritoneal cavity or adjacent tissues. In health the number of streptococci was so moderate that amid the healthy conditions prevailing along the intestinal tract—the presence of protective mucus, the continuous movement of the food products along that tract—they enjoyed a saprophytic existence amidst the abundant food products. But their presence in that tract was nevertheless always important. For they were always capable of taking advantage of any abnormal conditions—whether of the food or of the mucosa—and of becoming thereby not only saprophytic but actively pathogenic, both by increase in their number and by actual invasion of the mucosa. The best example of this was afforded in the case of the summer diarrhœa of children, all severe cases of which were characterized by the great abundance, sometimes to the extent even of preponderance, of streptococci in the intestinal flora. In view of the possible rôle which streptococcal organisms could play in intestinal processes, it was in his opinion important to keep a close watch on the conditions that favoured their presence or led to any accession of their numbers and virulence. Such conditions were in his observation pre-eminently furnished by the septic wounds of the mouth, the conditions termed by him "Oral Sepsis" (1900). For these were not only caused chiefly by streptococcal organisms, but they formed a perfect hotbed for their cultivation, both as regards numbers and virulence. They therefore furnished a continuous supply of streptococci to the alimentary tract, lower down, much of it doubtless exterminated by the bactericidal action of the gastric juice, when the digestion was in progress. But much of it equally without doubt passed unscathed through the stomach, especially in the interval between meals, and during the night when gastric digestion was at an end, into the intestinal tract, where they found everything ideal—short of this periodic removal by the peristaltic action of the bowel—for their propagation and persistence.

He had endeavoured to fasten attention on the rôle played by streptococci in intestinal disturbances, looseness, diarrhœa, &c., and lesions—e.g., congestions, inflammations, ulcerations, &c.—by giving to these disturbances and conditions the title of "septic gastritis," of "septic enteritis," meaning thereby "streptococcal enteritis," "septic colitis," "septic proctitis," "septic appendicitis," "septic cholecystitis," &c. He thought the time had come when ætiological titles of this kind should be attached to gastric and intestinal lesions rather than continue to be satisfied as heretofore with purely pathological titles, such as "gastric catarrh or ulcer," "intestinal catarrh," "colitis," "ulcerative colitis," &c., or still less with such vague names as "toxæmia." These names served to distinguish a definite septic ulcer or wound of the gastro-intestinal mucosa just as little as the names "cuticular erythema or dermatitis or ulceration" would help to characterize a septic wound or abrasion of the skin or an actual erysipelas. And as already said, the term "toxæmia," or "toxæmic," applied to such septic lesions in the mouth was, in view of their glaringly septic nature, quite indefensible.

Adventitious or Occasional Infections.—Under this title might conveniently be grouped the many other organisms that either occasionally or constantly got access to the alimentary tract through the mouth and nose in health. They were a large group—of little importance in health—for they were entirely saprophytic. But even their presence was an interesting physiological fact. It showed—what was continuously also shown in disease, but no less constantly belittled—that the bactericidal power of the gastric juice was a very much overrated property; that it did not suffice to keep even the most harmless organisms from getting into the intestinal tract, and the whole experience of disease testified to the fact that it was incapable of arresting the entrance of typhoid, tuberculous and many other infections. The existence of this group in health was therefore an important fact in connexion with disease. For the specific infections—e.g., typhoid, amœbic dysentery, tuberculosis, and many others were of this adventitious or occasional character—gained easy access to the intestinal tract and formed what we then termed the *specific infections* of that tract. To this group belonged in his observations the most powerful and remarkable of all gastro-intestinal infections—viz., the *hæmolytic infective organism* which was the cause of the remarkable form of infective disease termed "pernicious anæmia." He had definitely ascertained and satisfied himself as to the sites and characters of the infective lesions of this grave disease and as to their constant and distinctive character—viz., in the tongue, gastric mucosa, and intestinal mucosa. This was the disease, at once the rarest and severest of all blood diseases, which after pathological and experimental studies extending over three years (1885-88) he was first led to trace back to destructive changes in the portal blood induced by poisons and produced in and absorbed from the intestinal tract. It was these studies that established what was called "the doctrine of the enterogenous origin of severe anæmia," or "the toxic hypothesis" of their nature associated with his name. But they also led, as already described, to the result of far-reaching practical importance which had been testified to by every speaker in the discussion—viz., the recognition of the overwhelming importance of "oral sepsis" as a prevalent and all-potent cause of gastro-intestinal disturbance and disease.

CAUSES OF INTESTINAL TOXÆMIA IN DISEASE.

The alimentary tract was thus a river of mighty muddy waters, like the oldest and most remarkable prototype of all rivers, the Nile. Like the latter it derived its chief characters from three great sources of supply.

(1) *Food Supply.*—The first of these was that which brought the food and water supply, like the White Nile rich in nourishing material which nourished and fertilized the whole of the body, but carrying with it much debris, liable to be deposited all along its track and specially accumulating in its lower reaches; continually drawn on its passage downwards and doubtless also having no little material thrown into it; subject, like its prototype, to great variations in the quantity or quality

of its supply and to the effects of these on its channel banks; periods of rise or increase, alternating with periods of decrease or fall; periods of drought—to wit, constipation, alternating with periods of flood and overflow—to wit, diarrhœa.

(2) *Bacterial Supply.*—The second great source from which the intestinal canal derived another of its prominent features was from a great tributary which joined it lower down—like its prototype, the Blue Nile—viz., a large supply of bacteria, chiefly coli bacilli, which profoundly modified the quality and quantity of its food contents in various ways, adding much new material and sediment and affecting materially their nourishing value. This supply, like that of the Blue Nile, was always continuous, but was subject to periodic variations, and when in overflow capable of producing marked disturbances, even greater than those connected with variations in the food supply.

(3) *Streptococci.* — Although the chief organism in health, the *Bacillus coli* was by no means even in health the only source of supply of bacterial activity. The flora and fauna of the intestinal tract was abundant and in particular cases was constantly receiving, temporarily, supply from other classes of organisms both saprophytic and pathogenic. But there was one supply which, in no way so abundant as that of the *coli* group, was always there (like the third tributary of the Nile, the Black Nile), and like the latter always affected the character of the waters of the alimentary tract. This was the supply of streptococci, intermittently reaching it from oral sepsis of the mouth. The feature of the supply of the Black Nile was its intermittent character, sometimes running dry, sometimes overflowing, but always recognizable when in flood by the red character it gives to the other waters which it joins. As with it, so with oral sepsis and its effects. When in excess it was always recognizable by its effects on the gastric and intestinal processes lower down. When in overflow, it produced a more marked and more easily recognized effect than any infection other than perhaps typhoid or tuberculous infection, capable of profoundly modifying the severity even of these infections. It produced inflammatory ulcerative and even necrotic lesions of the most pronounced character, and short of producing these latter results it was capable of producing the most pronounced functional disturbances. It could, in short, and frequently did, make the whole alimentary tract run red with disturbances to a degree which had more and more impressed him with its importance and which had led him to recognize streptococcal sepsis—viz., "oral sepsis," "septic gastritis," "septic enteritis," and "septic colitis," &c.—as one

of the commonest and most important causes of disturbance throughout the whole alimentary tract.

The reasons why the mouth was so important in relation to septic infections generally were the following:—

(*a*) The structure and function of the gums and teeth exposed them in a special degree to violence and to injury and to consequent contraction of septic wounds of the gums and periodontal membrane (periosteum of the bone socket). This periosteum was the *only* one in the body, he pointed out, which came to the surface of the body—viz., at the neck of the tooth—and was protected from injury and infection solely by a thin layer of gingival epithelium. It was therefore peculiarly prone to injury and to infection, and when infected the infection was in immediate contact with bone tissue, both of the socket and the root of the tooth.

(*b*) When infection took place, it was, from its situation, particularly difficult to eradicate after it had got into the socket. It became, therefore, a chronic condition, and if, as frequently happened, the infection invaded the tooth itself, either directly or indirectly, from the periodontium, the result was a septic wound or a series of such wounds of the tooth or socket of great chronicity and far greater virulence than any similar wound of the same size anywhere else in the alimentary tract or in any other soft tissue of the body. For it was connected with bone tissue, and as he had emphasized in his first papers, and once more pointed out, bone sepsis was far more virulent and toxic in its effects than sepsis of soft tissues, and sepsis of the teeth and periodontium was always essentially bone sepsis. (He showed a large series of specimens illustrating the conditions of gums, sockets, and teeth, continually found in patients.)

(*c*) Lastly, the special feature of oral sepsis and of the septic wounds which it created and by which it was further propagated—the feature which distinguished it from septic wounds and lesions of the gastro-intestinal tract was that these wounds were in direct relation to the systemic blood without any intervening detoxicating organ like the liver to arrest or modify the toxins which they produced or the extension of the infection and the blood.

(*d*) Oral sepsis was, therefore, a double-edged weapon of offence in relation to the blood. It could act on the blood directly—and constantly did so. But the continuous strain of streptococci which it propagated passed also over the tonsils and pharynx into the gastro-intestinal canal where it modified the existing normal bacterial activities and produced in varying forms and degrees all the effects of congestion, inflammation, and ulceration, which it produced in and around the teeth. These included acute, subacute, and chronic septic tonsillitis, and pharyngitis; acute, subacute, and chronic gastritis, with formation of gastric erosions and ulcers; acute, subacute, and chronic congestions of the small intestine, appendix, cæcum and large intestine, and of the rectum, with and without ulceration, with or without formation of bands; conditions of septic "enteritis," colitis, proctitis, appendicitis, &c. All these conditions were obvious and recognizable and could be seen in the specimens he now showed. And the point he desired to emphasize, as the outcome of his experience and close pathological study of the intestinal tract for many years, was that definite infective lesions of the mucosa of the gastro-intestinal tract were far more often present than was recognized or described in cases of so-called toxæmia and in disturbances of the alimentary tract during life; that in a very large proportion of cases streptococcal infection was a common and important cause or aggravant of such lesions and disturbances; that fresh supplies of virulent streptococci were constantly poured into that tract from the mouths of those suffering from oral sepsis and reached the intestinal tract, where they found all the conditions favourable for their further growth and increase.

Lastly, the final proof of the importance of oral sepsis was the crucial one of result—viz., that on removal of the oral sepsis the conditions of gastritis, colitis, anæmia, eye affections, rheumatic affections, nervous affections, &c., forthwith disappeared, in many cases in his experience like magic, and the patient who was previously the victim of what was deemed to be a chronic malady became restored to complete health, sometimes in the course of as many weeks as the years of his chronic illness. The following case was one of many which had come under his care.

Oral Sepsis as a Cause of "Septic Anæmia." (Hunter.)

Date		Red corpuscles	Hæmoglobin
September	8	41 per cent.	40 per cent.
,,	14	(five roots extracted)	
,,	20	49 per cent.	58 ,,
,,	25	75 ,,	75 ,,
October	18	84 ,,	84 ,,
November	17	91 ,,	94 ,,

Sir BERTRAND DAWSON, K.C.V.O.: Although the terms ("Alimentary Toxæmia" and "Intestinal Stasis") are open to criticism, they convey to our minds a definite clinical picture. The sallow, dirty complexion, the inelastic skin, the dusky lips and nails, the dirty tongue, evil-smelling breath, constant abdominal discomfort of one kind and another, the doughy, inelastic abdomen, cold extremities, the physical and mental depression, are among the prominent features. That such a condition may be produced by colon block and stasis is, I think, clear; that, when thus produced, it is curable by putting the colon out of action by short-circuiting is demonstrable; and where cure does take place, the restoration of the patient to health after years of illness is so remarkable as to make it clear that a potent factor, if not the actual cause, of the disease has been removed.

My criticism would be that Mr. Lane gives too great a prominence to motor defects. For him intestinal health is too exclusively identified with efficient drainage. I venture to think that the question needs a broader basis. The gastro-intestinal tract from mouth to anus must be viewed as a whole and from two standpoints, the motor and the infective.

Alimentary toxæmia may undoubtedly follow colon block, but mechanical delay is not a necessary condition for its existence. It may equally be caused by chronic infection, say of the gums, stomach, or appendix. Some of the most marked cases of alimentary toxæmia I have seen have been associated with diarrhœa and an empty colon. These bad cases of stasis are benefited by operation, not only because short-circuiting removes the obstruction, but because the colon is diseased and has become an active influence for evil. That the colon has become hopelessly diseased and cannot be made a working structure is to my mind the chief indication for ileo-sigmoidostomy.

The key to a condition like alimentary toxæmia is often to be found in its early chapters. The sequence in time may be, in one case, infection—stasis; and in another, stasis—infection. If an infection, it may begin in the gums, tonsil, or stomach, and finally manifest itself in the appendix or colon; or the order of the infection may be reversed. Dealing with the infective side, I would like to emphasize that, though in health the stomach is amicrobic, this is not so in disease. Acute infection of the stomach has been long recognized. It is equally true that chronic infection, usually by a streptococcus, is a definite and fairly common cause of gastric disturbance. Sometimes such chronic infection exists alone, at other times in association with, say, disease of appendix or colon.

Perhaps, if I pass to the concrete, I shall make my contention more clear. A man looks and feels toxic. He has a sallow, unhealthy skin, blue lips, dirty tongue, and often foul breath. His abdomen is frequently uncomfortable, vaguely tender, distended, inelastic. His ill-health may be divided into chapters: Chapter I began fifteen years ago, and consisted of recurrent attacks of tonsillitis. His general health was good between the attacks. Chapter II had as its chief feature chronic gastric catarrh, though often the throat would give trouble at the same time. Chapter III: The patient presented the picture of an alimentary toxæmia, and the local symptoms were mainly referable to the appendix and colon. Cultures taken from the bottom of the tonsillar crypts and from the fasting stomach, and later from the excised appendix, showed growths of streptococci which my colleague, Dr. Bulloch, investigated and reported to be identical. There was some, but not much, colonic stasis. This patient's illness was a chronic infection, probably starting in the tonsil, and extending later to the stomach and intestine. His colon, already involved on the infective side, was probably well on the road to motor failure, and if he had remained untreated, I take it he might have become a completed case of colonic stasis, and no treatment would have been efficient which did not put the colon out of action. The sequence may be reversed, and the infection may first manifest itself in either the appendix or the colon, and secondarily cause a chronic infective gastritis, with or without an ulcer. This is illustrated by the frequent co-existence of gastric ulcer and appendicitis.

A study of appendix operations brings out well the infective and motor aspects of gastro-intestinal toxæmia.

(*a*) In one group of cases, by no stretch of imagination can the appendix be said to have been the seat of inflammation. The cæcum is distended and toneless, perhaps tied down in the pelvis; the ileum may be kinked. The prime cause of the trouble in such cases is mechanical; the small intestine cannot empty, and toxæmia ensues. It may be the cause lies in the cæcum or colon as a whole, or in over-action of the ileo-colic valve. The efficient treatment lies in curing the stasis, and removal of the appendix alone is futile, as we all know by bitter experience. Here Chapter I is motor failure of the colon, and the subsequent chapters are infective or toxic, the small intestine being involved first and the stomach and mouth later.

(*b*) In contradistinction, a chronically inflamed appendix, containing muco-pus, is found, and as the result of its removal the patient is cured of his colitis or his gastritis, his tongue cleans, his breath ceases to smell and he becomes well. Here the prime cause is infective. If by ill-luck his appendix focus is not removed, it may be that his colitis will progress, and what has begun as an infection in the colon may pass on to motor-failure, and everything which Mr. Lane describes as intestinal stasis may ensue.

Colitis again is often associated with severe intestinal toxæmia. What we term "colitis" comprises conditions which are widely different. In one case, the passing of mucus, and it may be of blood, goes hand in hand with obstruction of the colon, and if the condition is severe, the only cure would be to graft the ileum into the sigmoid. On the other hand, colitis may be associated with an infection of the appendix. Or, what is even more interesting, it may be that it is the final expression of an infective attack taking its origin in the throat, stomach, or upper intestine. It is not uncommon for a patient who passes mucus in the stools to have acute attacks, which commence it may be with sore throat, or gastric discomfort, and then pass on in a few hours to pain in the colon and the passage of much mucus. I watched such a patient for some years, and finally appendicostomy was carried out, and with this result: The patient still gets the acute attacks, starting in the throat, and extending to the stomach. Shortly after the onset of the attack she will wash out her colon through the artificial opening, and in this way prevent the pain in the colon and the passing of mucus which used to ensue. The material which she washes out consists of undigested food which has obviously been hurried through the small intestine. Her toxic symptoms have been materially abated, but not cured, by the opening into her cæcum. I take it that this patient suffers primarily from an infection of the upper alimentary tract, and the so-called colitis is its final expression. There used to be a certain amount of obstruction in her colon, which was a secondary and not a primary feature.

The following case illustrates the infective aspect of alimentary toxæmia: A boy was constantly getting attacks of fever, associated with foul tongue, distended intestines, and constipation with offensive stools, but no actual pain. He was fat and flabby, and in between the attacks looked toxic. His tonsils were ragged, and in some of the attacks became inflamed. The crypts of the tonsils contained a streptococcus, a vaccine from which effected no improvement. The abdomen was opened, when a large flabby colon and a chronically inflamed appendix were observed. This appendix contained a pure culture of pyocyaneus. After the removal of this appendix, the boy rapidly improved in health.

This, I take it, is an infective case, having its origin in the appendix; but, if it had been left, he would have very likely developed an obstructed colon and the symptoms of stasis. The father of this boy used to have somewhat similar abdominal attacks, and in two of them had catarrhal jaundice. His throat became inflamed with the onset of his abdominal symptoms. In his case a streptococcus was isolated from the crypts at the tonsils, and a vaccine made from it cured the abdominal trouble and the patient markedly improved in health.

TREATMENT.

I would urge that all cases should be systematically investigated, both from the infective and the motor standpoint. In this way the patient will be safeguarded and knowledge advanced. It would be a tragic result of this valuable discussion if the latter were followed by a "fashion" for ileo-sigmoidostomy. The history of gastro-enterostomy gives a warning. It will be in the memory of all how thoughtlessly at one time this operation was performed on wholly unsuitable cases. It would not be sound reasoning to put down vague symptoms to intestinal stasis merely because there was objective evidence of colonic block. With some people a loaded colon is not incompatible with health. In estimating stasis we get valuable information from X-rays, but this information needs to be carefully compared with clinical observations. If the existence of toxæmia is well established, careful investigations will be needed to show whether the prime cause is infective or obstructive. If obstructive, I would urge careful examination of the anus and rectum. Many of these obstructed colons are due to a fibrotic sphincter ani, and by treating this condition by means of a simple operation and following it by subsequent suitable treatment of the colon, the latter may be restored to efficiency. Or again, in other cases, the simple operation of appendicostomy will suffice. In a large proportion of cases, however, systematic medical treatment, carried out with persistency, is followed by success. Such treatment comprises suitable diet, hydrotherapy, massage, exercises, petroleum oil, carefully regulated lavage, and other measures. The success of such treatment depends on careful regulation of every detail carried out in an institution, and no class of disease emphasizes more the need of sanatorium treatment. The reason why the treatment of gastro-intestinal disease is sometimes more successful on the Continent than in England is solely because the treatment is more methodical and carried out in institutions which in this country are lacking. But without doubt there remain cases where the colon is hopelessly diseased, where it is not only powerless to perform its functions, but is in some way an active influence for evil; in these suitable cases, and by suitable hands, ileo-sigmoidostomy gives excellent results and is a distinct advance in treatment.

Dr. E. MELLANBY: The aspect of the subject of alimentary toxæmia that I propose to deal with is the chemical one, for recently certain chemical substances have come under consideration which seem to throw some light on the subject and are worthy of further investigation.

Before proceeding with the consideration of these substances, I think it is permissible, even if only because of the treatment they have previously received in this discussion, to suggest that we throw overboard one group of chemical substances, which until recently have received the exclusive attention of chemical researchers on alimentary pathology. I refer to the tryptophane derivatives, and especially to indoxyl sulphate, the substance responsible for the indican test. I make this suggestion for two reasons:—

(1) That this substance, although it has been the cause of tremendous labours, including that of Herter and his co-workers, and the Metchnikoff school among many others, has led to no results. No doubt the fact that its formation depends on several factors, of which intestinal putrefaction is only one, is the principal reason of the divergence of opinion on the subject. For myself, I may say that in order to obtain a urine which gives the best indican test for medical students, it has been my custom to get that of a case of cancer of the liver rather than of a case of marked constipation.

(2) The indol derivatives are all relatively innocuous compared with other substances which can be obtained in the alimentary canal.

From a positive point of view, the group of substances which I think can be readily assigned a position of importance from the point of view of alimentary toxæmias are the amines produced by splitting off CO_2 from the proteolytic amino-acids by intestinal bacteria. The most important of these amines have been referred to by the opener of this discussion, and are para-hydroxyphenyl-ethylamine—the amine of tyrosine, iso-amylamine—the amine of leucine, and β-imidazolethylamine—the amine of histidine. Research workers have recently been attracted to these substances in consequence of the discoveries of Barger and Dale.

who first isolated them from ergot and worked out their pharmacological action. Their actions I need not dwell upon, as they have already been dealt with by Professor Dixon, but I may remind you that they are all very potent stimulants of plain muscle, and whereas parahydroxyl and iso-amyl raise the general blood-pressure, β-i. has a marked depressant action on the blood-pressure. That para-hydroxyphenyl-ethylamine and iso-amylamine may be responsible for chronic symptoms of intestinal toxæmias, such as arteriosclerosis, seems to have been proved by the work of Harvey, who produced marked arteriosclerosis and a renal condition resembling the large white kidney by giving small quantities of these substances by mouth to animals over prolonged periods. I must confess that these results are surprising, especially when one remembers the ease with which para-hydroxyphenyl-ethylamine is rendered innocuous by having its amine replaced by a hydroxyl group and then oxidized to para-hydroxyphenyl-acetic acid by an isolated liver perfused with Ringer's solution, as has been shown by Ewins and Laidlaw. From the point of view of acute intestinal disturbances, β-imidazolethylamine must be considered. This substance was further isolated from the mucous membrane of the alimentary canal by Barger and Dale in their endeavour to identify it with the hypothetical substance of Popielski called "vaso-dilatin." The mucous membrane of the intestine offered the best material for such research, for it has long been known that a watery extract of this intestine produces a very large fall of blood-pressure and other toxæmic symptoms when injected into animals. In a paper on cyclic vomiting in children,[1] I stated that Twort and myself had isolated a bacillus from the alimentary tract capable of splitting off a molecule of carbon dioxide from histidine and converting it into β-imidazolethylamine. Dale and Laidlaw had shown that injection of small quantities of β-i. caused, among other symptoms, marked diarrhœa and vomiting, and I suggested that the absorption of this substance into the blood-stream from the intestine might be responsible for the exacerbations of symptoms in cyclic vomiting. Since this time Berthelot,[2] a Frenchman working in Metchnikoff's laboratory, had also isolated a bacillus from the intestine capable of carrying out the same action. This bacillus he has called the *Bacillus aminophilus intestinalis*, because of its power of attacking amino acids. We make no such general claim for our bacillus, although there is evidence that it will attack some substances such as tyrosine and alanin. One other point where we differ from Bertrand and Berthelot most emphatically I wish to deal with. They say they can only isolate the bacillus from the intestinal contents of cases showing intestinal disorder and not from normal healthy animals. This is absolutely contrary to our experience, for we have never failed to obtain most potent histidine-destroying bacilli from any alimentary canal of normal animals that we have examined. One surmises, therefore, that Bertrand and Berthelot ascribe the symptoms of intestinal disturbances to this bacillus whenever it is present. Twort and I, on the other hand, think it is present in all alimentary canals, and that its products of activity only produce symptoms under special circumstances. The fact that Barger and Dale isolated β-imidazolethylamine from the intestinal mucous membrane of normal oxen supports the view that the bacillus is a normal inhabitant of the intestine. Its distribution is affected by the diet to some extent, for a cat that has lived on an exclusive meat diet has the bacillus higher up towards the duodenum than a cat on a milk diet.

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[1] *Lancet*, 1911, ii, p. 8.

[2] *Compt. rend. de l'Acad. des Sci.*, Par., 1911, cliii, p. 306.

Twort and I have recently been able to obtain β-i. from another source as the result of bacterial action, for we have obtained an intestinal bacillus capable of acting on carnosin with the production of β-i. Carnosin was first isolated from meat extract by Gulewitsch, and he showed it to have the constitution of histidyl—β-alanin—and to be of the nature of a polypeptid. It is clear that the production of β-i. from carnosin necessitates a preliminary hydrolysis before the removal of the carbon-dioxide molecule.

Since β-imidazolethylamine is present in the normal alimentary mucous membrane, the question arises as to why it lies in an apparently innocuous state without the production of symptoms, and also what would happen if there was an increased production of this substance in the intestine.

The answer to the latter question seems clear. If minute quantities of β-i., say 10 mg., be injected either intravenously or subcutaneously into a cat, then very marked symptoms of diarrhœa, vomiting, and general collapse appear immediately. If large quantities—i.e., up to 500 mg. of β-i. phosphate—be given so as to obtain access to the small intestine—i.e., in keratin capsules—then the only symptom which follows is that of diarrhœa with watery, acidic stools. But there is no vomiting and general collapse—in other words the β-i. has acted locally on the alimentary canal, producing greatly increased peristalsis, but it has not been absorbed into the blood-stream to produce the most important toxæmic symptoms.

Without suggesting that β-i. is responsible for all the symptoms of such an acute condition as summer diarrhœa and vomiting of children, although I think it plays an important part in such conditions and may be regarded as typical of a larger group, it seems to me that the facts just related as to β-i. throw an important light on the subject. For it is clear that if β-i. is normally present in the alimentary canal and if even when large quantities are present no general symptoms of poisoning are produced, then clearly it can only mean that when acute intestinal symptoms are produced they are due to the breaking down of the defensive mechanism of the intestinal mucous membrane which prevents the entry of β-i. into the blood-stream. Such a view as this would imply that there are all the potential factors, including intestinal flora and toxic chemical substances, present in the alimentary tract of all normal animals, and that the difference between the summer diarrhœa child and the normal child is due to the breakdown of the absorptive mechanism of the intestine and to that alone. Further evidence to this effect—viz., that the factor of importance to be considered is the absorptive mechanism and not an excessive production of toxins in the intestine—is the fact we showed that β-i. is never produced from histidine in the presence of carbohydrates and acids. Also the acid fermentation of carbohydrates in acute intestinal disturbances is notorious, and it is unlikely that there is any production of β-i. at such times. The acid fermentation products are themselves relatively innocuous.

This view would further point to a different line of inquiry for the elucidation of alimentary toxæmic conditions than that usually undertaken. For it suggests that the key to the situation is not held by a consideration of the bacteria of the intestine, nor of the chemical end-products of bacterial action, but rather by knowledge as to the normal method of intestinal absorption, and more particularly of the mechanism which prevents the passage of toxic substances across the mucous membrane into the blood-stream. The important question is, What is the cause in acute intestinal disturbances which brings about the breakdown of the defensive mechanism and allows the entry into the blood-stream of toxic substances? All research on the subject is handicapped by the fact that from a physiological point of view the process of intestinal absorption is almost a virgin field. The general trend of modern physiological research work on the alimentary canal seems to me to indicate another method whereby pathological symptoms may be traced to the intestine. In the first place we had Bayliss and Starling's striking discovery of secretin—a substance liberated from the duodenum by HCl of the gastric juice, and so causing pancreatic activity. The detection of gastrin by Edkins followed this—a substance in the pyloric end of stomach mucous membrane which was liberated into the blood-stream by various substances, including meat extract, and caused a further secretion of pepsin and HCl by the fundus mucous membrane. Again, quite recently, the results of Cow of work done in Hans Meyer's laboratory indicate that water taken by mouth acts as a diuretic, not by its direct action on the kidney, but indirectly by liberating some substance from the alimentary tract which stimulated the kidney to secrete water. He showed that a watery extract of alimentary mucous membrane injected subcutaneously caused marked diuresis in dogs, and if the extract was boiled it lost its diuretic producing effect. Such results as these, which prove the presence of chemical stimulants in the alimentary wall, make one wonder whether a generalization is possible, so that each organ in the body is stimulated to activity by some specific chemical substance present in the intestinal mucous membrane, each specific stimulating substance being liberated by a particular foodstuff. If such a premise is correct, it is clear that all sorts of symptoms might arise when an excess or a deficiency of the specific chemical stimulants is liberated into the blood-stream in consequence of abnormal intestinal putrefaction or other disturbance. Work based on such a hypothesis might be useful in elucidating the causal relationship between some of the more obscure pathological conditions—as, for instance, diabetes—and the alimentary canal.

Dr. ETTIE SAYER: My slight contribution to this discussion is evoked by Dr. Hale White's remark that "alimentary toxæmia has never been produced experimentally." Since the difficulties in the way of such an experiment are obvious it seems important, when all aspects of the subject are under consideration, not to omit reference to any therapeutic procedure which involves the risk of producing the condition accidentally. One of the procedures which involves this risk is the attempt to reduce a raised blood-pressure by means of high-frequency currents, in cases of chronic auto-intoxication, during periods of constipation. An excessive absorption of entero-toxins from the intestine may be set up which will not only send the pressure higher up instead of allowing it to come down, but may also produce an acute exacerbation of toxæmia.

A typical case illustrating this occurred in my practice just before Christmas. The patient was a spare little old lady who complained of headaches, giddiness, insomnia, muscular rheumatism, chronic fatigue, and constipation alternating with occasional attacks of diarrhœa. Her blood-pressure was 228 mm. Hg. Examination of the heart and of the blood-pressure taken in various postures showed this organ to be sound and devoid of any evidence of latent failure of ventricular compensation. After an aperient had been administered she was started on a course of high frequency to bring down the pressure. All went well for a few days, but then she had to leave town for a week. On her return her pressure was 196. She was asked whether she had had relief that day and replied, "Yes, twice." But the current (which she had previously found agreeable) had only run for four minutes when she said she felt giddy and thought she was going to faint. She became an ashen colour; her whole body cold and clammy; her pulse irregular and her pressure had risen to 217—namely, 20 mm. Hg. Palpation of the abdomen revealed scybalous masses, which of course furnished the cause of the accident. She acknowledged the relief to which she had referred to have been entirely inadequate.

In order to understand the production of this train of symptoms, it is as well to go back to the year 1896, when d'Arsonval published the results of a series of experiments showing that by careful dosage high-frequency currents can be made to increase the eliminations of the skin, the lungs and the kidneys. The heat production as measured by the anemo-calorimeter can be made to rise from about 80 to about 130 calories per hour. The output of CO_2 through the lungs from 17 to 37 litres per hour. With regard to elimination by the kidneys, numerous subsequent experiments corroborate d'Arsonval's original statement, that the output of nitrogenous waste, phosphates, and chlorides is increased, and that the urotoxic co-efficient is raised. These effects of high frequency are chiefly due to its *thermal* action. When a current flows through a wire the latter becomes heated in proportion to the amount of energy absorbed in overcoming resistance. When a current flows through the human body, it likewise becomes heated in proportion to its own resistance and the magnitude of the current flowing through it. The magnitude and therefore the heating power of high-frequency currents is very great. The heating power depends not only upon the potential of the current, but also upon the character and frequency of the particular oscillations which one arranges. The increased heating produces increased chemical activity of the various cells of the body.

and consequently stimulates metabolism and tends to overcome stasis anywhere.

So far as the alimentary tract is concerned Dr. Samuel Sloan has shown by means of electrodes variously arranged in the mouth, rectum, colotomy and other wounds, that it is possible to cause an appreciable rise of temperature throughout its entire length by means of an ordinary therapeutic dose of high frequency.

It was Dr. Doumer, of Lille, who first pointed out that the absorbent cells of the alimentary tract naturally share in the general increased chemical activity, and that if the bowel is loaded with retained matter which has undergone prolonged bacterial and possibly putrefactive changes there will be a correspondingly increased absorption of enterotoxins into the system, to the extent of causing a flooding which will quite overbalance the unloading otherwise effected by the current, and so lead to accidents such as that which I have just described. in the patient whom I have cited as an illustration.

TREATMENT OF ALIMENTARY TOXÆMIA.

I have never yet seen so severe a case of simple constipation and toxæmia that it necessitated the removal of the colon, but I have come across a considerable number which after resisting all drug and diet treatment have yielded to electricity. The forms most frequently indicated are either static or galvanic.

Static acts more powerfully on muscle than any other form of electricity; and the static wave currents, or in very severe cases the static induced currents, are indicated in atonic or debilitated cases where there is want of muscular power—cases characterized either by ptosis or by dilatation of any of the organs; and especially in cases where local symptoms of intestinal stasis preponderate over the symptoms of systemic toxæmia. Hitherto, I have always set the spark gap so that it was synchronous with the patient's pulse; and have thus got the thrust of the peristaltic wave downwards from the epigastrium to the lower electrode synchronous with the heart-beat. The results obtained have been all that could be desired; but on observing the intestinal movements on some bismuth meals recently it has seemed to me that there was also so much harmony between the alimentary and the respiratory movements that these ought also to be taken into consideration during treatment.

Galvanic currents act more particularly by ionization. They cause no muscular contraction except when the current is switched on or off, and their potential is too low for them to have any appreciable heating effects on the tissues. One employs them when one wishes to affect the mucous membrane and secretory cells and thus to alter or to reinforce the chemical defensive mechanism of the inner coat, rather than to stimulate the muscular coat of the intestine. High frequency causes an increase of normal chemical activity, galvanism produces chemical changes by ionic alterations in the molecules.

With regard to the treatment of cases where systemic toxæmia preponderates over the local symptoms of intestinal stasis and where the whole system including the muscular circulatory and eliminating organs are in a state such as has been so admirably described by Mr. Arbuthnot Lane, I have found a combination of high frequency, radiant light baths, and massage the best treatment.

Most of my own cases of advanced systemic auto-intoxication have been in overworked women who lead irregular, strenuous, worrying lives. Usually possessed of more than average ability themselves, they have confidence in their own sex, and hence form a considerable percentage of women-doctors' patients. Oral sepsis is frequently present. Their constipation is made decidedly worse by any extra fatigue, especially by any excessive expenditure of nervous energy in worry or late hours. They are usually addicted to taking large and irregular doses of aperients; the cæcum and ascending colon become dilated and overloaded; deposits of waste products can generally be felt in most of their muscles, especially in those which are most used for their work—e.g., the arm in writers. I cannot imagine the removal of their colons doing them any good unless it was by virtue of the enforced rest in bed for a few weeks. Quietude, rest, and repose of body in the fresh air, combined with mental diversion in the form of congenial society, seem indispensable for restoration to health. After attention to any point of sepsis, medically most help is obtained from gentle skilful massage, which seems most reliable from Swedes who have gone through a complete three years' training.

For bad local patches of fibrositis ionization with lithium iodide gives relief.

It is in cases of auto-intoxication *where the blood-pressure is raised that high frequency* has its most important value. There are two factors in this reduction:—

(1) A sedative action upon the nervous system, which directly decreases the reflex excitability of all nerves, including the vaso-constrictor ones, and so relieves the spasm of hypertonus.

(2) The thermal action, which helps to metabolize the products of defective combustion, which when circulating in the blood irritate the vasomotor nerve-endings in the vessel wall, and so produce vaso-constriction.

The best effects are generally obtained when the drop is about 7 or 8 for every dose, and goes up about 2 during the interval between the doses. Very great discretion is needed if there is any failure of ventricular compensation. Unless the pressure is unduly high it is now best left alone. An ordinary dose of high frequency given to these cases would cause quite an alarming drop, perhaps 20 to 30 mm. Hg. or even possibly a syncopal attack.

Cases of simple extra-systole, even when it occurs as often as every fifth or sixth beat, derive benefit from high frequency, the irregularity often completely disappearing provided there is no cyanosis nor cardiac dilatation, and that the elastic recoil of the arteries is not seriously impaired.

As I stated to begin with, a strict rule should be made never to administer high frequency on any day in which the bowels have not been efficiently moved. Should this by any inadvertence occur the treatment is to give an immediate colon irrigation, then copious draughts of plain water, and a radiant light bath to aid elimination by the skin; after which the patient will probably drop off into a comfortable sleep.

In conclusion, I would like to heartily concur with those who consider that a healthy colon is very much better than no colon. The fashion for removing it is so new, that it still remains to be seen whether or not these patients do not in later years develop insidious and profound malnutrition, from deprivation of so lengthy and highly specialized a portion of mechanism that Nature destined for use. Before being subjected to its removal, patients ought to have every guarantee that all the resources of conservative medicine have been exhausted on their behalf.

Dr. J. SIM WALLACE: A careful perusal of the opening papers indicates that alimentary toxæmia cannot be said to be due simply to the presence of micro-organisms, or even particular kinds of micro-organisms in the alimentary tract. Rather must we conclude that alimentary toxæmia is due to certain abnormal conditions giving rise to undue lodgment and proliferation of micro-organisms, together with defective elimination or control of the toxins generated. We may further say, in general, that these conditions are induced or predisposed to by lack of function in or near the situation where the toxins are produced. Dr. Hale White has referred to a function of the liver as being one which protects us from the toxins produced during the process of the digestion of foods. But the great significance of the functions of the liver in its relation to alimentary toxæmia seems in general to have been but lightly touched upon, and the important part played by the muscular activity of the alimentary canal seems to a certain extent to have been neglected. My intention, however, is more especially to direct attention to the physiological processes which keep the mouth in a hygienic state, and to indicate how the ever present bacteria of the mouth are prevented from stagnating and producing toxæmic effects; for upon recognition of the natural or physiological methods of control of the bacteria and the toxins produced can we best base a scientific and practical treatment. Although as medical students we were all taught that the chief function of the saliva was to digest starch, this is not so. The chief function of the saliva is to keep the mouth in a hygienic state, to remove food which by stagnating would undergo fermentation or putrefaction, and to control the products of fermentation sufficiently thoroughly to prevent injury to the teeth or mucous membrane of the mouth. We were taught also that the function of the mucus secreted was to lubricate the food bolus and facilitate its transit to the stomach. Firstly, however, we should note that boluses should not be transmitted to the stomach; and secondly, that mucus has an even more important function in the hygiene of the mouth. The mucous coating over the teeth prevents acid foods from doing them harm, and if a meal is finished with due regard to the hygiene of the mouth—e.g., with fresh fruit—the mucous plaques are, according to Professor W. G. Gies, disorganized. Thus then their removal together with adherent bacteria and food particles is facilitated.

We have said in general that lack of function predisposes to alimentary toxæmia; this is well illustrated in the mouth, where more than in any other part of the alimentary canal what happens can be easily seen. The primary function of the teeth is the mastication of food, but the mastication of food of a detergent nature has a second function—namely, the prevention of the undue stagnation of bacteria and food debris about the teeth. The earliest effect of such stagnation is a slight inflammation of the gum margins surrounding the necks of the teeth; that is to say, where from lack of function the greatest amount of stagnating bacteria and food debris is located. When the stagnating bacteria and debris at the necks of the teeth give rise to such toxæmic symptoms as we have just referred to for a long time, the inflammation may become more than superficial and a rarefying osteitis may be set up. More especially is this liable to occur when through lack of function the ligamentous tissue and the alveolar processes are weak. It should be noted here that superseptic states, together with recession of the gum and extensive absorption of the alveolar process, may take place without the occurrence of pyorrhœa alveolaris. The rational treatment then of toxæmia arising from oral mal-hygiene is the restoration of the normal function of mastication and the stimulation of the self-cleansing processes of the mouth. Inasmuch, however, as we are most frequently called upon to treat cases of alimentary toxæmia when the state of the mouth is such that artificial aid is necessary to put the mouth into a hygienic state, and as, moreover, to a certain extent this is easily done, generally speaking the first step in treatment should be the artificial cleaning of the teeth—I do not mean with a tooth-brush merely, but with surgical instruments specially adapted for their thorough cleansing. When this is done, or when it is being done, it may be found that some tooth or teeth may require restoration to make it functional, or it may be found that some tooth or teeth on account of the state of the alveolar process in its neighbourhood cannot be restored to full functional activity. In this latter case immediate extraction is desirable; there is no use in keeping a tooth in the head which cannot be made functionally active, for a tooth which is not functional is always a superseptic tooth within a day of its having been cleaned, even by skilled hands. Moreover, to a certain extent such a tooth being tender, efficient mastication on it and the neighbouring teeth is hindered, and there is no justification in prolonging the diseased states originating from such teeth. I would accordingly here endorse the views advocated by Mr. Colyer, for even although it were not possible to restore the function of mastication on artificial substitutes, the harm resulting from lack of mastication is not so great as the harm resulting from functionless superseptic teeth.

The next point in the treatment is the restoration of the patient to a dietetic regime which will bring the teeth into functional activity and stimulate thoroughly the self-cleansing of the mouth. I have already referred to the necessity of food requiring efficient mastication, but in addition to this it is desirable to insist, as far as may be possible, on a reasonable supply of food containing a certain amount of acid, for this promotes the cleansing function of the saliva and mucus most effectually. Acids call forth a copious flow of saliva rich in ptyalin. There need be no fear of the acid in fresh fruit or vegetables injuring the enamel of the teeth; this is amply provided for by the mucous coating on the teeth and a copious flow of alkaline saliva. Furthermore, the after-flow of saliva, under such a regime, clears the mouth not only of stagnating food particles and bacteria, but also washes away the disorganized mucus. Remarkably excellent results are obtained by the ordinary surgical procedures and by washing out the dirty ulcerated pockets round the teeth, if it is deemed permissible to let them persist, with normal saline solution and not with irritating antiseptics; provided always that these artificial procedures are more or less consistently supplemented by a dietetic regime which stimulates the functional activity and self-cleansing processes of the mouth.

When we turn to a consideration of alimentary toxæmia beyond the mouth we note a general similarity in causation—i.e., insufficient motor activity of the stomach and intestines, and insufficient or perverted secretion of the bile. In a discussion on alimentary toxæmia it is surely of the greatest importance to take into consideration the fact that "although itself without bactericidal property, the bile nevertheless checks intestinal putrefaction—(*a*) by exerting a selective influence in favour of certain organisms, such as the colon bacillus, to the disadvantage of anaerobic promoters of putrefaction; (*b*) by inhibiting the secretion of the bacillary ferments; and (*c*) by stultifying the action of these bacterial ferments on putrescible material." Moreover, the bile "countervails the effects of the toxic products to which the intestinal bacteria give rise."[1]

I am convinced that if a tenth part of the attention had been paid to the value of food from the point of view of alimentary hygiene that has been taken with regard to its nutritional value, the treatment of alimentary toxæmia would have been far more rational and satisfactory than it at present is, and, moreover, the trouble would have been relatively rare.

Dr. W. Bezly Thorne: As evidence that the few clinical observations which I have to offer on this subject are not the result of recent and hasty generalization, I may perhaps be allowed to mention that, some seventeen years ago, I read before a Midland medical society a paper on "Self-Poisoning and Heart Disease," which was published under that title in the *Lancet*.[1] In it I called attention to the fact that a great proportion of the affections of the circulatory mechanism are associated with a catarrhal condition of more or less of the alimentary canal and especially of the gastro-duodenal portion; and that coincident with that condition it is common to find evidences of rheumatoid arthritis. With the lapse of each succeeding year that view has been so far confirmed that I now believe there are few phases of cardio-vascular trouble with which disorder of some part of the alimentary tract is not causatively associated. In so saying I fully recognize the gravity of sepsis originating in the mouth, the nares and the accessory cavities, and especially of that derived from hæmorrhoids and fissure of the sphinctorial region of the anus. But I desire to emphasize that, potent as such sepsis is in morbific influence on the circulatory organs, the great majority of such affections are associated with an alimentary toxæmia which presents certain easily recognizable features.

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[1] H. Roger, *Universal Med. Record*, Lond., 1913, iii, p. 300.

For the sake of brevity I will, as far as possible, condense the result of my observations, which have been purely clinical, into aphorismal form. Normal fæcal matter—namely, that which is extruded as duly moulded in the pelvic colon and the rectum, and of full brown colour, and free from putrid odour—is not toxic, and may be retained for two or even more days with, at the most, unimportant discomfort. Fluid stools, if of frequent or habitual occurrence, are always associated with toxæmia. They are the product of an interference with the bio-chemistry of digestion. Pale-coloured stools, that is, drab or yellowish, if of frequent or habitual occurrence are always toxic, whether formed or fluid, but especially so if fluid. They only occur when the chyme has been deprived of a normal amount of the pancreatic and biliary juices as the result, generally, of an interference with the patency of the common bile-duct which, in the great majority of cases, is due to gastro-duodenal catarrh. Without a sufficient supply of the biliary and pancreatic juices the foundation of normal intestinal digestion cannot be laid, and the door is at once opened to morbific bacterial activity. The fluid stool, especially if of watery consistency, is more active in the promotion of toxæmia than the formed, because it is in greater or less degree absorbed from the pelvic colon and the rectum, the mucosa of which is not endowed with the faculty of discrimination between fluid fæcal matter and, say, liquid nourishment or a normal saline injection. The individual who frequently or daily passes a watery stool, whether as the result of a morbid intestinal condition or of the ingestion of saline aperients, is undergoing rectal feeding on his excremental poisons. All such autotoxis is an immediate cause of a rise in blood-pressure ; and few therapeutic phenomena are more striking than the fall in blood-pressure, and the improvement in general condition, which ensue on restoring to the evacuations a normal consistency. The result is most remarkable if at the same time the patency of the common bile-duct is restored by relief of duodenal catarrh. Subjects who frequently or habitually have loose, pale stools are especially liable to indicanuria. That condition, as recently pointed out by Dr. Gilchrist, of Nice, in connexion with this discussion, is especially noticeable in hyperthyroidism. The toxæmia in question conduces in a high degree to generative change in the myocardium and the arterial tunics, especially in those of the tunica media.

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[1] *Lancet*, 1896, i, p. 755.

It may be mentioned, in this connexion, that, shortly before he retired from practice at Plombières, Dr. Bottentuit informed me that practically all subjects of long-standing colitis presented myocardial weakness, generally with dilatation. This form of toxæmia is specially favourable to the production of fibrositis the incidence of which, where there is any degree of cardiac dilatation, is on the third and fourth left costo-cartilaginous junctions, the site of the apex beat, and the confluence of the cartilages to the left of the ensiform cartilage, and on that structure itself. That affection of the thoracic structures is the basis, in the greater number of the cases of pseudo-angina that come under my observation ; and it is interesting to note that pressure on the third and fourth costo-cartilaginous junctions, when they are thus affected will, not uncommonly, transmit pain down the left arm. When the fibrositis is limited to the epigastric region it is frequently mistaken for indigestion. When it extends, as it frequently does, to the tips of one or both floating ribs the pain or aching is sometimes attributed to renal calculus ; and in one case within my knowledge in which the fibrositis remained for a long time incident mainly on the structures adjacent to the ensiform cartilage on the right side, a futile operation for impacted gall-stones was performed.

I submit, therefore, that the treatment of gastro-duodenal, duodenal, and intestinal catarrh is frequently an essential element of the successful treatment, and permanent relief, of many cardio-vascular affections ; and that where the duodenum is involved in the morbid process, dietary precautions, especially as regards the limitation of carbohydrates and reliance on proteid foods, mainly those of the lighter kind, are indicated both on physiological grounds and by clinical experience. In confirmation of the toxic effect of carbohydrate indigestion it may be mentioned that overwhelming somnolence coming on at or immediately after meals and persisting for some two to four hours after may be completely relieved by abstinence from starchy food until such time as a normal flow of the pancreatic and biliary fluids is re-established.

The following are three illustrative cases the number of which could be almost indefinitely multiplied:—

Case I.—March 15, 1913: Man, aged 50. Thirty-five years' service in India. During the last ten years had intestinal catarrh with heartburn, flatus, drowsiness, and "slackness." Bowels act once daily. Stools of the consistency of porridge, dark brown, offensive. Rheumatoid arthritis attacking both hands.

	Pulse	Balance or diastolic pressure	Radial tactility ceases	Brachial obliteration
March 15, 1913	84	100	150	170 mm. Hg.
April 16, ,,	66	80	110	125 ,,
Fall	18	20	40	45 ,,

April 16, 1913: Bowels open once daily, motions formed and free from fœtor. No drowsiness nor "slackness." Treatment: Paraffin and bismuth emulsion night and morning; 5 gr. tiodine (iodide of fibrolysin) pill three times a day with food.

Case II.—June 22, 1912: Woman, aged 52. Palpitation and dyspnœa on exertion and on lying down, can sleep only when propped up; thoracic fibrositis simulating angina pectoris. One or two liquid stools of yellow colour every morning after Apenta water.

	Pulse	Balance or diastolic pressure	Radial tactility ceases	Brachial obliteration
June 22, 1912	80	170	240	250 mm. Hg.
,, 26, ,,	78	140	190	200 ,,
Fall	2	30	50	50 ,,

June 26, 1912: Bowels open once daily, motion formed and brown. Can sleep lying on either side. Other symptoms relieved. Treatment: Paraffin and bismuth emulsion night and morning; occasional mild dinner pill in the place of saline aperient.

Case III.—August 16, 1912: Man, aged 57. Aortic regurgitation; one or two fragmentary or loose yellow motions daily; dyspnœa on exertion, general weakness: 6 to 7 dropped beats per minute.

	Pulse	Balance or diastolic pressure	Radial tactility ceases	Brachial obliteration
August 16, 1912	82	90	150	220 mm. Hg.
,, 23, ,,	72	80	160	170 ,,
Fall	10	10	Rise 10	Fall 50 ,,
April 14, 1913	68	80	155	175 ,,
Fall	14	10	Rise 5	Fall 45

Adrenalin and strychnine, June 28, 1912.

Treatment: Bismuth mixture night and morning. Since August last the patient has been able to lead an ordinary though somewhat restrained life, and, during the season, did a moderate day's shooting once or twice a week. The pulse became regular on the addition of strychnine and adrenalin, orally administered, twice daily.

Dr. H. G. ADAMSON said that although alimentary toxæmia in relation to skin diseases was a subject which had attracted considerable attention, it was one of which we knew really very little. Among the laity, and to some extent also among medical men, there was a tendency to attribute many skin eruptions to gastro-intestinal disturbances, and hence to attach very much importance to dietary and regulation of the bowels in treatment. On the other hand, those who made a special study of skin diseases, though they also might incline to that idea, relied largely upon local remedies for these eruptions.

There were, of course, certain well-known examples in which an eruption seemed to depend on some food poison or drug poison. It was a common experience to meet with erythemas, urticarias and purpuras after the ingestion of some particular food—such as shellfish, tinned meat, or, in the case of certain persons, eggs, cheese, or strawberries—or after the administration of such drugs as copaiba, quinine, antipyrin or belladonna; also after the administration of horse serum by the mouth. Opinions differed as to the exact way in which these eruptions were produced, but there was a recent tendency to look upon them as phenomena of anaphylaxis, or a hypersensitiveness to foreign proteids. This would explain why a person who had once had an eruption consequent on eating a certain kind of food might become so sensitive to that poison that the eruption might eventually be caused by quite minute quantities of that particular food. Frequently one saw patients who, after an attack of urticaria following some food poisoning, for months or years afterwards were subject to urticaria. And sometimes it was possible to discover that the rash followed the taking of a particular kind of food in minute quantity. The same remark would apply equally well to drug eruptions. It was long ago suggested by Behrend that drug eruptions did not result from the physiological action of the drug, but were due to the production of some poison by its action on the blood. That idea had latterly been revived, and certain experiments by Bruck and others went to show that drug eruptions were produced by toxic albumins resulting from the action of drugs upon the living cells. A drug eruption was a manifestation of anaphylaxis to a foreign albumin.

But this class of skin disease was not due to alimentary toxæmia in the sense in which it had been used in this discussion. These eruptions were due to foreign bodies introduced into the alimentary canal. The question we were now concerned with was whether skin eruptions might result from toxins due to bacterial activity in the gastro-intestinal canal, or from the chemical products of defective digestion. Several speakers had stated what were the common bacteria of the alimentary canal, and the chemical poisons produced there; but it was certain that no skin eruption was known which could be definitely traced to any of those bacteria, or which were known to be due to any of the chemical bodies elaborated in the intestines. Certain eruptions, such as eczema, acne vulgaris, furunculosis, lichen urticatus, erythema multiforme, were thought by many to depend largely upon digestive derangement, but ideas respecting this relationship were vague, and they were not founded upon scientific data. No one had any definite views as to what particular toxins caused these eruptions, and in practice the already known factors stood out more prominently. In eczema local irritation was a known factor in its production; the existence of alimentary toxæmia was uncertain. The same might be said of acne and furunculosis; the presence of organisms in the skin could be demonstrated, but one did not know what toxin, if any, was a contributory factor. In erythema multiforme there were abdominal symptoms associated with the skin eruption; but it was probable that the abdominal symptoms were produced by the same kind of lesions occurring in the mucous membrane of the intestinal canal; that they were co-existent, not causative. Before one could say that any skin eruption was due to gastro-intestinal toxæmia, one ought to be able to fix upon the toxin which produced the eruption, just as we must demonstrate the microbe before we could definitely state that an eruption was of bacterial origin.

Nevertheless, the possible relationship of certain skin eruptions to alimentary toxæmia remained an attractive theory. There were many skin eruptions — psoriasis, lichen planus, pemphigus, scleroderma, pityriasis rosea, alopecia areata, lupus erythematosus, and others—which were certainly definite entities, and almost obviously due to some specific cause, of the nature of which, however, we were as yet ignorant. There were circumstances which rendered it very improbable that these eruptions were due to the local presence of micro-organisms, and the only alternative seemed to be to regard them as due to toxins. The most likely source of such toxins seemed to be the alimentary canal. That was why so many attempts had been made to prove that these eruptions were due to alimentary toxæmia. For most of the diseases, examination of the urine had been made to ascertain the amount of ethereal sulphates, and other aromatic bodies as indications of absorption of toxic bodies from the alimentary canal, but without any uniform and convincing results. Excessive eosinophile cells had been found in pemphigus and other conditions, and that had been thought to indicate toxæmia, but for no very adequate reason. Perhaps the most suggestive point was that some of these affections might be imitated by those due to drugs taken by the alimentary canal, such as pemphigus by bullous iodides or alopecia areata by alopecia due to acetate of thallium.

One thing which had been learned from the experimental work in regard to the erythemas and urticarias due to food poisons and drug poisons was the infinitesimal amount of poison which might suffice to cause an eruption; and it seemed probable that if these other eruptions were due to toxins, they were toxins which were too small in quantity to be measurable by ordinary chemical methods. At present we had no clue to their discovery.

Dr. R. MURRAY LESLIE: There seems to be a great divergence in the views expressed during this discussion in regard to the nature, causation and effects of alimentary toxæmia. Dr. Hale White adopts a cautious, agnostic attitude in respect to some of the phenomena, particularly in regard to the relationship of alimentary toxæmia to intestinal stasis. He believes, however, that an important function of the liver is to protect us against poisons absorbed from the intestines, which is practically an admission that poisons are so absorbed, and admits further that in hepatic disorder (e.g., cirrhosis) the liver may not be able to deal adequately with these poisons. It is surely only one step further to maintain, as Mr. Arbuthnot Lane and his followers do, that in certain abnormal conditions of the alimentary canal, such as intestinal stasis, more poisons may be absorbed than can be adequately dealt with even by a healthy liver. I am strongly of opinion that in some instances the symptoms of alimentary toxæmia are certainly aggravated by defective functional action of the liver. This fact has been exemplified in one or two of my own cases, the characteristic toxæmic symptoms being found associated not only with abnormal intestinal flora and the presence of indican and skatol in the urine, but also with slight icterus and the presence of urobilin in the urine indicating some hepatic insufficiency.

I believe that under normal conditions in healthy persons putrefactive changes in the intestines produce no evil results whatever, and probably fulfil a most useful rôle in the animal economy.

We are all, I imagine, believers in the power of the *vis medicatrix naturæ*, and being so are necessarily of opinion that in the great majority of instances the protective mechanism of the healthy organism is more than sufficient to counteract the deleterious effect of any toxins that may be produced by the various bacterial flora that normally inhabit the gastro-intestinal canal. For our present purpose it does not matter what these protective agencies may be—whether specific destructive action by the liver, the action of the internal secretions of the thyroid and other ductless glands, or the production of antibodies in the blood. In the course of evolution the human host has adapted itself to the presence of those bacteria which have selected the intestinal canal as their normal habitat. Nature, indeed, as in her other vital manifestations, no doubt provides a fair margin of reserve, sufficient to deal with even occasional excessive doses of poison. It is equally true, however, that there is a limit to this reserve, and should it happen on the one hand that the vital protective forces are themselves at a low ebb from any cause, or on the other hand that the intestinal flora as the result of stasis are abnormally numerous or consist of abnormal types, or invade portions of the intestinal tract remote from their usual habitat, and should either of these two factors persist for any length of time, then ill-health is bound to ensue.

It seems to me that sufficient stress has not been laid by previous speakers on the *first factor—viz., the power of resistance on the part of the individual*. It is true that reference has been made more than once to the fact that there is no necessary relationship between the amount of stasis and the degree of toxæmia. It is a matter of daily clinical observation that some persons can remain constipated for days and even weeks without apparently suffering any ill-effects, while in other instances the missing of the usual after-breakfast evacuation is invariably followed by headache, malaise, and inability for physical or mental exertion. Habit tolerance, important though it may be, does not altogether account for such phenomena. There seem undoubtedly to be individual idiosyncrasies in respect of liability or resistance to alimentary toxæmia. The importance of the personal factor is exemplified by the well-known liability to bacterial invasion (including *Bacillus coli* infection) of persons debilitated by injury, disease, over-fatigue, senile tissue-degeneration, and other conditions leading to diminished vital resistance.

Thus, for instance, it is known that shortly before death when the vital protective forces are more or less in abeyance, the *Bacillus coli*, streptococci and other bacteria can freely pass through the intestinal mucous membrane and so into the circulation, while their toxins, not being neutralized or destroyed, accumulate in the circulating blood, and are no doubt responsible for many of the symptoms preceding dissolution, including ante-mortem pyrexia. Metchnikoff, indeed, believes that such absorption is the primary cause of death in most cases of senility, while any check to such absorption tends to greatly prolong life.

Evidence is accumulating that the specific action of the thyroid and other ductless glands plays an important part in regard to vital resistance, and I was particularly interested in Mr. Rowell's reference to cases of intestinal stasis associated with thyroid enlargement, and in which the gland diminished in size on removal of the stasis, in one case as the result of administration of liquid paraffin, and in the other following operative interference. Mr. Carson has also referred to the effect of relief of stasis on parenchymatous goitre. I believe that the internal secretions of the genital glands also play an important part in counteracting the effects of alimentary toxæmia. It is a curious fact that in my series of well-marked cases of alimentary toxæmia associated with visceroptosis and intestinal stasis the majority of cases have occurred, not in married women who have had repeated pregnancies, and who accordingly might be presumed to have lax abdominal walls and relaxed mesenteric attachments, but in unmarried women between the ages of 30 and 40. In not a few of these cases considerable benefit has accrued from the giving of various preparations of glandular extracts. This has also been the experience of other recent observers. Future investigations in regard to internal glandular secretions will no doubt throw additional light on the "vital resistance" factor in alimentary toxæmia.

It is, however, on the *second factor—viz., the increased quantity or virulence of the alimentary toxins*—that the discussion has hitherto turned, and it is of paramount importance to decide which are the conditions in the alimentary tract that predispose to increased production of such toxins. The bacteriological evidence brought forward by Dr. Andrewes points unmistakably to intestinal stasis as pre-eminently the condition favouring such production. He has shown us that stasis is a fertile adjuvant to bacterial multiplication, and also states that any part of the alimentary tract in which there is retention of the gastro-intestinal contents tends to show an abnormal intestinal flora. He has further indicated that such retention of intestinal contents affords abundant opportunities for the full exercise of the fermentative activities of the bacteria and for the absorption of their toxic products, in addition to giving facilities for the upward spread of the coliform bacteria into the ileum and to other portions of the small intestine the mucous membrane of which is more absorptive than that of the colon. The most characteristic of my own cases of alimentary toxæmia have all been associated with visceroptosis[1] and intestinal stasis. In several of these there has been stasis also in the stomach, associated with chemical alterations in the acidity and other characters of the gastric contents—a condition which naturally facilitates the passage of buccal bacteria into the alimentary canal, whether pyorrhœa alveolaris be present or not. In these cases there must be a proportionate increase in the quantity and virulence of the toxins produced.

The question then seems a comparatively simple one. Whenever the toxicity is in excess of the resistive power of the organism the balance of forces is upset and toxæmic symptoms manifest themselves, either in an acute or in a chronic form. Professor Dixon's investigations in regard to the action of the histidine group of decomposition products in contracting the bronchioles, throw an interesting light on the not infrequent association between flatulent dyspepsia and acute spasmodic asthma, while the acute pathological and clinical manifestations resulting from the subcutaneous administration of sepsine—a substance closely allied to cadaverine—go far to explain the phenomena of acute ptomaine poisoning. His microscopic preparations have also revealed to us the permanent effects in the form of arterial degeneration and pathological changes in the kidneys, produced by the long-continued action of certain of the more toxic derivations of protein cleavage. It is evident from the observations of several of the speakers that the actual quantities of indican and skatol—comparatively inert substances—that may be found in the urine, must not be accepted as in any sense a criterion of the degree of alimentary toxæmia present in any individual case. The degree of toxæmia must be judged for all practical purposes by the intensity of the clinical manifestations, while bacteriological and chemical investigations are principally of service in indicating the nature and source of the toxæmia. I have at present under my care an interesting case of Henoch's purpura in a boy, aged 16, in which the purpuric eruption, the abdominal pain and vomiting, the epistaxis and the albuminuria were all preceded by marked constipation, the patient stating that there had been no action of the bowels for seven days. This case is interesting in view of the theory held by some of the toxæmic origin of this affection. Dr. William Hunter has already referred to the main points in regard to his classical investigations on the relationship of pernicious anæmia to oral sepsis and intestinal auto-intoxication.

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[1] "Visceroptosis," *Clin. Journ.*, Lond., 1912, xl, pp. 49-63

There has been much discussion in regard to the peritoneal adhesions and kinks described by Mr. Lane and others. Professor Keith stated that in the human embryo peritoneal adhesions begin to be formed as early as the third month of fœtal life. I was particularly interested in the unmistakable evidence he produced at his recent Epidiascope Demonstration that in man and the anthropoid apes a definite adhesive structure is formed between the cæcum and the right kidney at the end of the third or fourth month of fœtal life, and that it is only at a considerably later period of development that the cæcum descends into the right iliac region. Professor Keith stated that similar attachments are gradually formed during fœtal life, fixing the large bowel and abdominal viscera to the posterior wall of the abdomen, and that in a large percentage of children at birth well-marked adhesions can be demonstrated attaching the posterior wall of the cæcum to the iliac fossa. As many of these fœtal adhesive structures described by Keith and others are peculiar to the anthropoid apes, they are presumably connected with the assumption of the erect attitude, and are doubtless developed to prepare the animal for the upright posture. Microscopic sections of these fœtal adhesive structures reveal active cell proliferation, indistinguishable from that of inflammatory tissue—suggesting that we are dealing with a physiological peritonitis, if one may use such a term. Professor Keith, however, did not profess to claim that all the adhesive structures found in adults are embryonal in origin, but frankly admitted the possibility of acquired adhesions being formed in post-natal and adult life. In common with many others, I was at first extremely sceptical as to the presence and significance of the various adhesions and kinks described by Mr. Lane, but the frequent witnessing of X-ray and operation demonstrations in my own patients has convinced me of their existence and importance, and I feel that I can with confidence predict that a similar view will sooner or later be universally accepted by the medical profession. Whether these adhesions are evolutionary, or inflammatory in the sense referred to by Professor Keith, does not really matter. They are developed so as to gradually counteract the increasing weight of the abdominal viscera, particularly the cæcum and colon, which tend to get overloaded with solid and semisolid contents. Such adhesions are almost invariably found along the lines of resistance to vertical downward displacement, and are therefore intimately associated with the erect posture. They are protective in function and, like all other evolutionary structures, generally succeed in effecting their purpose.

It would serve no useful purpose to detail the various symptoms present in my series of cases of alimentary toxæmia. The symptomatology of the condition has been fully discussed by Dr. Saundby and other speakers.

As regards prognosis, this will depend entirely upon the therapeutic measures adopted. If a woman with well-marked chronic stasis and definite continued symptoms of alimentary toxæmia is left untreated, she will almost certainly relapse into a condition of physical and mental asthenia and chronic invalidism, and is further peculiarly liable to intercurrent acute disease. Treatment of some kind must be initiated to adjust and redress the balance of opposing forces. This can only be done in one of two ways: (1) By stimulating the vital forces themselves by hygienic, dietetic and other methods, by vaccine and enzyme therapy and by glandular extracts containing the products of internal secretion; or (2) by diminishing the production of bacterial toxins and facilitating their elimination. In the large majority of instances, if the cases are treated at a comparatively early stage, simple remedial measures such as diet modifications, physical exercises, abdominal massage, supporting belts, aperient remedies and lubricants (such as liquid petroleum), are usually quite efficacious. Most of these measures favour elimination by relieving intestinal stasis, and although association of stasis with toxæmia does not, as Dr. Hale White states, prove causation, still it is surely more than a coincidence when it is found that the toxæmic symptoms often entirely disappear when the remedial measures adopted have removed the stasis. Petroleum ought to be given in large doses (½ to 1 oz.) two or three times daily, and in some instances may be accompanied by 15-minim doses of liquid cascara. I believe strongly in the importance of diminishing the intake of animal proteids, and there is a great deal of truth in Professor Keith's contention that the colon is more of a misused than a useless structure, owing to the extraordinary change that has taken place in the diet of man in civilized countries. I agree with Metchnikoff in thinking that the simpler food of uncivilized races may be best, and that it would be true progress to abandon much of the modern cuisine and to go back to the simple dishes of our forefathers.

I have found considerable benefit from resorting to a modified vegetable and purin-free diet for a certain period, reducing animal food to a minimum, at all events for a time, until the stasis has been relieved. It is the excessive cleavage of animal proteins which is so harmful. In my experience intestinal stasis is much commoner in private than in hospital practice, and I attribute this fact to injudicious diet on the part of the better classes. The substitution of a two-meal diet for the three-meal diet of to-day would be a great advantage. There is much in the contention that the beginnings of stasis are frequently found in children, as the result of gastro-intestinal disturbances, and it may be that the substitution of artificial for breast-feeding is to some extent accountable for the apparent increase of alimentary toxæmia in modern times. Dr. Eric Pritchard has shown that the meconium of the maternal milk has a special protective action on the intestinal mucous membrane similar to that of petroleum.

While speaking of the effects of civilization I might add that although we are erect beings, we are not intended by Nature to keep in the erect attitude (as we usually do, either sitting upright or standing) for eighteen out of the twenty-four hours. Our ancestors probably spent a good part of the day lying down, and great relief is experienced by patients with stasis adopting the recumbent attitude even for an hour or two every day and with the foot of the bed raised at night. I am one of those who attribute much of the visceroptosis associated with intestinal stasis to the almost universal adoption in civilized life of the high sitting posture instead of the crouching attitude of our forefathers in the daily act of evacuation, thus forcing the colon into the pelvis and causing the characteristic sagging and elongation of the rectum along the pelvic floor.

There is a minority of aggravated cases with progressive emaciation and debility and other symptoms threatening the life of the patient, where simple measures, however carefully carried out, will fail, and my experience of a considerable number of such cases has led me to strongly advocate having recourse in these circumstances to surgical interference, with the object of removing the poison manufactory, which is usually in the colon and terminal portion of the small intestine. By far the most satisfactory surgical measure is that of short-circuiting the ileum into the pelvic colon, thus throwing the greater portion of the large intestine out of action, and thereby enormously accelerating the passage of the intestinal contents, including the bacteria and their toxins, so that there is now much less opportunity for fermentative and putrefactive changes to occur. The position is somewhat analogous to the opening of an abscess in pyæmia. In many instances the vital forces will gain the victory even if the abscess is left alone, but, on the other hand, the opening of the abscess and consequent removal of the poison manufactory will immediately turn the balance in favour of the patient. Similarly in cases of intestinal stasis where simple measures are found ineffective to check the toxæmia, which is endangering the patient's life, it is not only unreasonable, but also inimical to the patient's best interests, to refuse to resort to operative interference.

In my experience it has been in those cases of visceroptosis where the cæcum (and to a less extent the sigmoid) is bound down below the level of the brim of the true pelvis and cannot be raised up by manual manipulation that simple measures—medical and otherwise—often fail to give permanent relief. Visceroptosis, however extreme, accompanied by free mobility of the prolapsed portions, can be much more readily dealt with by such measures as abdominal support and the administration of petroleum. It is also in the former type of case that kinks are so apt to occur, many of which, even when temporary, may be accompanied by acute attacks of intestinal colic. I have one or two such cases now under observation. If it be true that there is a controlling mechanism at the ileo-cæcal opening, as suggested by Dr. Elliott and others, this would be specially apt to be interfered with when the cæcum is bound down in the true pelvis.

I should like to state definitely, as a matter of personal experience, that in most of my cases in which simple measures have entirely failed to give relief, and recourse has been had to a short-circuiting operation accompanied in one case by colon resection, the resulting benefits have been little short of marvellous. One patient in whom a complete colon resection and short-circuiting had been performed by Mr. Lane seven years ago and who was a complete chronic invalid for many years previously, is now absolutely well. Another patient short-circuited last June came last week to report herself as quite well; she had been a complete invalid for five years, with constantly recurring attacks of mucous colitis accompanied by constant pain in the left iliac fossa, for which Plombières treatment and other measures had been tried without avail. She came, she said, to thank me for having advised operation; she said that "life was now worth living," that she "looks and feels quite different," and her friends had remarked that she had completely lost "the sallow, dragged, weary look" of former years. She has now a natural-formed evacuation daily and can walk six miles without fatigue.

I am glad to be able to offer this personal testimony to the success of operative procedure in suitable cases.

Mr. J. Lewin Payne: The relationship between oral sepsis and intestinal auto-intoxication has been already sufficiently demonstrated in the course of this discussion, but there seems to be a considerable difference of opinion as to what this relationship actually is. Some, like Mr. Arbuthnot Lane, consider that oral sepsis is a result and not a cause of chronic intestinal stasis. They hold that it arises from the lowering of the resisting power of the tissues induced by the condition of the intestine. It is maintained that organisms which exist normally are thus able to manifest their presence by starting an infective process in the mouth. Others regard oral sepsis as the primary focus of trouble. They consider the simplest alimentary toxæmia is that which is due to pyorrhœa, since marked improvement in general resisting power may result from the effective treatment of pyorrhœa. Others, again, consider that both intestinal auto-intoxication and oral sepsis arise from a common cause. The chronic nature of the complaints associated with alimentary toxæmia is largely due to the fact that a vicious circle is established. Hence when once oral sepsis has led to gastro-intestinal trouble, the oral sepsis itself may be kept active by the secondary complaint.

It has been made clear already that the chief factors in oral sepsis are dental caries, pyorrhœa alveolaris, and deposits of tartar and food against the teeth. The amount of sepsis arising directly from dental caries is probably small, but indirectly dental caries leads to many infective processes, for by causing inflammation and suppuration of the pulp the infection extends into the periodontal membrane, and finally results in alveolar abscess. Further, by rendering the teeth useless as masticating organs, caries may favour deposits of food and tartar, and lead to the degeneration of the periodontal membrane. The constant presence of food and tartar between and against the teeth gives rise to fermentation and putrefactive processes. But pyorrhœa alveolaris is at the present day the commonest source of infection in the mouth.

It is interesting to note that dental caries and pyorrhœa alveolaris are seldom both well marked at the same time; this may be explained on the ground that caries is caused by fermentation and the production of acids, whereas pyorrhœa is a putrefactive process, and the two conditions are therefore unlikely to be active in the same region of the mouth simultaneously. I am doubtful whether direct absorption of organisms and toxins occurs in the mouth through the gum, periodontal membrane, or the alveolus in cases of pyorrhœa alveolaris. An examination of the surrounding tissues leads one to think that this may not be so. The chief source of infection from the mouth appears to arise from the swallowing of unmasticated and contaminated food, and from septic matter which sets up gastric and general intestinal trouble.

In the able and lucid contribution to this discussion presented by Professor Keith last week, reference was made to the importance of considering the functional significance of the large intestine. From a dental standpoint I would like to urge the importance of considering the functional significance of the mouth and the teeth. This aspect of the subject has not received the attention it deserves; yet the restoration to normal activity of the mouth and teeth is an important factor in the treatment, as well as in the prevention, of alimentary toxæmia.

As we are all aware, the function of the mouth in its relation to food is to prepare it for the digestive fluids by efficient mastication, and, with the aid of the saliva, to begin the process of carbohydrate digestion. At the end of a meal the tongue and lips have something remaining more to do in the shape of cleansing the surfaces of the teeth and gums. There should be intolerance to the presence of food on the teeth after the meal is completed, and so long as that food remains the tongue should work for its removal. For this reason it is well to end the meal, as most of the lower animals do, by drinking. When the mouth is normal and its functions are properly carried out, the teeth and gums are kept clean and free from deposits of food. At the present day amongst civilized races it is the exception rather than the rule for this function to be performed efficiently. Some years ago Dr. G. V. Black demonstrated that the force of the bite, or the masticating power, varies greatly amongst different individuals. He found also this force depended more upon personal habits in the use of the teeth in mastication, and upon the condition of the periodontal membrane, than upon muscular development. By employing a specially designed instrument called a "gnatho-dynamometer," he accurately estimated in pounds pressure the force of the bite in 1,000 persons. The average force of occlusion on the molar teeth was registered at 171 lb., and the variation was from 25 lb. to so much as

275 lb., seventeen people being able to reach the maximum. As Black has pointed out, this force of the bite is modified by diseases of the periodontal membrane, by loss of the pulp, and by the use habitually made of the teeth. Any considerable limitation in the use of the teeth soon shows itself in tenderness of the periodontal membrane, and, in the case of a person who has fallen into the habit of swallowing food without proper mastication, the power of the bite may be only 50 lb. or even less. Just as other tissues lose tone if allowed to remain idle, the periodontal membrane loses power to withstand the normal amount of pressure.

Probably the chief influence in reducing functional activity and the masticatory force is the diet, and Dr. Sim Wallace many years ago insisted on the importance of this point, and has fully dealt with the question. As he has shown, a large proportion of the food presented to us to-day can be swallowed with little, or no, mastication. This evil influence is begun in early childhood by the almost exclusive use of pap food, and the function of mastication is often allowed to remain so long in abeyance that even when a diet of suitable solid food is given, the habit of bolting has become so established, and seems so natural, that mastication is never properly performed. Many people merely pat their food into a bolus with the teeth, and then, in swallowing, help it down with a mouthful of water.

TREATMENT.

New light has been thrown on the treatment of oral sepsis, and I cannot believe that those who advocate wholesale extraction of teeth are really satisfied with the advice they give. I am one of those who believe pyorrhœa alveolaris to be a curable disease and, in my opinion, the proportion of cases which can be cured without resorting to the last extremity of extraction is constantly increasing. I entirely agree in insisting on the importance of a clean mouth. I admit also that, in a large number of cases, the disease is so far advanced that extraction is not only desirable but necessary. But I feel very strongly that, by recommending the wholesale sacrifice of teeth, we are shirking the problem which faces us; and I am convinced that the dentist of the future will not rest satisfied with such an attitude. It is, to my mind, an unwarrantable acknowledgment of defeat. Loss of teeth tends to increase the difficulty of maintaining a functional alimentary tract. By saving teeth which can be made healthy and serviceable our patients retain a valuable means of protection against alimentary toxæmia.

Carbohydrate dyspepsia has an important bearing in this connexion, and the removal of all the teeth only aggravates this trouble, for salivary digestion cannot take place with any degree of efficiency unless the food is broken up and turned over and over in the process of mastication. Is there any evidence to show that salivary digestion is less important to us in the twentieth century than it was to our ancestors? Some of those who advocate the removal of all the teeth do not even consider artificial dentures necessary, except for the sake of appearance. But, even if we do substitute artificial teeth, we cannot provide so efficient a means of mastication as Nature has given, for it is estimated that the masticatory power with artificial teeth is only one-fourth to one-tenth that of the power which ought to be obtained with the natural teeth. Then again, even when all the natural teeth have been removed the condition which induced the pyorrhœa still remains. There is also ample evidence to show that, in a very large number of cases where all the teeth have been removed, the intestinal symptoms have not been relieved. In other cases, where relief appears to have been given, its effect is only temporary. I will not go so far as to say that cure can be found for every case of pyorrhœa without extracting all the teeth, but surely we are poor dental surgeons if we do not seek something less drastic than the total removal of the organ involved in the trouble.

The consensus of opinion of those who have systematically treated patients suffering from pyorrhœa alveolaris with vaccine therapy is that in no instance has the vaccine done any harm to this condition. In a very considerable proportion of cases it has been of such value that cures may be attributed to its use. During a period of nearly six years I have been watching the results in a large number of patients who have undergone this treatment. Autogenous vaccines were employed in nearly every instance. The conclusions reached are as follows: That although vaccines are not essential for all cases of pyorrhœa, and although it is not possible in every case in which vaccines have been employed to demonstrate any marked improvement as the result of this special treatment, yet in most of the patients the value of the vaccine has been sufficiently clear. Personally, I believe that those who can be counted as cured owe the result, in a very large measure, to the help which this method of treatment affords. I have taken different types of cases, and treated them side by side—some with local surgical treatment alone, and some with vaccines in combination with local treatment—and my experience has been that those treated with vaccines have improved more rapidly and more completely than those subjected to local treatment alone. The part played by the vaccine is to give an increased resistance to the spread of the disease, and to the action of the micro-organisms producing the local disturbance.

In conclusion, I have no desire to underestimate the influence of diet, of mouth-breathing, the condition of the saliva, and the form of the teeth as factors in dental sepsis, but I am of opinion that the question of functional activity, both in its physico-chemical and muscular aspects, is the one which needs greatest attention at the present day. Believing that a large number of cases of pyorrhœa alveolaris are curable, I strongly urge more effort in conservative treatment.

Dr. R. ACKERLEY: The experience of twenty-five years of medical practice—of which nineteen were in general practice and the last six in special practice at a spa in which I see a number of patients, who for the purposes of this discussion may be regarded as suffering from some form of alimentary toxæmia—has led me to believe that these conditions result mainly, if not entirely, from certain common habits of civilized life, and especially from our ways of dealing with our food. There is only one place where we have complete control of the alimentary canal—i.e., the mouth. We have control over what we put into it, so that in a country like this none of us, or very few of us, need ingest the articles of food enumerated by Professor Saundby as containing toxins in themselves. In the vast majority of cases it is improbable that the source of any toxin is food as we put it into our mouths. Then how do toxins originate? If we accept Herter as our guide, the suckling, whether breast-fed or bottle-fed, has an alimentary canal with a somewhat simple and fairly constant bacterial flora. But, to quote his exact words,[1] "the products of intestinal decomposition in normal nurslings are remarkably small in amount when we consider the large numbers of bacteria that inhabit the lower part of the intestinal tract," and fæcal matter from the nursling can be infected *in vitro* with Bienstock's *Bacillus putrificus* without any putrefaction resulting.[2] If this condition could continue through life it is probable that we should see and hear very little of alimentary toxæmia. Now as, according to Herter, there is no material difference, so far as putrefaction goes, between the breast-fed suckling and the suckling fed on cow's milk, and as the suckling quite as much as the adult is liable to get micro-organisms in the alimentary canal through dirt and air—(Herter[1] even suggests that the *Bacillus bifidus*, which he describes as "the characteristic micro-organism of the human nursling's digestive tract," enters the body[2] "by way of the anus and that the air is the source of infection")—the happy condition of the suckling must depend not on its freedom from infection but on its power of destroying pathogenic organisms. Now how does the suckling vary from the child and the adult? Apart from the fact that it is edentulous, the main thing is that the suckling thoroughly insalivates its food and that the child as it gets older does not.

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[1] Herter, "Bacterial Infections of the Digestive Tract," Lond., 1907, p. 64.

[2] *Ibid.*, p. 14.

In theory, we are all in agreement with Professor Saundby that "all food should be taken slowly, properly masticated and *mixed with saliva*." But what really serious attempt is made to teach children or adults the necessity of insalivation? Practically none at all. From the moment the infant ceases to be a suckling food is imperfectly insalivated. Take milk alone—a food entering very largely into the diet of the child and the invalid—milk that is sipped or drunk excites no flow of saliva. Before writing this I repeated an experiment that I have performed frequently. Taking 100 c.c. of milk, I sucked it from an Allen and Hanbury's feeding-bottle, using the teat sent with the bottle, and ejected the milk every four or five seconds. This when measured was found to amount to 115 c.c. and was obviously intimately mixed with saliva. Another 100 c.c. was taken in small sips and sucked when in the mouth and then ejected; the amount of saliva obtained in this way was less than when the bottle was used, the acts of sucking being less strong and less sustained, the total amount was 110 c.c. 100 c.c. was taken in sips, retained in the mouth a few seconds and moved about by the tongue but without any act of sucking; this when ejected and measured amounted to only 102 c.c. That is to say, that only by active sucking was any appreciable quantity of saliva obtained. But is the saliva of any use so far as milk is concerned in preventing putrefactive changes in the alimentary canal? In most works on physiology it is implied that the main use of saliva is (1) to moisten the food and (2) to act to some extent on starches, but, as Bunge says,[3] "as saliva is very abundantly secreted by carnivora it is apparent that the decomposition of starch is not its main function," and surely as the food of the carnivora, the bones possibly excepted, is already moist the mere moistening of food cannot require the quantities secreted by them.

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[1] Herter, "Bacterial Infections of Digestive Tract," 1907, p. 38.

[2] *Ibid.*, p. 57.

[3] Bunge, "Physiological and Pathological Chemistry," Lond., 1902, p. 130.

The results of insalivating milk are very suggestive that saliva has a bactericidal or anti-putrefactive function. As evidence of this I venture to quote the report of some remarks I made at a meeting of the late Balneological Society in July, 1909:[1] "For the purposes of observation he put himself, two or three months ago, on a milk diet, bolting the milk, as most people do, which resulted in the formation of hard curd in the stomach, and imperfect absorption. He had headache, constipation, and the frequent passage of very unpleasant flatus. He continued that for seven or eight days. The fæces during that time were very hard. After a few days of ordinary diet he resumed milk diet, but now took it slowly, a mouthful at a time, being careful to intermix saliva freely with each portion. The result was much as he had anticipated. The first two days he had slight constipation, but after that motions were passed in the ordinary way, and they were comparatively inoffensive, and less in bulk."

This is corroborated by a statement of Horace Fletcher.[2] "A, B, C, and D were placed on an absolute milk diet. A drank his milk in the ordinary way, and at the end of three days begged to discontinue the experiment, owing to disgust at the monotony of the diet. B, C, and D continued the experiment for seventeen days, insalivating the milk, but to a varying extent, B the least and D the most. Though D took most milk he excreted least solid egesta, C excreting less than B." "Can one infer," adds Fletcher, "that increased insalivation of a non-starchy food ensured its better digestion and assimilation?" I think we can. I may add that in general practice, more than once, I had to evacuate the rectum of patients who had taken a milk diet, sipping or drinking the milk in the ordinary way, and found the excreta hard and mortary and exceptionally foul.

But to pass from milk. The starch foods which are given almost immediately after weaning, and which on any hypothesis require insalivation, are usually rendered soft, given with a spoon, or drunk out of a cup, and get no insalivation. Moistened starchy food, even when chewed, according to Pavlov,[3] causes no flow of saliva. Still further to retard and prevent the flow of saliva, we begin in the nursery the unphysiological and purely human habit of drinking with meals. The use of the knife removes another stimulus to salivary secretion. If one eats an apple in a primitive way, sucks sugar-cane or gnaws the meat off a bone, there is a pull on the mouths of the salivary glands; at the civilized dinner-table there is no such pull. So that I think it must be agreed that our habits necessarily lead to imperfect insalivation of all our food, but especially of the softened fermentable starches, to which Dr. Hale White, among others, draws attention in the remarks with which he opened this discussion. In fact, to summarize, one may say that "you can go to almost any nursery, to any school, preparatory or public, to the dining-room of any hotel or restaurant, and even if the diners at the latter are eminent members of this Society, you will find large quantities of soft foods and starchy foods being swallowed without any attempt being made to insalivate them."

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[1] *Journ. of Balneo. and Climat.*, Lond., 1910, xiii, p. 147.

[2] Fletcher, H., "The AB—Z of our own Nutrition," Lond., 1908, p. 36.

[3] Pavlov, "The Work of the Digestive Glands," 1910, p. 71.

The wild carnivora may be said to bolt their food, though they swallow a large amount of saliva with it and even before eating it; but the herbivora masticate and necessarily insalivate their food very thoroughly. The human civilized animal has reversed this process, and if he masticates anything it is his meat; his vegetables and grains and pulses and other vegetable foods he grinds and softens artificially *outside his body* and then passes them so rapidly through his mouth that practically no admixture with saliva is possible.

My own belief is that the main factor in causing those septic conditions of mouth which the dentists, especially Mr. J. G. Turner and Mr. Frank Coleman, have spoken of, is imperfect insalivation. I should like to quote H. P. Pickerill,[1] who states his convictions "that in the saliva is provided a natural and potentially perfect mouth-wash acting continuously day and night (not merely for a few minutes a day); that it is, moreover, completely under control; that it may be altered or varied in amount or composition; that its beneficial effects may be increased or decreased absolutely at will."

A second factor of alimentary toxæmia partly, but not wholly, dependent on the first, which also depends primarily on faulty habits, is the dilated or at any rate mechanically imperfect stomach. Herter[2] says, "In a stomach which secretes little or no HCl and which is sluggish in emptying its contents the chances for anaerobic developments are good, and hence we frequently find under these circumstances that there are evidences of putrefactive decomposition of food that has been unduly retained in the stomach."

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[1] Pickerill, H. P., "Prevention of Dental Caries and Oral Sepsis," 1912, p. 195.

[2] Herter, "Bacterial Infections of the Digestive Tract," 1907, p. 30.

Beginning in the nursery, apart from quick mastication and imperfect insalivation, we have: (1) The admixture of food and drink. This leads to unnecessary stretching of the stomach wall, and also to prolonged stretching, as the liquid, even if it be water, is retained unduly in the stomach.[1] (2) Too frequent meals; food being put into the stomach before the previous meal has been digested. (3) Hot, really hot beverages, leading, when repeated day after day for years, to atony of the coats of the stomach. I think, too, that hot drinks by lowering the tone of the gums are factors in causing gingivitis. (4) Use of pungent substances, known as condiments, which produce protective pyloric spasm and so lead to the stomach unduly retaining its contents.

I venture to submit that, as it is impossible to prevent micro-organisms from entering the alimentary canal, our attention should be directed, beginning with the nursling, to ensure their rapid and complete destruction by developing habits which will ensure a clean mouth, thorough insalivation of food, and prevent stasis, fermentation and putrefaction in the upper part of the alimentary canal. The best proof of the value of this line of procedure is the great improvement that results in general health, even in persons of advanced age, when the following habits are acquired and persisted in:—

(1) Thorough insalivation, which is only obtained by active sucking of all foods, especially of soft foods.

(2) Taking no beverage of any kind with meals, water being taken on an empty stomach three-quarters to a half-hour before meals.

(3) Avoidance of hot drinks of every kind.

(4) Allowing at least four hours between any two meals.

(5) Avoidance of condiments and all substances of an irritating character.

Mr. F. St. J. Steadman: In a discussion such as this on alimentary toxæmia, as it might have been anticipated, the disease known as pyorrhœa alveolaris has played an important part, for this complaint is not only very common, but it is situated at the very commencement of the alimentary tract, and must therefore inevitably have a profound influence for good or for evil upon it.

[1] *See* Cannon, "Mechanical Factors of Digestion," Lond., 1911, p. 118 (footnote).

In the first place, with regard to Mr. Arbuthnot Lane's theory that pyorrhœa alveolaris is secondary to intestinal stasis. I think that no one who has had much experience of pyorrhœa alveolaris or has studied its ætiology could possibly support this view. Thanks largely to the work of such men as Mr. J. F. Colyer, Mr. J. G. Turner, and others, we have now a fairly good idea as to the factors causing the disease. It is due to the lodgment and subsequent decomposition of the sticky carbohydrate food, or as Mr. J. G. Turner well says, "to germ-carbohydrate stagnation."

If Mr. Arbuthnot Lane is correct in his proposition that pyorrhœa alveolaris is secondary to intestinal stasis, which in its turn is largely brought about by man's upright posture, how does he explain its occurrence in the domestic animals, such as the cat, dog, and horse? Pyorrhœa alveolaris is rampant in these animals. It becomes common among animals which are subjected to the will of man, that is to say, when their natural diet of raw uncooked food is replaced by soft carbohydrate or other sticky food which readily lodges about and between the teeth. In these domesticated animals every stage of the disease is found as in man. Exactly how the disease is brought about is very interesting, and I cannot do better than quote a few remarks from a recent article by Mr. J. F. Colyer on periodontal disease.[1] "If the normal mouth of a horse is examined the muco-periosteum will be noticed to fit snugly into the interproximal spaces. The first sign of trouble is the destruction of the muco-periosteum at this place, with the result that a triangular space is formed. In these spaces one may find a grain of corn, a little chaffed hay, or other material. At this stage the muco-periosteum appears to be only slightly inflamed. The space gradually increases in size and the periodontal membrane becomes more and more involved, and with the increase in size of the spaces there is a greater accumulation of debris. The muco-periosteum becomes thickened and there is a profuse muco-purulent discharge around the teeth, the breath being extremely offensive. With the advance of the trouble, the teeth tend to separate and the food wedges between the teeth, the destruction of the bone continues, the teeth loosen, the infection spreads in the maxilla to the antrum setting up suppuration and in the mandible causing an abscess." I have myself observed these various stages of the disease in the mouths of dogs.

To Mr. Arbuthnot Lane's statement that pyorrhœa alveolaris cannot be primary because the removal of the teeth does not cure the intestinal stasis, I would reply that one would not expect it to. I can see no more reason for thinking that removal of the teeth must cure a secondary infection in the large bowel, once the organisms have obtained a firm hold, than that a fire which has spread to St. Martin's Church from the National Gallery would be extinguished in the Church by putting out the fire at the starting-point.

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[1] *Lancet*, 1913, i, p. 1085.

In the second place, I listened with very great interest to my colleague Mr. Goadby's excellent speech. I find myself in close agreement with his statement that the toxins absorbed by the blood-stream in intimate contact with the areas of rarefying osteitis in the jaws, are of even greater importance than the mere swallowing of pus. I cannot agree, however, with his remarks on the treatment of the disease. Some few years ago as house surgeon, and later on as medical superintendent, at the National Dental Hospital, I had an excellent opportunity for fifteen months, of which I availed myself, of observing Mr. Goadby's work in the treatment of pyorrhœa alveolaris by vaccines. I am bound to confess that I am not able to corroborate the results he claims for this line of treatment. During that period I was not able to satisfy myself that the vaccines had any permanent beneficial effect upon the disease. In those cases where there was no local treatment I cannot recollect a case in which I could detect improvement either in the local condition or in the constitutional symptoms. In those cases where local treatment, such as the removal of septic stumps, and scaling of the teeth, was carried out, there was often improvement, occasionally well marked, in the local condition, and sometimes in the constitutional symptoms. This improvement, however, was not, as far as I could see, any more than I have frequently been able to obtain in my own practice, by carrying out local treatment alone without resorting to vaccines. In one case, however, the vaccines did appear to be beneficial. It was a case of a woman, aged about 35, who had that muddy complexion so often associated with chronic toxæmia. Her complexion and general health improved very much under the vaccine treatment; but owing to some domestic trouble she was prevented from attending the hospital for a few months, and when she did attend her muddy complexion and general ill-health had returned. This case is interesting because it seems to support my view, that in those cases in which one is fortunate enough to have isolated the organism causing the disease, and obtained a vaccine from it, the vaccine simply treats the symptoms and does nothing to improve the local disease itself. This leads me on to another objection to the vaccine treatment—namely, the great difficulty of isolating the organism causing the disease. Up to the present no specific organism has been discovered. The examination of the pus from the pockets, even when the most careful precautions are taken to prevent outside contamination, and to obtain only the pus in the deepest part of the pocket, generally reveals a mixed infection, often as many as ten or even more distinct organisms can be isolated. It is extremely difficult to decide which of the organisms is the cause of the disease. The only method at present known to us of ascertaining this is to take the opsonic index of the patient to each one of them. This is a lengthy and laborious proceeding which, however, might be well worth doing if the result obtained was reliable, which, in the present state of our knowledge, it is not.

I have found in some cases an almost pure culture of a variety of the staphylococci, and in other cases a variety of the streptococci and a very large number of other organisms have been found by various observers. It does not seem to me probable that a specific organism will be found, for in other parts of the body very many different kinds of organisms can produce suppuration in bone, and pyorrhœa alveolaris is, after all, merely a suppuration of the alveolar processes of the jaws.

Mr. Goadby advocates the laying open of the pockets round the teeth and claims that this proceeding in conjunction with vaccine treatment cures the disease. This laying open of the pockets, however, is not quite so simple as it sounds, for while it is perfectly easy to lay them open on the labial and lingual aspects of the teeth, how are the pockets in the interproximal spaces to be treated? If the case is at all advanced, the pad of gum in these spaces is soft, swollen, and spongy and the pockets deep, so that in order to get free drainage, which is the object of the operation, the whole of this pad must be cut away. This, however, leaves a triangular space between the teeth which I have already shown in describing the pathology of the disease in the horse is so detrimental. The function of the pad of gum, which normally should be quite firm, is to prevent the lodgment of food and debris between the teeth. If this is removed a space is left in which food must inevitably lodge and decompose. Now, considering the important part the lodgment of food plays in the production of the disease, the deliberate creation of these spaces does not seem, to my mind, to be either a logical or reasonable line of treatment. The result of it is that the disease almost invariably recurs within a few months, as one would expect.

Mr. Goadby further says that these pockets must not be laid open or teeth extracted until the patient has been subjected to a more or less prolonged course of vaccines, for fear of setting up ulcerative stomatitis in the one case and dangerous constitutional effects in the other. I must confess that, for reasons I have already indicated, I have not had very much experience in the laying open of pockets, but in the cases I have treated in this way, where the patients have refused to have the teeth removed, I have never seen an ulcerative stomatitis develop, nor any other immediate ill-effect from the operation, though I have not resorted to vaccines in the majority of these cases. I have, however, had a very considerable experience in the extensive removal of teeth for pyorrhœa alveolaris, which is a much more severe operation than the above. My experience has taught me that if the area of operation is thoroughly cleansed before operating, no ill-effects whatever will result in the vast majority of cases. Indeed, it is remarkable how speedily the patient recovers after the operation. In very many instances among my hospital cases, patients have returned to work the next day or even on the same day. I do not consider that this is a wise proceeding on their part, but among the poor it is often difficult for them to be absent from their work long.

I have operated on patients in the wards running a temperature from the toxæmic absorption from the disease, and I find the temperature in these cases generally comes down to normal within twenty-four hours. In a recent case at the Metropolitan Hospital, where I was called in by my colleague, Dr. Langdon Brown, in which the patient had been running a temperature for some weeks before admission, the temperature was rising at the time of the operation. It did not even complete its usual rise, but dropped immediately to normal and remained normal.

I cleanse the area of operation by swabbing out the pockets carefully with hydrogen peroxide and then painting the whole surface of the gums and pockets with a 2 per cent. solution of iodine in rectified spirit just before operating. When the mouth is in a very septic condition, it is wise to scale the teeth thoroughly and put the patient on a hydrogen peroxide mouth-wash for a week before operating. In view of these facts, and when it is remembered that hundreds of similar operations are being carried out in our various hospitals every week, I do not think that Mr. Goadby's statement that a course of vaccine is absolutely necessary before operation can be accepted, although in some cases it may be advisable as an additional precautionary measure.

for, as Dr. Hale White said, they can do no harm and may do good. Indeed, if his statement were correct, considering the very large number of poor patients suffering from pyorrhœa alveolaris, the outlook would be black indeed, for he would be one of the first to admit, I think, that a preliminary course of vaccines among them is utterly impracticable.

Mr. Goadby said that the removal of the teeth does not always cure the disease, because there are areas of rarefying osteitis left after their removal which make the wearing of artificial dentures painful and uncomfortable for a long time. My reply to this is that the teeth have been extracted too late in these cases. The disease has been allowed to progress until the rarefying osteitis has spread beyond the alveolar process or that part of the jaw which we dental surgeons know as the bone of attachment—viz., that part of the bone which is developed after the eruption of the teeth for their support and which is absorbed after their removal. If the teeth are removed early enough, this trouble not only never arises, but the patient will be left with well-marked prominent alveolar ridges to which comfortable and efficient dentures can be fitted. Whereas, if the teeth are removed too late, or are allowed to drop out, there will be practically no ridges left, and it becomes impossible to adapt efficient comfortable dentures to them. This is a strong argument for the early removal of the teeth. If it be granted for a moment that the disease is progressive and incurable, it is surely wiser to adopt the first course. He also said that the removal of teeth for pyorrhœa alveolaris is a confession of failure, which must be admitted. The same may be said of the surgeon who removes a leg for sarcoma. An honest confession of failure is often the best course in the interest of the patient. It is no advantage to a man to save either his teeth or his leg and lose his life.

In conclusion, I must reply to one more remark of Mr. Goadby's—namely, that the extraction of teeth in a young adult for pyorrhœa alveolaris is "an unwarrantable and barbarous procedure." In this disease I think that in the early stages, in some cases at any rate, there is undoubtedly one law for the rich and another for the poor. An educated person can sometimes, by the expenditure of a considerable amount of time each day, by very thorough and careful cleansing of the mouth, arrest the disease. But not so a poor person of the uneducated class. He cannot afford either the time or the money. I agree with Mr. J. G. Turner that once the normal healthy condition of the periodontal membrane has been lost in such a person it is never afterwards regained. In the present state of our knowledge there is no known method of arresting the disease in them once it is established. It will progress until the teeth eventually drop out. What, then, is to be done in the case of the young adult with the disease well established? To my mind, knowing as we do the risk he is running of becoming practically a chronic invalid; realizing how likely it is that such a man will be constantly among the unemployed on account of his inefficiency; and realizing, if he be a breadwinner of a family, the consequent hardship upon them, to allow that man to retain his teeth without at least placing these risks clearly before him, is an unwarrantable, a barbarous, and, indeed, a grave offence against the patient and the community.

Dr. LENNOX WAINWRIGHT sent the following communication: As the subject of alimentary toxæmia has been discussed, and many causes assigned for its production, I shall confine myself to those examples which we know are produced directly by food products.

There is the classical tetany produced by irritation and toxæmia in children. Rickets, again, is a disease that is probably caused by toxæmia in the alimentary tract, and it must be borne in mind that the first time an infant takes artificial food it lays itself open to alimentary toxæmia. Food fever and convulsions are other examples of alimentary trouble in infant life. In all these cases corrected dietary does wonders and in each case where the dietary is altered the media for bacterial support is changed, which means that the microbes are more or less deprived of their pabulum and of their power to produce products which are inimical to human health and life. It is difficult to assign the part microbes play, but I believe that they act by producing chemical toxins which are of such character that they act as poisons. In depriving microbes of their pabulum and changing the media in which they live we render them unable to produce the poisons which are detrimental to the human system.

In adult life the results of bad early feeding are shown in the form of adenoids, tonsillitis, granular pharyngitis, dilated stomachs, and intestinal weakness. All of these may be present, or any one of them, but they are generally associated with melancholia and misery in which there is no future hope and the happiness of the home is impossible.

Incidental causes such as epidemic poisons and the various diseases that produce paralysis and loss of power are responsible for a great deal of want of proper action of the muscle wall of our viscera. These ailments are much more commonly the cause of stasis than they are given credit for. Given that there is inefficient action due to want of proper innervation the bowel is liable to get into a toxic state, because the inherent activity and proper secretions are in abeyance.

Œsophageal pouches have not been mentioned as a cause, but I once had a remarkable case in which an œsophageal pouch produced symptoms diagnosed as typhoid fever, recurrent influenza and intermittent fever. The patient had tetanic spasms of the hand, and an enormously dilated stomach. Some years after the pouch was X-rayed and removed.

The medical treatment of alimentary toxæmia is based upon the common-sense line of treating the cause. Where one treats by vaccines when such diseases as coli infection and pyrrhœa co-exist, the vaccine is useful, but utterly fails unless the large focus of infection is removed, and I know of many instances in which the teeth were to all intents and purposes perfect and had been for many years, and yet the patients have had intestinal toxæmia of a very severe character producing rheumatism and more or less melancholia. Although pyorrhœa and mouth infection have a very important bearing on this trouble I think we must admit that the large area of the intestines frequently plays a greater part in the production of ill-effects; I am quite sure of this, that the mental effect on many patients of prolonged intestinal toxæmia is such as to make them almost demented, and I believe that if many of our asylums were invaded by a good, sound clinical physician who would approach the subject without any preconceived idea, in many cases supposed to be hopeless the melancholia and hypochondriacism would yield to common-sense treatment and reduce the number of insane people.

Where toxins have produced severe debility and low pressure it must be admitted that the brilliant results obtained by Mr. Arbuthnot Lane in apparently hopeless cases are of some weight in favour of surgical interference. If neuralgia is the prayer of a nerve for healthy blood there can be no greater argument for attending to intestinal stasis than a constant neuralgia in any individual. If the proper drainage of tissues is essential to life, then the removal of toxin-bearing areas is justifiable by surgical procedure. I do not want to be biased in any shape or form; I want all credit to be given to medicine and all credit to be given to surgery, and should like to approach this question with the enthusiasm of Mr. Arbuthnot Lane and the judicial impartiality of the opener of this debate.

The Royal Society of Medicine.

May 5, 1913.

Dr. AMAND ROUTH, Vice-President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.[1]

PROFESSOR H. J. HUTCHENS, D.S.O.: In continuing this discussion to-day I feel some hesitation in laying my views before you, as many of the points I would have submitted have already been adequately dealt with by others.

As I understand the term "alimentary toxæmia," it means the circulation in the blood of certain poisons which are elaborated in the alimentary canal. These substances circulating in the blood will combine with, and to a certain extent injure, those cells of the body for which they have a chemical affinity. First, in regard to the source of these poisons. They may conceivably be elaborated in the alimentary canal in two ways: either what we may describe as chemically—by the action of digestive juices acting on the foodstuffs ingested—or as the result of the activity of bacteria, and it is on this latter part of the subject I wish to dwell briefly.

The poisons produced by bacteria in the alimentary canal may be of two possible kinds: (1) those which are the specific products of the bacteria elaborating them; (2) the non-specific products, which may be produced by bacteria apart from their specific products. One knows, for instance, that outside the body the colon bacillus produces indol, but indol is not the specific toxin of the colon bacillus. It is not yet known what the specific toxin of that organism is; indol is merely one of the non-specific products. With regard to the relation of bacteria to intestinal toxæmia, a very great deal has been said against micro-organisms, but very little, it must be admitted, has been proved. The problem is one which has been under discussion and experiment for very many years. It originated out of certain experiments of Duclaux on the nutrition of plants. That worker came to the conclusion that plants could not subsist by their own activities, that they depended for their nourishment upon the activity of micro-organisms with which they were associated, and the question was then raised by Pasteur whether or not bacteria were equally important for the nourishment of the animal body. As you know, a very large number of experiments were conducted to ascertain whether or not animals can exist by the aid of their own digestive juices, apart from any aid derived from bacteria. The experiments were very inconclusive, so that we have now what are practically two schools of thought, one of which maintains that bacteria are essential to animal life—that they provide nourishment by breaking up food material in such a way that it can be more readily absorbed by the animal body. And they declare that not only do the intestinal bacteria provide nourishment, but they act as a protection to the human body against pathogenic species of organisms.

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[1] Adjourned from April 28.

Very little work, so far as I know, has been done to ascertain what good effect bacteria may have on the body; almost all the experimental work has been monopolized by the other side, which is largely led by the French, who maintain that bacteria are altogether harmful; that they are unnecessary for the nourishment of the body; that the digestive juices present in the animal are quite sufficient to elaborate the products necessary for absorption and the maintenance of life. And in support of that argument they raise the point that it is in the large intestine chiefly that bacteria are found, where, in fact, practically no digestion takes place. They say that the small intestine is the seat of digestion, and the number of micro-organisms there is comparatively small. And they go further and maintain that certain products elaborated by micro-organisms, such as indol and cresol—definite chemical compounds—are distinctly harmful to the body, and that these are produced characteristically by a large number of species which are present in the alimentary canal. In addition there are a number of other organisms in the alimentary canal which do not yield products harmful to the tissues, and these they regard as saprophytic species. But those bacteria which produce such substances as indol, skatol, &c., are regarded as particularly harmful, seeing that the latter are said to contribute to the formation of those sclerotic changes which are characteristic of old age. And in order to obviate those changes Metchnikoff urged the use of certain other bacteria which would make the conditions in the alimentary canal such that those micro-organisms could not exist, and so could not elaborate the products. In other words, he recommends that one should take large quantities of bacteria which produce a certain amount of acidity in the alimentary canal, the acid medium making the latter unfavourable to the residence of the colon bacillus and such organisms. The difficulty is that this is merely hypothesis; there are very few facts in support of it. The bacteria found in the alimentary canal are now being isolated and systematically studied in order to determine the products which they form and the effects of those products on the body. What will be more difficult to ascertain will be what products these organisms form out of the more or less digested foods, not when they act alone so much as when they are associated with other species. The problem becomes extraordinarily complicated, and it seems a long way round to get at the solution, but it is the line along which the French school of investigators have been working.

The view that the intestinal flora is altogether harmful has been criticized in many ways. Firstly, on the ground that substances such as indol, skatol, &c., are not harmful to the human body. It would appear from animal experiments that indol is not as poisonous as it had been thought to be; but whether it is harmful to man is still indefinite. It may be that these products, which are said to be absorbed from the alimentary canal and to produce sclerotic changes in old age, are only the result of a further chemical digestion taking place in that part of the alimentary canal where the residue of foodstuffs remains before being excreted. Again, though as a rule decomposing flesh is rejected as food, yet it is notorious that some peoples eat unsound food with impunity. Similarly, it is a fact that many of the chemical substances which have been isolated by chemical means from decomposing meat produce severe symptoms when inoculated into animals, but it has yet to be shown that these so-called ptomaines can cause food poisoning in man. When illness follows the consumption of unsound meat it is not because the meat contains the non-specific products of bacterial activity, but because it contains certain species of organisms well known to be pathogenic to man, or because it contains the specific toxin of a highly pathogenic organism, *Bacillus botulinus*. Another criticism advanced against the view that bacteria cause harmful products is, that such symptoms as those of infantile diarrhœa are manifested under quite different conditions of the intestinal flora; that is to say, that the intestinal flora may vary very much, and yet under these different conditions you can still get the ordinary symptoms of intestinal diarrhœa. So, in opposition to Metchnikoff, there is a school which maintains that the non-specific products of bacteria are not harmful to man. And, for the moment, it must be admitted that the facts are not altogether sufficient to enable a dogmatic statement to be made in favour of one side or the other.

When one considers the question of specific toxins normally present in the alimentary canal, one has to realize that little or nothing is known of the nature of specific toxins; they have not been isolated even in approximately pure condition. But there is one fact in connexion with these toxins to which I would draw attention, and that is, that in many cases of alimentary toxæmia one finds that colon bacilli are present in the urine when one would not have suspected it. I was led to that observation in the course of certain other work which I was doing, and I found a considerable percentage of cases of intestinal toxæmia had this bacillus in the urine. It looks as if the colon bacillus can produce a true bacillæmia or septicæmia. In these cases there may be no cystitis, or anything to direct attention to the state of the urine, and I think this fact may prove to be a significant one.

Sir JAMES F. GOODHART, Bt.: I am somewhat in doubt whether I ought to have responded to the invitation that I should join in this discussion, for much that I could wish to say seems to have been implied or said by those who have preceded me. There can, of course, be no doubt that alimentary toxæmia of one kind or another does occur, and the chief question, perhaps the only one, that arises is, under what circumstances, and to what extent, does it do so? On this head it appears plainly, from the admirable survey of Dr. Hale White, and from the remarks of Dr. Andrewes and Dr. Ledingham amongst others, that there is considerable doubt; indeed so much to me is this the case that after a careful perusal of the discussion as far as it has gone, I have not been in such a state of mental indecision since the days when I first essayed to make choice of a motor-car.

There may therefore still be room for a few words from those who approach the subject by the road of clinical medicine, to give such impressions as they have arrived at in the course of years; and mind, I have no wish to dogmatize—they are but impressions. When I entered the portals of medicine forty-nine years ago, there was a maxim abroad that no one became pyæmic—most septic troubles in those days were so-called from surface wounds. So that abrasions, skin lesions of various kinds, even such things as boils and carbuncles, and it may be assumed superficial ulcerations of the mouth, and perhaps of the intestinal tract, were not considered likely to lead people into danger, some normal reaction in the part protecting them therefrom. And *this being born of experience was largely true.* We have gone far since those days, and we now know a great deal more about sores as being all prone to the implantation and cultivation of germs, so that we dwell very insistently upon their importance as foci of infection, but what seemed self-evident before, and indeed then unduly dominated our methods of treatment, we now seem almost to neglect; I mean those existing and active protective agencies within our bodies, our knowledge of which has also been advanced, but the power of which, it must be admitted, it is difficult to sum. In my view this has a special bearing upon alimentary toxæmia, because that subject has been in a very special manner linked up in these later days with the mouth and the colon.

As regards the mouth, the disease that we now know as pyorrhœa alveolaris was never heard of in the days that I speak of, whereas now I seldom go long without something so called being shown me, or where oral sepsis with a big O is suggested as the cause of symptoms all and sundry, and when, on being asked if I am prepared to agree to the extraction of some good teeth, or to a course of vaccine, I pause for a time to think, I suspect I am summed up as too old-fashioned to be able to agree. But it is not that: it is that there, and here this afternoon, one asks the question, Is there a sufficient body of evidence as yet accumulated to make sure that after we have deducted the influence of the natural restorative reaction of the part, a vaccine has permanently established itself as the most successful line of treatment? We have long known, of course, that deep-seated suppuration in connexion with tooth or jaw may give rise to a fatal septic poisoning, and there is no doubt that there was and is room for man to take more care of his mouth; but notwithstanding that admission, I think that the fascinations of oral sepsis have run away with very many of us, and that it is pushed for more than it is good for. The natural antisepticity of the cavity seems to be overlooked, and the experience that everyone of us has in common comes to be overlaid. Some three or four years ago I saw a man—a doctor, too—who had been actively engaged, I was told, in treating a pyorrhœa alveolaris for nearly a year. The condition of the gums was really of the slightest, and examination showed that he was suffering from advanced cirrhosis of the liver, from which he died in the course of a few weeks. And then with regard to the disease pernicious anæmia, let me ask the same question whether there is any sufficient evidence as yet that Dr. Hunter's suggestion of its origin in oral sepsis has had any effect in controlling the disease? Years ago there came to me a man of middle age with a very severe anæmia and a very diffuse pyorrhœa associated with it. He had all his teeth removed and he recovered, I think, quickly; but I have not seen any case like it since then, and I cannot say for my own part that I have any evidence that oral sepsis has any cardinal place in the causation of this very fatal disease. Nor, for the matter of that, do I know that the treatment of constipation has done anything either, and arsenic still remains for me the only treatment that seems to have any effect upon it. I should therefore venture to hold that, while offering suggestions that may ultimately become fruitful, the ill-effects of much of the oral sepsis at the present time is somewhat visionary, and that the toxic hypothesis of the treatment founded upon it must still be considered as in *the experimental stage*, and that we need to be a little more careful to make clear that this is so.

When we turn for a few moments to the nether end of our digestive tract, here also is to be found an undue insistence upon what is but an occasional pernicious perversion of function, to the disregard of a consideration for those self-protective agencies that characterize healthy organs and tissues. I agree with Professor Keith that this discussion centres round the nature and function of the great intestine, and it is upon these that I wish to remark. But as I have mentioned Professor Keith's name I hope I may be allowed to say in passing how much the entire medical world must admire the work that he is doing at the Hunterian Museum in helping us "to solve the practical problems of applied medicine." The subject of the functions of the colon, and of constipation, have always been of interest to me, because, as I have often said, the teachings of physiology in respect to them are not, I believe, altogether sound. For the last few years we seem to have been obsessed by the maleficent action of the one and the obstinacy of the delay in the other, and, as is well known, these have led to the suggestion of doing away altogether with the large bowel in certain cases. Surgery has indeed demonstrated that certain persons can live apparently in comfort without it. A very interesting fact this, but no argument that the colon is useless to mankind at large—a surely distorted view. But there is another view that cannot be disregarded, which is that the colon is a great benefactor and has gradually elaborated itself, probably is still doing so, as a great organ of digestion. It is said that the colon absorbs little but water. I cannot bring myself to believe it. In the post-mortem room in days gone by, often has one endeavoured to cleanse the surface of its mucous membrane, for purposes of demonstration, under a strong stream of water from a tap. I have done it not once but hundreds of times, and I know that the cleansing can only be accomplished by considerable manipulation, and why? Because the mucous surface clutches the smear with such great tenacity. To my mind the essential object of the existence of the colon is to display as great a mucous surface as possible to the food that is supplied to it, and to this end it has become an organ for *delay* and *storage*. Yet, astonishing as it seems, say half the world considers these functions morbid, and is bent upon defeating them. That the colon should be *full* and *not empty*; satisfied and not hungry, is, I maintain, sound physiology; yet, encouraged by us doctors, hundreds of thousands of human beings have grafted into themselves the idea that they were born into the world for the main purpose of getting a daily evacuation of their bowels, for should they fail in this they will be poisoned by the absorption of the noxious products that are, they suppose, grown in this their kitchen garden.

We talk of the sacculations of the colon as if their sole use were as traps for bacteria, that prove sources of septic inflammation and of toxic fever, and so they may be, and no doubt *are*, upon occasion, but their main service is as extensions of the mucous surface of the bowel, which thus becomes more of a *multum in parvo*, and I doubt if many of us could do without this, our Gladstone bag.

Then what of the contents of the bowel, the mucus (yes, mucus has its good points), the various colonies of organisms, the gases, the juices, of which we are all talking with seemingly only one idea in our heads, and that a poor one. Have these no *useful* office? Who can controvert the assertion that all in their turn are not only serviceable but necessary, by breaking up the food, and in changing its nature, so that it may be presented to the mucous surface in a more intimate and more assimilable state. Then these bacteria of various strain, what evidence have we that *they themselves* are poisonous agents? Do we know for certain from whence they come; can we yet state positively what they are? I gather that Dr. Ledingham thinks "No." And for myself I would like to hazard the opinion that with the loose habits known to prevail in these lowly symbionts it must often be a wise child to recognize its own father. Who will gainsay that in these low strata of living atoms life may be, so to speak, amphoteric?

Then as regards *delay*. Surely it cannot be in doubt that it is an essential function of the colon. Years ago I was fortunate in coming across two or three cases of fistula low down in the small intestine, and from them I learned that stout (coloured liquid) when taken in by the mouth appeared again at the abdominal opening in about seven minutes, from which I assumed that there was very little delay in the small bowel. That this is so has now been repeatedly demonstrated by Hertz and others by means of the X-rays. That creamy chyme alone is found in the small intestine, and that as soon as the contents reach the cæcum scybala may be found, show, both that an enormous amount of absorption *must* go on in the large intestine, and also what necessity there is for delay, and I would venture to submit that although upon occasion they may prove a source of danger, the kinks and pouches that are present in all cases are but the natural convolutions of a pleated surface brought about by this necessity.

Lastly, I would ask you to consider the character of the intestinal muscle and of its vermicular action. Short are its fibres; little by little is the feature of its action. A kink to you and me is no kink to it; it is occupied every minute of the day in negotiating kinks and in getting round corners, and it is, I suspect, only when a kink becomes something *more*, and approaches a volvulus—and that is comparatively rarely—that mechanical interference is required. And may I be pardoned for adding just this word of caution, that to see a kink with one's own eyes is not necessarily a proof that an obstruction or even delay is in being.

Yet one other point as regards the muscular function of the large bowel that is often forgotten. In this poison-bag theory of the colon it seems to be suggested that it is a mere sac in which food lies passively and rots. Is that any adequate idea of the power of the colon? I shall never forget an afternoon when I stood by the side of one of my colleagues, now many years ago, as he performed the operation of inguinal colotomy. The operation was to be done in two stages, and the colon having been carefully secured to the edges of the abdominal wound, the patient was about to be put back to bed, when suddenly the muscle woke up in a fury, and with a snap that I can hear now, it tore away the stitches and retreated into the abdominal cavity. You must, many of you, have often felt the stony hardness of the muscular tumours that the colon will produce, when it is really in labour. If it *wants* to get rid of its contents it needs no assistance provided that the passage is open, and yet we are asked to suppose that it is a mere cloaca full of noxious contents, and that it can make no effort to expel them. I draw the inference that these contents are not of this refuse character, that it *wants* them, that it *needs* them, until very low down in the intestinal canal, and that then expulsion is of far less moment, and is accomplished without supreme difficulty.

I put these few points, Sir, because I have thought the subject of intestinal toxæmia has been edging of late a little off sound lines, and is occupying not quite the position that the facts justify. Indeed, I think it would be a good thing could it be recommitted to the quiet of the physiological and pathological laboratory, for still further investigation, and to the bedside for further patient clinical observation. In saying this, no doubt, I lay myself open to the retort that the physician in the present day is too often little more than an obstructive. It is not so, he is as ready as anyone to seize upon every modern idea that seems likely to be useful in the treatment of disease, and there is no one of us, I suppose, who is not indulging in the hope—nay even in the expectation—that a wider knowledge, say, of the effect of grafting, and of the symbiotic fusions of vegetable physiology, may have a far-reaching and useful bearing upon the processes of *animal* life, and upon the management of disease ; but it is no more than our duty to try and see things in their just proportions and to be as sure as may be that we are on solid ground before committing other people to everything that the pioneer puts before us. This is, indeed, but justice to the pioneer. Greatly as one must admire the skill of Surgery in marshalling its facts, and the energy and courage with which it has obtained them, we may, I am sure, be allowed to question its inferences, for, if not, we may almost be persuaded to weep for humanity that it ever emerged from the four-footed beasts, for so dire are the effects that are pictured of the upright posture, that even colectomy might reasonably take its place as the bedfellow of popular remedies. But, Sir, I must gently demur ; when appealing to its own experience Surgery declines to allow me to appeal to mine, and bids me to accept without quibbling "the hard facts," as I agree they are, "of operative experience." You remember the old remark—supposed to be consolatory—that a man can only die once. I prefer another aspect of the same, that a man can only live once—it is the larger truth. For what if a going concern should have been scrapped in place of a crooked doctrine.

Let me leave it there. I dreamed that as I walked along in the evening of my day there passed a band of my distinguished confrères on their way by new and dangerous methods of locomotion to visit the Garden of Eden, and as they arrived at the entrance of that enchanted land there occurred a temporary hitch—a portal or intestinal stasis, in fact, within, and as I gazed I saw that it was occasioned by a body of attenuated and diaphanous creatures, evidently of the colectomied ones sent there to convalesce. But as my friends severally entered in, I heard this question addressed to them, in tones more lugubrious than might have been anticipated: "We have arrived here safely! But now what are we to do, for we cannot take fruit?" Which thing is an allegory.

Mr. W. Lang: In the admirable contribution which Mr. Lawford has made to this discussion he names me as one of the first to recognize the connexion between pyorrhœa and inflammation of the iris and ciliary body. This observation was made many years ago, and although I have never written on the subject, I have for the last ten years been teaching, both at Moorfields and at the Middlesex Hospital, that a chronic source of sepsis could cause an inflammation in any part of the eye.

My attention was first drawn to the subject of oral sepsis as a factor in the causation of eye diseases by an original observation made by my friend and colleague Mr. William Hern, who told me he had found pyorrhœa in every case of acne rosacea, and noticed that the eruption improved or disappeared after the mouth was treated. At that time a patient with acne rosacea was under my care for attacks of keratitis, which had recurred, at intervals, in spite of local and general medication, but after treating the pyorrhœa the keratitis ceased to return. From this time onwards I examined the gums in all the cases of inflammatory affections where the ordinary accepted causes of the disease were absent, and thus gradually I came to recognize that pyorrhœa caused inflammation in every part of the eye. After a time, in cases where all the other recognized causes were absent and the mouth was clean, I began to look elsewhere for the source of sepsis. In this way I gradually extended my searches until I recognized that the source of sepsis might be in any of the mucous membranes, in a chronic sore on the skin or in a sinus opening on the skin or on a mucous surface.

Although in common with other observers I failed, after much investigation, to discover the nature of the poison, nevertheless, after watching the effects produced by removing the suspected source of sepsis I was convinced that the diagnosis was correct; for as soon as the source of sepsis is removed the inflammation begins to subside, the rheumatic pains in various parts cease, a feeling of well-being takes place, and in recent cases of central choroiditis the lost vision quickly returns.

In hospital practice, where clean mouths are the exception, one sees every day many cases of inflammation of the eye due to oral sepsis. In order to find out what proportion of the cases of inflammation of the eyeball occurring in private practice were due to sepsis, I have had a table made from the notes of my last 10,000 patients. In it is included every case of inflammation, with the exception of those limited to the conjunctiva, where a cause for the disease had been found. The results shown in these tables were unexpected, especially as the cases due to sepsis must have been underestimated in the earlier years, because no search was made in many of the parts of the body which now would be investigated. For example, in the absence of bladder symptoms the urine, when it appeared to be normal to the eye and to the smell, was not examined for micro-organisms, though it might have contained many thousands to the cubic centimetre and have been the cause of the eye affection. Owing to this increase of knowledge the number of cases in which the source remained undiscovered has decreased, until it is now half what it was at first and forms 1·2 per cent of last year's patients.

On comparing the number of cases in Table I and II, the importance of sepsis in the causation of eye diseases is obvious, since 215 are attributed to it alone, and only 168 to all the other recognized causes. Of the 215 toxic cases 180, if the three tonsil cases are included, are due to alimentary toxemia, and 139 are credited to pyorrhœa alone, hence the importance of oral hygiene. Cases due to gonococcus have been included in this table, because when these patients come to the ophthalmic surgeon they are suffering from iritis and cyclitis, due generally to an infection of the prostate, the remains of an uncured urethritis, which must be dealt with by massage and vaccines if a permanent cure is to be effected. The remaining causes are very important, although they form such a small minority, since they indicate the sources that must be investigated if an obscure case is to be elucidated. As an example I will instance that of an unmarried lady who consulted me in 1890 for an error of refraction, and came again in

1905-06, first with patches of choroiditis, and later with an irido-cyclitis; she was anæmic, and the dentist said she had spongy gums due to tartar, which he removed; still the eye did not recover. But when in October, 1906, Sir John Bland-Sutton removed a mucous polypus from the neck of the uterus, and thereby relieved dysmenorrhœa of some years' standing, the eye quickly got well and has not relapsed.

Tables IA and IIA will be of interest chiefly to the ophthalmic surgeon. They record the number of different parts affected; in many patients the disease attacked more than one part. In the septic group, Table IA, it will be noticed that the uveal tract is the part chiefly involved, hence the greater number of secondary cataracts; whereas in Table IIA the sclerotic and cornea, the retina and optic nerve are more frequently affected.

TABLE I.—THE SOURCE AND NUMBER OF THE CASES ATTRIBUTED TO SEPSIS.

Lachrymal sac	1
Antrum of Highmore	1
Nasal inflammation	2
Inflamed tonsils	3
Pyorrhœa	139
"Indigestion"	2
Appendicitis	3
Large gut infection, including colitis and the like	33
Kidney and bladder	4
Male urethra	20
Uterus and appendages	3
Skin diseases	4
Total	215

TABLE II.—THE CAUSES TO WHICH THE OTHER CASES WERE ATTRIBUTED AND THEIR NUMBERS.

Syphilis, congenital	40
,, acquired	35
Tubercle	27
Albuminuria	24
Gout	23
Diabetes	12
Following acute fevers	7
Total	168

TABLE IA.—THE NUMBER OF TIMES THAT THE DIFFERENT PARTS OF THE EYE WERE INFECTED IN THE 215 SEPTIC CASES OF TABLE I.

Sclerotic	20
Cornea	12
Iris	87
Ciliary body	79
Choroid	68
Retina	28
Detached retina	3
Optic neuritis or atrophy	4
Lens (secondary cataract)	14

TABLE IIA.—THE FREQUENCY WITH WHICH THE DIFFERENT PARTS OF THE EYE WERE AFFECTED BY THE DISEASES IN TABLE II.

Sclerotic	24
Cornea	47
Iris	25
Ciliary body	4
Choroid	35
Retina	36
Optic neuritis or atrophy	12
Lens (secondary cataract)	1

Though it would appear to be obvious, after seeing these tables, that sepsis from pyorrhœa is an important agent in causing inflammation of the eye, nevertheless this view is not yet universally accepted. It seems incomprehensible that a surgeon, whose single aim is to avoid sepsis in his operations, can think it a matter of no importance that a person should have a chronic source of sepsis in any part of his body. The usual objection made is that one sees very bad cases of pyorrhœa in people who say that they are enjoying the best of health and who do not complain. These objectors seem to forget that Nature begins to raise a protecting barrier the very moment that the tissues are irritated by bacterial toxins or are invaded by the bacteria themselves. If the invasion is slow enough, as is the case in pyorrhœa, the barrier is efficient and the individual does not appear to suffer; whereas when the micro-organisms are introduced direct into the circulation there is no time for an efficient barrier to be raised. As long as the patient is in fair health the barrier is capable of doing its work; but this barrier may give way directly the general resistance of the tissues is lowered under the strain produced by some other illness or by traumatism, either surgical or accidental. Then the organisms, or an excess of their toxins, will be able to pass through the defending barrier, and some tissue in the body may become the seat of disease. The enormous number of cases of pyorrhœa that one sees, and the comparatively small number of diseases resulting therefrom, shows how very efficient is Nature's barrier. These people with bad pyorrhœa say, and presumably believe, that they are in the best of health. They are of course living, without knowing it, over a hidden drain which is constantly emitting poison, in such an insidious manner that they are unaware they could possibly feel in better health. When the drain is removed they are astonished at their increased vigour and feeling of rejuvenation.

Although marked and long-standing cases of pyorrhœa are easily recognized and would be condemned as inimical to health by most surgeons, I would like to draw the attention of my colleagues to the importance of recognizing even mild cases of the disease, as they also can cause central choroiditis. Quite recently an ophthalmic surgeon who recognizes that pyorrhœa does cause central choroiditis sent me such a case for my opinion as to the cause. The patient, a youth aged 18, had been reported upon by a dentist, who said nothing about pyorrhœa; the edges of the gums were only just pink, but there were pockets between the molars. Some ophthalmic surgeons examine their own cases for pyorrhœa, but as this is not universal, though it is useful to be able to do so, and at times is necessary, when the patient lives in the country, I have asked my son to take some coloured photographs to demonstrate the method that can be employed by surgeons who do not practise as dentists. It will be noticed that an ordinary 2-mm. platinum loop is in contact with the edge of the gum and cannot be buried out of sight in a healthy mouth, whereas in the case of pyorrhœa the loop has sunk into a pocket out of sight; a second loop is being held on the edge of the tooth above the first to show its size.

Until the medical profession at large recognize the importance of pyorrhœa and the gravity of leaving it untreated, one cannot reasonably expect the dental branch of the profession to do so either, especially as they are being taught to treat the mouth in a way that makes it impossible to avoid creating sepsis, by putting on crowns, and building bars and bridges that cannot be kept clean. Until this policy is reversed and everything is done to enable the mouth to be kept aseptic, the loss of sight, and even total blindness, due to pyorrhœa will continue to occur.

Our greatest hope of reform in the treatment of the mouth lies in imitating the Zulus and some other savages who rinse out their mouths and brush their teeth, with a frayed-out pencil of a special kind of wood, at the end of every meal. The condition of the mouths and teeth of these savages is the ideal for civilized communities; whereas the condition of the mouths and teeth of neighbouring tribes, who do not follow this practice, can be seen any day in our hospital wards and out-patient departments. If this habit of the Zulus were taught in early childhood and practised throughout life, there would be fewer blind eyes and much less ill-health in the world.

I am unable to express an opinion as to the merits of the various kinds of treatment for pyorrhœa that have been mentioned in this discussion, but as long as the protecting barrier is intact, ionization and local measures should be tried. When this barrier has broken down and the eyes are affected by a serious inflammation, which may destroy the sight, it is too late to do anything except to remove the teeth at once. Before the extraction the gums should be carefully cleaned and treated with antiseptics. By doing so the amount of septic material that enters the system through the large raw, unprotected tooth sockets is greatly reduced. Failing this precaution, the eye frequently becomes worse immediately after the extractions. If the sight is to be restored in a recent case of keratitis or of central choroiditis, the affected teeth must come out on the chance that they are the cause, and if the inflammation does not subside then other possible causes must be investigated.

I wish to draw especial attention to the baneful influence that a chronic septic focus frequently exerts upon eyes that have recently been operated upon. The eye often becomes acutely inflamed and remains red for a much longer period than the severity of the operation would warrant, but rapidly quietens down when the exciting cause has been removed. Although no permanent harm usually results, such a complication to the operation as this is to be avoided if possible, and therefore, before I operate on any patient, I always have their mouths examined and put in order if necessary, and have a search made for other septic foci, should there be any indication for so doing.

I have devoted most attention to pyorrhœa because of its frequency in the present day, and consequently its importance to the ophthalmic surgeon. The comparatively small number of my cases attributed to the affections of the alimentary tract shows that either toxæmia from stasis does not affect the eyes so frequently as pyorrhœa, or that the majority of patients with stasis are too ill to trouble about their sight, and when they are cured the eyes have recovered. That the alimentary tract is capable of producing central choroiditis is almost certain, if one may judge by the effect produced by removing the appendix.

A Dominion surgeon brought his young wife for rapid loss of central vision in one eye; with it she could only count fingers. No definite cause for the central choroiditis could be found beyond very slight pyorrhœa, which was very soon cured. Improvement did not take place, and the vision of the second eye began to fail from the same affection. In a week it had fallen from $\frac{5}{5}$ to $\frac{5}{18}$, during which time it had been decided to remove the appendix, which had been acutely inflamed three years before, though now it was pronounced to be quiet. It was also decided to examine the gall-bladder, large bowel, ovaries and uterus at the operation; they were all found to be healthy, and the appendix was free from adhesions, but had a bulbous end, and on being examined showed evidence of past inflammation. Just over three weeks after the operation, which was performed by Mr. Gordon Taylor, the vision of the first affected eye was $\frac{5}{9}$ and that of the second $\frac{5}{5}$. Four months later the vision of each eye was normal.

The following case illustrates the fact that exposure was the exciting cause, and the predisposing cause had been present without producing any effect for more than two years. This cause consisted in a single septic sinus from a root abscess.

A physician in good health went for a day's fishing in mid October, 1908, before beginning his duties as an examiner at the College. When walking home in the evening he felt some discomfort in one eye, and although this got worse he continued for about ten days to read the candidates' papers with the other eye. When he came to me at the end of the examination he had a severe iritis with several adhesions; on being asked where was the source of sepsis he said he had a discharge from a molar tooth, which had existed for more than two years, and had followed an abscess at the root. This tooth was removed within twelve hours, the iritis subsided quickly under atropine and hot bathings, and on the ninth day the eye was white and the vision normal. A week ago he told me he had had no further trouble and it was now the better eye.

This case illustrates the necessity of examining the mouth oneself or obtaining the opinion of someone who knows what pyorrhœa is, in view of the frequent absence of recognition of the disease.

T. V. B., aged 38, was sent to me by Dr. Bullar, of Southampton, who first saw him in January, 1911, with mutton fat K.P. and vision of $\frac{6}{5}$ in each eye. As some teeth were in a bad state Dr. Bullar made him go at once to the dentist and have out all about which there seemed any doubt. In February, 1912, when I saw him he could count fingers at 2 ft. with the right eye, and at 1 ft. with the left eye. The vitreous was too hazy for the condition of the fundus to be seen. The tension was raised and the right field of vision contracted at one part up to the fixation point. I sent him to Mr. Turner, who condemned all the teeth on account of bad pyorrhœa. Soon after they had been removed the vision began to improve, and to-day the right sees $\frac{6}{24}$, Sn. 1·5, and the left $\frac{6}{6}$ and Sn. 0·5. The tension is normal and the vitreous steadily clearing.

This case illustrates the rapid recovery from a long-standing and severe inflammation by removing the teeth, and the recurrence of the inflammation when the gum was damaged by the spicule of bone.

A gentleman, aged 52, who had been sent to Harrogate after six months' treatment for a badly inflamed eye, came to see me at Mr. E. S. Steward's suggestion. I agreed with Mr. Steward that the inflammation was due to sepsis from pyorrhœa and that the treatment of the mouth should be continued. After leaving Harrogate the patient was given an unfavourable opinion as to his chance of regaining any useful sight. He came to me the same day with a badly inflamed eye that could count fingers at 3 ft. only. The sclerotic, cornea, iris, and ciliary body were inflamed and the vitreous hazy. His remaining teeth, which were all very foul and septic, were removed two at a time, a plan insisted upon by his dentist. The inflammation began to subside as the teeth were removed, and two days after the last ones came out the eye was free from injection, and he went home assured by me that the inflammation would not return; but at the end of a fortnight the eye was again very red and at the same time a spicule of bone came through the gum. The loose piece of bone was pulled out, next day the eye was white again, and ten weeks after the first teeth were removed the vision was normal.

This patient had a chronic predisposing cause and the fatigue of manœuvres was a sufficient exciting cause. It was an early case and the recovery was most striking.

A Yeomanry officer, who thought he was in good health, though he had not felt well after breakfast for about a month, whilst out in camp with his troop noticed, two days before, a brown stain on the newspaper; he had read the paper earlier that morning without noticing anything unusual. On covering one eye there was a blur, the size of a shilling, involving the fixation point. He consulted Mr. Frank G. Thomas, of Swansea, who found a small patch of recent central choroiditis, but could not discover a cause, and sent him to me for my opinion. I found the vision of the bad eye to be $\frac{5}{6}$ slowly and Sn. 0·37, the good eye seeing $\frac{5}{3\cdot5}$ and Sn. 0·25. In the affected yellow spot there was a small patch of choroidal disturbance. Five days later he no longer saw the brown mist, the vision had improved to $\frac{5}{5}$ slowly, and he was feeling better. In the interval his mouth had been cleaned by removing three septic roots and treating the pyorrhœa.

A case to illustrate the rapid recovery after treating the cause.

Such a case was that of Mr. McL., a gentleman with one eye blind from secondary glaucoma after dysentery in India many years before. The vision with correcting glasses was $\frac{5}{4}$ and Sn. 0·25 in the other eye, which appeared to be quite healthy, on the two occasions four and a half years apart, when I ordered a change of glasses. At the third visit he complained of seeing a blur near the fixation point; the vision was reduced to letters of $\frac{5}{9}$ and Sn. 0·5 slowly, by a small patch of recent choroiditis, just below the macula. He was edentulous, but his plates were foul. Nothing else was found amiss, no diarrhœa, no remains of dysentery, but his normal looking and smelling urine contained staphylococci, streptococci, and Bacillus coli communis. He was put on a course of autogenous vaccine: within two weeks his sight had improved to letters of $\frac{5}{6}$, the blur went a few days later, and within seven weeks the vision was again $\frac{5}{4}$, Sn. 0·25, and where the patch of choroiditis had been there was some disturbance of the uveal pigment.

This case illustrates the length of time an untreated cause may continue to produce recurring attacks.

Mrs. L. from the age of 12 had many attacks of tonsillitis every August; these have returned at varying intervals for thirty-five years. Her tonsils have been treated and the attacks have ceased. Her first attack of iritis occurred when she was aged 18, and recurred frequently until the tonsils were treated. In the interval she has almost lost the sight from the recurrences.

In conclusion, I regret I have had to travel beyond the strict limits of the matter set down for discussion, but I hope I have made it clear why the ophthalmic surgeon cannot limit his inquiries to the alimentary tract if he is to rid the patient of the inflammation that is affecting the eye. How dependent he is upon the physician, the general surgeon and his confrères, the other specialists, is obvious. As an ophthalmic surgeon I can look forward full of hope to a future when these serious eye affections will cease to occur, because the physician has taught mothers how to feed children properly, the children how to eat properly, and the dental surgeon has impressed upon the population at large the importance of proper mastication and the hygiene of the mouth.

Dr. DAVID SOMMERVILLE: In my remarks to-day I shall confine myself to a brief consideration of one type of alimentary toxæmia with which I have had some acquaintance during the past dozen years. It is known that when certain individuals of middle age or over consume excessive quantities of proteins—i.e., larger quantities than they can assimilate and finally oxidize—a more or less definite set of symptoms appears, characterized by headache, drowsiness, lassitude, and irritability. Such symptoms are accompanied by abnormal physical and chemical changes in the urine, by physical, chemical, and bacteriological changes in the fæces, and by increased arterial tension. The urine of such a patient is more toxic to animals than normal urine; its surface tension is lowered; it contains increased quantities of indol, indol-acetic acid, phenol, skatol, and aromatic oxyacids—all products of intestinal putrefaction. The ratio of ethereal to preformed sulphates is increased. The ratio of ethereal sulphates to total nitrogen is increased. It is known that chlorides, phosphates, sulphates, &c., raise the surface tension of urine; that other bodies, such as urea, sugar, and albumin, act indifferently, whilst aromatic compounds and certain fatty acids depress it. The curve of surface tension runs parallel with the curve of aromatic constituents. The quantity of urine is variable; frequently it is less than normal. The specific gravity is variable; not infrequently it is less than normal. The fæces may be solid and dry or diarrhœic, or alternate between constipation and diarrhœa. The odour may be highly offensive. Undigested proteins, fats, and carbohydrates are present in varying quantities. A carefully prepared 10 per cent. suspension in normal saline examined microscopically by Gram's method yields a field in which the dominant organisms are *Bacillus ærogenes capsulatus* and *Bacillus putrificus*; whereas a corresponding preparation from the fæces of a patient of the same age in perfect health furnishes a field in which the dominant organism is *Bacillus coli*. The chemical reaction is alkaline. Saponification in KOH solution followed by distillation yields phenols which may be fractionally separated and estimated. Phenols may be recovered from the liver of such a patient (should he die suddenly by accident, &c.) by the same method. Is there a causal relationship between the phenols of the fæces of this patient and his clinical condition? I think there is.

It will be objected that the evidence deduced from experiments on animals is not sufficient to show that the indols and other aromatic bodies are toxic. It will be further objected that individuals can be found who excrete large quantities of indols and other aromatic bodies conjugated with sulphuric and glycuronic acids, and yet have no toxic symptoms. These individuals, I reply, completely oxidize their phenols. That the phenols and other aromatic bodies under consideration are produced by the hydrolysis of proteins by anaerobic bacteria must be admitted. These products do not occur in the fæces or urine of the healthy breast-fed infant whose alimentary tract is free from putrefactive anaerobes. They occur only in small quantities in the healthy adult. In adults, in whom they occur in larger quantities and who suffer from toxic symptoms, they can be greatly lessened and made to disappear by substituting carbohydrates for proteins in the diet. The toxic symptoms decrease and disappear at the same time. In this, as in all other problems of metabolism, the most important matter for consideration is not what happens in the alimentary canal, nor what bodies are found in the urine, but what is constantly happening in the intermediate body tissues—blood, liver, cord, brain, &c. This patient suffers because he is unable to oxidize sufficiently the aromatic bodies formed in his large bowel by the action of anaerobic bacteria on proteins. Perhaps the epithelial cells of his intestinal mucosa are damaged and fail in their antitoxic action; or being damaged, they let through more toxic material than the other portions of the oxidizing machinery (hepatic cells, cells of the thyroid and suprarenals, &c.) can deal with. Or it may be that other parts of the oxidizing machinery are at fault. Here I do not distinguish between reducing or synthetic reactions effected in the cells of the intestinal mucosa, in the liver, and elsewhere, and direct oxidation, as such preliminary syntheses and reductions are necessary stages on the way to final oxidation.

The statement that dogs and monkeys have been injected with indols and phenols and have failed to show any symptoms of toxæmia in no way militates against the other statement that such products damage certain men. The oxidizing powers of the dogs for these bodies are greater than those of the men. The dogs can deal efficiently with the toxins; hence no toxæmia. The quantity of aromatic bodies in the fæces furnishes no information concerning the work that is being done by the oxidizing machinery. The latter can only be measured imperfectly by the aromatic sulphates and salts of glycuronic acid in the urine. In cases of intoxication we have no means of accurately determining how much of the phenols, &c., are retained and in what form they are to be found in the different tissues. That some of them can be obtained in phenolic form from the liver has been noted above. In health the oxidized end-products (aromatic sulphates) in the urine go up and down fairly regularly with increase and diminution of putrefactive changes in the large bowel. But in the form of auto-intoxication under considertion these sulphates in the urine do not keep pace with the putrefactive processes in the bowel. On substitution of carbohydrates for proteins in the diet of such cases the ethereal sulphates gradually diminish. That they do not fall suddenly in many cases points to the conclusion that oxidation goes on in the tissues after putrefaction has ceased in the gut. Such inability on the part of the oxidizing machinery to keep pace with the formation of putrefactive products is a definite indication for the reduction of protein in the diet. In the treatment of this form of auto-intoxication animal proteins may be discarded entirely for a time, as they yield larger quantities of phenols than vegetable proteins.

There is a certain amount of analogy between the bio-chemistry of this subject and that of the putrefaction and oxidation of sewage. As the proteins of sewage pass through the various hydrolytic processes on their way to the formation of amino-acids and ammonia ever-changing types of bacteria are encountered. When ammonia is to be oxidized to nitrous acid, new forms of bacteria appear on the scene; and these are superseded by others just as different in their characters when the final formation of nitric acid is to be effected. In like manner the chemical characters of different foodstuffs determine more or less sharply the types of bacteria in different parts of the alimentary tract. The analogy fails somewhat towards the end, as the oxidation of aromatic toxins is effected by the oxidases of tissue cells (intestinal, hepatic, &c.) in the presence of a limited supply of oxygen; whereas the final stages of oxidation in sewage are effected by bacterial enzymes in the presence of excess of atmospheric oxygen. It would appear, accordingly, that the more this limited supply of oxygen in the tissues is increased, and the more the aromatic material to be oxidized diminished in a case of this trouble, the better, as thus alone can one hope for any degree of *completed* oxidation. Whether in certain cases special forms of acid-producing bacteria should be ingested does not seem to be a matter of primary importance, as carbohydrate dietaries will quickly establish their particular bacterial flora.

In conclusion, let me briefly cite one case from my case-book: A busy professional man, aged 55, who worked seven days a week, suffered for two years from the symptoms described in the opening portion of this paper. He was advised to change his dietary and sedentary life. He cut down his proteins, substituting carbohydrates, and took up systematic exercise in the form of golf two days a week. At the end of a year he had recovered his health and the powers for sustained mental work which he had lost. His own statement then was: "I now do more work and of a better type in five days a week than I previously did in seven." He took no drugs; but by change of diet diminished the production of aromatic compounds in his gut; at the same time he improved his oxidizing machinery.

Any practitioner of medicine who will educate himself to do these chemical and bacteriological estimations—they are not difficult, only tedious—and who will do them systematically as controls on his treatment, will come to the conclusion, I think, that this condition which I have so briefly discussed is in essence a failure of oxidation; but a failure that can be largely corrected.

DR. ALFRED C. JORDAN: In the short time allotted me I propose to give examples of some of the results of intestinal stasis when allowed to advance unchecked.

The X-ray appearances of the gastro-intestinal tract in intestinal stasis have been described by me in several recent communications,[1] and I should take them for granted were it not that several critics have challenged my work lately, saying that my results are due to my method, and inferring that normal persons would present the appearances I have described. Those who have seen anything of my work at first hand know that this is not so. There are many, however, who have not yet had opportunities of gaining direct experience of the X-ray investigation of the gastro-intestinal tract. My results, both normal and abnormal, have stood the test, repeatedly, of critical examination in the operating theatre. I see many instances of the normal duodenum daily, and the "static" duodenums can be picked out from among the normals with the greatest certainty. Similarly with the end of the ileum; I have explained repeatedly how an ileal kink is to be diagnosed on fluorescent screen observation, and how a genuine kink due to bands is to be distinguished from an apparent sharp angle due to the manner in which the shadow of the ileal coil falls upon the photographic plate.

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[1] *Proc. Roy. Soc. Med.*, 1911, v (Electro-Therap. Sect.), pp. 9-37; or *Lancet*, 1911, ii, pp. 1824-1828; *Brit. Med. Journ.*, 1912, i, pp. 1225-1229; *Practitioner*, Lond., 1913, xc, pp. 441-454.

Incidentally, I may remark that the fœtal structures described to you the other day by Professor Arthur Keith have no relation whatever to the acquired bands of Mr. Lane. Both are dealt with in the current number of *Surgery, Gynæcology, and Obstetrics* (April, 1913).

My description of the duodenum and my X-ray photographs of the duodenum have been specially favoured with criticism, but it is noteworthy that none of my critics appear to have applied my method; in other words, they have not placed themselves in a position to offer effective criticism. Those who are examining the duodenum in the manner I have described tell me they are confirming my results; this was inevitable, for the observer has no control over the behaviour of the duodenum, except to arrange his method of investigation so as to ensure its proper filling.

I cannot show a skiagram of the normal duodenum for the reason that the bismuth runs through it so rapidly that it does not remain well filled for more than a few moments; I am compelled to be satisfied with a tracing. I wish to make it clear, however, that a thorough examination of the normal duodenum can be made in all cases; the bismuth can be followed through its four parts, and the outline of these parts can be sketched on the glass covering the fluorescent screen. The manner of passing through the duodeno-jejunal junction can be observed in normal as well as in pathological cases.

In severe stasis the duodenum behaves very differently; the bismuth remains in it many minutes, and strong "writhing" contractions occur, but are powerless to overcome the obstruction offered by the kinked duodeno-jejunal junction. It is a simple matter, in such a case, to insert a photographic plate under the fluorescent screen, and allow the exposure of the plate to proceed while observing the behaviour of the duodenum. In the case of the normal duodenum the bismuth has run out of it before the photographic plate can be got into position.

Dr. Hale White said that the duonenum was stated to be dilated without giving its size, or that of the normal duodenum. Such criticism is legitimate, and it is easily answered. I have been measuring the duodenum systematically in my recent cases, taking the length of the second (vertical) part orthodiagraphically, avoiding every source of fallacy, and my figures show that the elongation of this part is very material. The text-book figures for the second part of the duodenum vary somewhat, that usually given being 3 in., while the limits are 2 to 3¼ in. The results of my first morning's measurements were interesting. There were five cases:—

(1) A large woman, whose duodenum behaved normally (she proved to have no stasis), gave 2¾ in. for the length of the second part.

(2) A large man, whose duodenum remained rather full, and showed slight regurgitation, and subsequent evidence of slight stasis in the ileum and large intestine. Length of duodenum, 3¼ in.

(3) A girl, with marked stasis (ileal and large intestinal), whose duodenum was large and long, and showed "writhing" peristalsis. Length of duodenum, 5 in.

(4) A somewhat older woman with well-marked stasis. Length of duodenum, 4¾ in.

(5) A young man with moderate stasis in the ileum, but no appreciable delay in the large intestine. Length of duodenum, 4 in. His duodenum looked large, and remained full of bismuth; it showed strong peristalsis, and there was repeated regurgitation of bismuth from the fourth and third to the second part of the duodenum.

Such measurements are the rule; the normal duodenum measures 2½ to 3¼ in., while the "static" duodenum often measures 4 to 5 in. and more in severe cases. It will be understood that my measurements, being orthodiagraphic, are quite distinct from the apparent length as shown on a photograph or on the fluorescent screen; the orthodiagraphic measurement cannot exceed the actual length of the duodenum, though it may fall short of the true length if the vertical part of the duodenum does not lie accurately in a vertical plane. It is certain that the other parts of the duodenum are elongated too, but exact measurements cannot be given, since they do not lie, even approximately, in the plane of the fluorescent screen.

All my patients are examined in the same way, using the same amount of bismuth and the same method of giving it in all cases; hence my results are strictly comparable. No fact stands out more clearly, in confirmation of the reality of my results, than the constancy with which duodenal and ileal stasis occur together. If I find a "static" duodenum in a patient I am certain to find some form of ileal stasis in the course of my investigation of that patient. The converse is equally true and equally striking: if I find a patient's duodenum normal in size and behaviour I shall not, at the following examinations, find stasis in the lower ileum. I do not find an ileal kink except in patients whose duodenum is distended, and who show clinical evidences of intestinal toxæmia. I have seen the duodenum "writhing" vigorously with nothing in it more opaque than Benger's food; a proof that the weight of the bismuth has nothing to do with the causation of the phenomenon.

In the *Practitioner* for February, 1913,[1] I endeavoured to emphasize this point by actual photographs showing, side by side, the duodenum and the ileum in the same patient. Ten such pairs are given in the illustrations to this paper: they include examples of the three forms of ileal stasis described by Mr. Lane: (1) Simple stasis, (2) stasis with an ileal kink, and (3) stasis due to the appendix obstructing the end of the ileum. The parallel pictures of the duodenum contain examples of all degrees of pathological change in that organ—viz., (1) distension, (2) distension and congestion, (3) ulceration, and (4) cicatrization.

The effects of intestinal stasis on the stomach were described by me in a paper to the Electro-Therapeutical Section of this Society on April 18, 1913. This paper is published in the *Proceedings*[2] (see abstract in the *Lancet*, May 3, 1913, p. 1242.) The duodenal distension leads to pyloric spasm, this is followed by dilatation of the stomach. The weight of the overloaded stomach together with that of the overloaded transverse colon subjects the pylorus to a great strain, and renders it liable to attack by the microbes which have gained entrance to the duodenum from the large intestine. This microbic invasion is a consequence of the stagnation in the lower coils of the ileum. Thus an ulcer may appear at the pylorus. The ulcer leads to great permanent pyloric spasm, and so to increased gastric dilatation. If the liver drops, carrying the pylorus with it, the point of greatest strain is shifted to the lesser curvature, and an ulcer may form there, producing a spasmodic hour-glass constriction, and a very characteristic appearance when examined on the couch with bismuth. The pyloric portion of the stomach is usually very large in these cases, showing that pyloric spasm or stenosis was there before the formation of the ulcer on the lesser curvature. It is not unusual to find two ulcers, one at the pylorus, another on the lesser curvature.

These chronic ulcers tend to become cancerous, and in several cases

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[1] *Practitioner*, 1913, xc, pp. 441-54.

[2] *Proceedings*, 1913, vii (Electr.-Therap. Sect.), pp. 117-38.

I can show the actual transition from simple chronic ulcer to carcinoma of the stomach. Thus we have a further instance of the importance of intestinal stasis in the causation of cancer. The distended duodenum shown on the same picture is evidence of intestinal stasis, and the ileum and large intestine in all cases afford confirmatory evidence of stasis; moreover, the clinical examination of the patients shows them to be suffering from intestinal toxæmia.

In many subjects of intestinal stasis I find marked radiographic evidence of atheroma of the aorta at an unusually early age—another instance of the havoc wrought on the tissues by the intestinal poisons. In Mr. Lane's cases I am never told the results of his clinical examination until my report has been sent in. The close correspondence between his diagnosis and my findings is surely another striking testimony to the accuracy of his observation and the validity of his deductions.

(The paper was illustrated by a number of lantern slides, showing the X-ray appearances of the normal alimentary canal, and the changes in the several parts caused by intestinal stasis.)

Mr. Ernest Clarke: A statement made several times during this discussion forms the text of the remarks I wish to make in adding my small contribution. The statement was that "Intestinal stasis may exist without toxæmia." What proof have we in such cases that toxæmia is absent? Although not generally recognized, the eye, as a matter of fact, is an organ that registers in a very delicate manner certain conditions of the system, and amongst these conditions intestinal toxæmia is markedly one. My object is to try and prove this statement and show that in a large number of cases, although this toxæmia does not apparently exist, according to the ordinary methods of estimating its presence, yet all the time it is present and slowly and gradually effecting irreparable harm to the individual.

In quite early youth the crystalline lens is practically a small bag of semifluid jelly, and accommodation takes place by its being squeezed by the action of the ciliary muscle in such a manner that its antero-posterior diameter is enlarged. So great is the squeezability (if I may use the term) of the lens in the very young, that an accommodation power of 20D. can often be recorded. As age advances a hardening process, or sclerosis, goes on in the lens as in all the other tissues of the body, and so its elasticity becomes less and less, until a point is reached when the near point of accommodation which represents the fullest accommodative power has so far receded that the normal eye requires assistance in the shape of a convex lens in order to see near objects distinctly. This hardening of the lens may be delayed by the absence of, and accelerated by the presence of, certain poisons in the system, and intestinal toxæmia takes a very high place in the list.

I have a record of over 3,000 cases in which the accommodative power has been recorded after all errors of refraction have been corrected. In every case the patient was thus made normal, and in none of them was there any manifest disease such as cataract, iritis, cyclitis, conjunctivitis, &c. Diagram 1 gives the accommodation of 1,600 individuals.

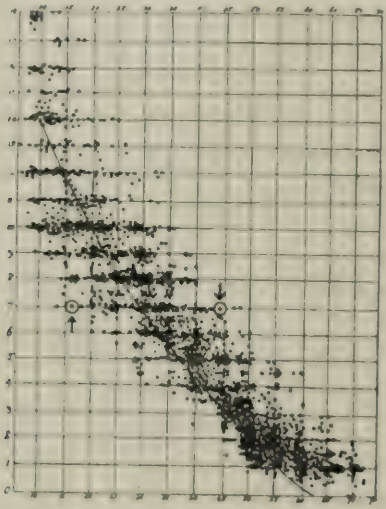

DIAGRAM 1.

The numbers above represent ages, and those on the left the accommodative power expressed in dioptres. In order to ascertain whether the error of refraction, although corrected, had any influence on the accommodation, I made another diagram, consisting entirely of hypermetropes (700) (diagram 2), and a third one consisting of myopes (600) (diagram 3); and the diagram formed by these is practically the same as diagram 1, which included every variety of refraction.

Now it will be observed that although there is a distinct grouping of the cases by which we can obtain a curved line indicating the mean accommodative power at different ages, still, if we take any particular age we notice a very wide range for that particular age. Take the line of age 40 (diagram 1), one individual has only an accommodative power of 2·5D., while another has 8·5D.! What is the difference between these two individuals? In the one aged 40 with only 2·5D. accommodative power the lens has hardened prematurely and become equal to the lens of a man aged 55. That is, he is suffering from premature senility, and in the great majority of cases, in physical appearance, habits and powers he is aged 55. There are many causes which help towards this premature senility, but the factor common to a very large majority of them is intestinal stasis. On the other hand, those whose accommodative power is higher than normal look much younger than their years, and in every way are younger, and on going into their history it will be found invariably that they have taken the greatest care to avoid the least suspicion of intestinal stasis.

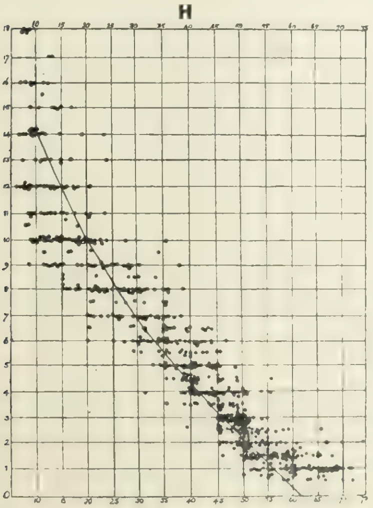

DIAGRAM 2.

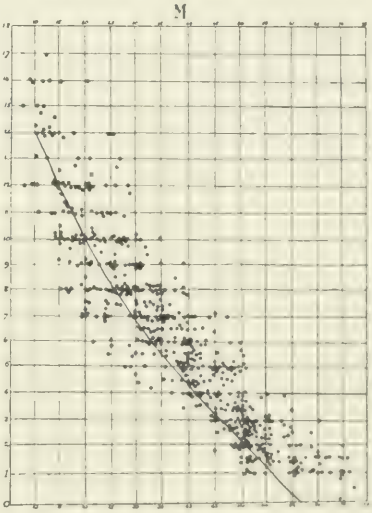

DIAGRAM 3.

These facts, in connexion with the premature senility of the lens, are another proof of the correctness of the old saying that "a man is as old as his arteries": the premature sclerosing of the vessels often taking place at the same time as the process in the lens.

I have given above an example of two individuals of the same age with a wide difference of accommodative power; as an example of two individuals with a wide difference of age and the same accommodative power the following is perhaps more remarkable:—

Two females, one aged 15½, and the other aged 44, had the same accommodative power—viz., 7D. In the girl this is about 4D. worse than normal, and in the woman 2D. better than normal. The girl was too young to show any outward sign of ageing, but she had a most unhealthy appearance and confessed to habitual constipation, and asserted that the bowels were often open only twice a week. On the other hand, the woman aged 44 looked ten years younger and had always paid the most scrupulous attention to the bowels, and for some time past had taken a daily small dose of petroleum oil. (The two cases are indicated in diagram 1 by a ring and an arrow.)

I have notes of another patient, aged 49, with only 2D. of accommodative power, the amount that many men aged 60 have. He had white hair, no teeth, a flabby, dirty tongue, and habitual constipation which he did not trouble to correct.

To show how short a time is required to induce premature senility by intestinal stasis, I cite a man who two years ago, aged then 48, had 5D. accommodative power. In the interval he has suffered very badly with intestinal stasis, which he has very often not troubled to overcome (he had never heard of petroleum oil!). His accommodative power is now only just over 2D.! He has lost more than half his accommodative power in two years. There was no other cause ascertainable except the intestinal one to account for his ageing ten years in two years.

I could cite many other cases where the onset of intestinal stasis was apparently the only symptom or condition which existed to account for the premature sclerosis of the lens, accompanied by premature senility in other parts of the body.

One more case I should like to cite, which bears out incidentally Dr. Hertz's statement that a daily evacuation of the bowels is no proof of the absence of intestinal stasis. A man, aged 48, with an accommodative power below the normal, who had no suspicion that he was suffering from constipation, had appendicitis. Mr. Lane operated and found a very bad state, pointing to many years of habitual stasis. On his recovery he was made to attend most carefully to the bowels, and especially to take lubricants, such as petroleum oil. The result is that now, after some years, his accommodative power is three years above the average, and he looks and is a much younger man than his years. This case also shows that by treatment we can give back a patient some of his lost youth by arresting the senile process in the lens and often in other parts of the body.

I think I have said enough to prove that premature hardening of the lens is a very constant sequela of intestinal stasis, that this is a very useful and easily ascertained index, and that accompanying this process premature senile changes are occurring in other tissues of the body which may often be unrecognizable for some time, and that removal of the intestinal stasis tends to an arrest of the sclerosing processes. An ounce of fact is worth pounds of theory, and I hope these facts will be useful in this important discussion.

I need hardly add that I have enormous faith in petroleum oil as a lubricant in curing stasis.

The causal relation between intestinal toxæmia and various diseases of the eye, notably iritis and cyclitis, more often than not associated with pyorrhœa, has been discussed by others, and I need only add that thirty years' experience in the out-patient department of a large hospital have taught me the paramount importance of obtaining a clean mouth and tongue and emptying the bowels as the first part of the treatment of all eye diseases.

I cannot close these brief remarks without adding my personal conviction that humanity will owe an enormous debt of gratitude to Mr. Arbuthnot Lane for his persistent and obstinate insistence on the paramount importance of effectually treating and removing intestinal stasis.

Dr. FREDERICK LANGMEAD: There is little room in this discussion for dogma. At the best, as Professor Keith has said, "it is but the child of our ignorance." The scientific basis on which it is founded is altogether too meagre to sustain the weight of the superstructure which those who are taking part are building upon it. Yet it must be hoped that the correlation of the views of the different speakers will both lead to a better understanding of this far-reaching condition, and promote investigation into the cause of alimentary toxæmia, a territory at present almost entirely unexplored. There is little which can be said which is more than merely speculative, and perhaps that part of the subject with which I am dealing, the relationship of the thyroid gland to alimentary toxæmia, is as intangible as any.

Evidence is accumulating to show that among the many functions of the thyroid gland, one of the most important is a protective action against circulating toxins. Complete removal of the thyroid leads not only to myxœdema, a condition which may be partly or completely cured by the administration of its internal secretion, but also, as is well known, brings with it a considerable risk of a rapidly fatal toxæmia. An important manifestation of this toxæmia is the symptom-complex known as tetany. On very good grounds, tetany is thought by many to be produced by toxins generated in the alimentary canal. It occurs in gastric dilatation where stagnation is great and disappears if the stomach can be kept washed out. Moreover, in such cases a substance has been isolated from the gastric contents by Bouveret and Dévic, which when injected into animals reproduced the clinical picture of tetany. I have described a series of cases where the same syndrome has accompanied dilatation of the colon. When stagnation in the bowel was accompanied by grey pultaceous and offensive motions, tetany followed and was only relieved by colonic lavation. As is well known, tetany in this country is most common in rickety children, in whom some degree of atony and dilatation of the stomach and intestines is common, and I think most observers will agree that the tetany is almost invariably preceded by an unhealthy condition of the stools.

Tetany is sometimes treated with success by the administration of thyroid extract. We have, then, in tetany a condition which on the one hand is produced by removal of the thyroid gland, and on the other is related to alimentary toxæmia. It seems, therefore, a reasonable inference that the thyroid gland among its antitoxic functions includes that of combating poisons absorbed from the intestinal tract. In cases of excessive absorption of alimentary toxins we should expect some compensatory hyperplasia of the thyroid gland, and it is interesting that in the course of this discussion Mr. Lane, Mr. Rowell, and Mr. Carson, all stated that they have seen ileo-sigmoidostomy followed by shrinking of a goitre.

Another clinical link in the chain of evidence is afforded by rheumatoid (sometimes known as infective) arthritis. The disease is not uncommonly accompanied by enlargement of the thyroid gland, by Graves's disease, and by tetany, or a condition of the hands and feet closely resembling it. It is also regarded by many as an effect of oral sepsis or intestinal intoxication. An important support of this view is the work of Mr. Kenneth Goadby on the subject. Points which favour this contention are the almost invariable presence of oral sepsis, the beneficent effects, sometimes leading to complete cure, which follow attention to the mouth, the improvement after lavage of the colon in some cases, and in a case recorded by Mr. Lane after ileo-sigmoidostomy. In rheumatoid arthritis, then, we have a condition probably in many cases due to alimentary intoxication; in this condition overgrowth of the thyroid is apt to occur, and tetany, a symptom of ineffective functioning of the thyroid gland, is also found. Moreover, rheumatoid arthritis is occasionally distinctly benefited by the administration of thyroid extract.

Rupert Farrant has studied the effects of several types of clinical toxæmia upon the thyroid gland. The following changes occur: First, the colloid becomes granular; secondly, vacuolated and partially absorbed; thirdly, the cells become more numerous, elongated, and arranged in masses; fourthly, the colloid is entirely absorbed and the walls of the vesicles become crenated and folded; and fifthly, the infolding and cell increase go on to transform the vesicles into solid masses of cells. In other words, there is a reaction of the thyroid gland, with signs of hyperplasia and increased functioning, amounting in some cases to changes like those found in Graves's disease. This holds true for such diverse toxæmias as those of infantile diarrhœa, diphtheria, measles and broncho-pneumonia; and whooping-cough and broncho-pneumonia. A similar hyperplasia was caused in guinea-pigs by the injection of diphtheria toxin, but was mitigated if thyroid was given at the same time.

Equally conclusive inferences are to be drawn from the epoch-making researches embodied in the recent Milroy Lectures of my friend Major McCarrison, I.M.S., who has studied goitre as it occurs endemically in the valleys of Chitral and Gilgit in Northern India. He has shown beyond dispute that thyroid enlargement can be produced experimentally in man, within a few weeks, by the administration of suspended matter, separated by filtration from goitre-producing waters; and can be cured by taking thymol, one of the most powerful of gastro-intestinal antiseptics. He has shown that if the source of the toxin be avoided or removed, the glands will return to normal size unless secondary changes have occurred.

It is to be expected that like other glands the thyroid, as the result of over-action, may undergo degenerative and retrogressive changes. Normally at the menopause, with the atrophy of the ovary, part of its work ceases, and it is conceivable that if it has previously been subjected to undue activity, the normal involution may overshoot the mark and lead to hypothyroidism or myxœdema. The last three cases of myxœdema which I have had under observation have all shown, together with severe oral sepsis and very offensive motions, anæmia of the pernicious form which Dr. William Hunter has for so many years ascribed to lesions in the alimentary tract. If the thyroid undergoes enlargement to combat undue toxæmia, it is reasonable to suppose that relieving it of part of its burden may cause a simple goitre to shrink; this, of course, cannot be expected if adenomata or cysts are present.

McCarrison has caused goitres to dwindle or disappear by using vaccines prepared from different forms of organisms—staphylococcus, a coliform bacillus, and a spore-bearing bacillus. The good results which he has obtained have led me to try a similar method of treatment, and I have followed out the plan which he adopted. The vaccine which I have generally employed has been one prepared from the coliform bacilli of the patient's own bowel by Dr. Arthur Davis. The bacterial growth has been obtained from the fæces by culturing on Musgrave's medium, which consists of beef extract 0·5 grm., sodium chloride 0·5 grm., agar 20 grm., tap-water 1,000 c.c., and is of an alkalinity of minus one. This gives a growth of coliform bacilli of fairly constant character. The initial dose has been usually 125 millions of the dead organisms, suspended in about 1 c.c. of saline. Subsequently, if the goitre has shown signs of diminishing, I have increased the dose weekly by 25 or 50 millions. As a rule, the injections have been given at weekly intervals.

It is too early to make definite statements as to the results, but in the few cases, eight in all, treated they have been very encouraging. In one case the goitre disappeared entirely. In all the others it has diminished. As a rule, after the first injection the tumour becomes softer, and, after subsequent doses, gradually gets smaller, although occasionally periods of quiescence supervene. Two of the patients have been able to do up their neckbands for the first time for several months after the first or second injection. A man who had taken to larger collars has now returned to those of his usual size. One girl, who had definite stridor even when lying in bed, has now no embarrassment of breathing except slightly after vigorous exertion. All are charitable enough to say they are better, and free from the symptoms which caused them to come for treatment, such as feeling of suffocation and difficulty in swallowing and breathing.

Another effect has been that by producing absorption of the parenchymatous enlargement, adenomata previously undetected have become obvious, and operative treatment has in this way been facilitated.

To sum up, the thyroid is a gland, a useful function of which is to counteract intoxication. One of the sources of the intoxication is probably the bowel. If the intoxication be proportionately great for the gland, it undergoes hyperplasia and visible enlargement. If the amount of intoxication can be lessened, the extra burden is removed and the gland diminishes. This has been done by the use of thymol and of vaccines, especially those prepared from cultures of the coliform organisms obtained from the patient's own intestinal flora.

Dr. J. M. H. MacLeod: Although a considerable number of cutaneous diseases have been at one time or another attributed to food toxins or to poisonous substances produced in the imperfect digestion of food, in only a few of them is the evidence sufficient to warrant this assumption. The principal cutaneous affections in which a causal connexion with alimentary toxæmia may be established are *urticaria*, the so-called *toxic erythemata*, including the group known since the time of Hebra as erythema multiforme, probably certain cases of lupus erythematosus, *rosacea*, and an *acneiform eruption* which occurs in adolescence, especially on the face in women.

There are observers who have seen in gastro-intestinal derangements resulting from an improper diet a potent factor in the causation of eczema, and others who have blamed excessive meat-eating for causing psoriasis, but their views are not generally accepted. In infants, where the symptom-complex which we at present designate as eczema is particularly common, the number of cases in which the eruption is associated with wrong dietary and digestive disturbances sufficient to cause definite symptoms such as vomiting or diarrhœa is, in my experience, so small that their association cannot be regarded as more than a coincidence, nor have I ever met with a case of eczema in the adult where the primary attack resulted from food poisoning or from some definite gastro-intestinal derangement.

Rosacea is invariably connected with a disturbance of the digestive system, especially of the stomach, and is usually associated with hyperacidity or a coated tongue. It begins as a flushing or transient reflex dilatation of the cutaneous blood-vessels in the central area of the face, the result of the irritating action of food and hot drinks on an inflamed gastric mucosa, and does not come under the category of toxæmia proper. After frequent repetition of the flushings the dilatation becomes permanent and telangiectases are liable to appear.

The cutaneous affections which chiefly concern us in this connexion are the urticarias and the toxic erythemata. The time has long passed since urticaria was regarded as a distinct disease, and more recently it has become recognized that the erythema multiforme of Hebra does not exist as an entity, but that the polymorphic cutaneous lesions grouped under that heading are simply cutaneous reactions, due to a variety of toxic irritants, and often associated with more or less marked visceral disturbances, pains in the joints and muscles, coated tongue and fever, and other evidences of a general toxæmia.

Urticaria.—An attack of urticaria may follow the ingestion of some article of diet, itself definitely poisonous, or it may be caused by some food in itself innocuous but acting harmfully owing to a peculiar susceptibility of the individual or an inflamed state of the gastro-intestinal mucosa. There are certain articles of diet which are apt to cause urticaria in anyone, such as shellfish of the mussel type, crab, sausages, tainted pork, and other sources of alkaloidal ptomaine. In many cases, however, the food which is responsible for the attack is only harmful owing to a peculiar idiosyncrasy of the individual, and there are comparatively few articles of diet towards which someone may not be found to be susceptible. It has been observed frequently in connexion with chronic recurrent urticaria that the primary attack is the result of some gross error of diet; in the hospital class of patients a not infrequent cause being a meal of fried fish. This is followed by an acquired susceptibility by which small quantities of the same food, or some other usually harmless article of diet, which in the first instance would have been powerless to cause an attack, is liable to determine a fresh outbreak, the severity of which may be quite independent of the quantity of the food taken. This has been explained as an anaphylactic phenomenon and it has been suggested that the poisonous food substance calls forth a response on the part of the organism, with the production of antibodies for that special food, and that after a time a second dose even minute in quantity is so rapidly disintegrated by the antibodies that toxic heterogeneous proteids are formed which cause the cutaneous lesions. This susceptibility sometimes occurs in families and would appear to be capable of being transmitted by heredity. The toxin which is responsible for the cutaneous reaction may be derived from the food—as, for example, ptomaines from bad pork, &c.—mytilotoxin, the poisonous substance derived from mussels which is believed to cause the urticaria in mussel poisoning, but as a rule it is some unknown substance, possibly a heterogeneous proteid resulting from the disintegration of the food or some toxic body formed by the cells of the organism. But not only are the toxins which cause the urticarial reaction as a rule unknown, but the manner in which the reaction is produced is also uncertain.

When arsenic is taken in sufficient quantities to cause toxic symptoms, cutaneous lesions of a definite type, consisting of inflammation, pigmentation, and warty thickenings, are liable to occur. These result from the presence of arsenic in the skin and its elimination by the sweat apparatus. In the case of the urticarial reaction there is no definite evidence that the toxin is eliminated in the sweat, and the precise manner in which it causes the wheal to form is open to discussion. There are two rival theories with regard to it—namely: (1) That it is an angio-neurotic phenomenon due to the stimulation of the vasomotor apparatus, either central or peripheral, by the toxin; and (2) that it is an acute inflammatory process. Recent experiments have gone far to support the latter theory, and the view is gaining ground that the wheal is simply an inflammatory disturbance called forth by the action of the toxin on the walls of the cutaneous blood-vessels, or circulating in the blood. It has been proved experimentally by Philippson that a large number of toxic substances such as peptone, morphine, &c., are capable of producing urticaria when injected into the skin of the dog, and that this is independent of nerve action, as these substances have the same effect after division of the sympathetic nerve to the part, or the removal of the sympathetic ganglion.[1] This was corroborated by Török v. Hári, who found that small quantities of toxic substances, ptomaines, &c., such as peptone, trypsin, butyric acid, cadaverine, as well as the toxins from pyogenetic micro-organisms, if introduced in small quantities in the corium of a dog caused typical wheals.[2] It has also been pointed out by Kreibich that the fluid in the wheal is rich in albumin, and more in the nature of an inflammatory exudate than a simple transudation of serum, such as might be expected in an angio-neurotic process. On the other hand, it is somewhat difficult to explain the formation of wheals artificially produced, as in factitious urticaria, on an inflammatory basis alone.

The Group of the "Toxic Erythemata."—In this group are included the polymorphic eruptions collected under the heading of erythema multiforme, which vary so indefinitely in their clinical appearances, some being urticarial, others papular, nodular, vesicular, bullous, or purpuric, but showing a marked tendency to be uniformly distributed on the face, backs of the hands, forearms, and legs, and occasionally on the mucous membranes, and the more generalized eruptions of the morbilliform or scarlatiniform type. These eruptions, like urticaria, are symptomatic of general toxæmia. The exact pathogenesis of the lesions is not fully understood, but it is generally recognized that they result from the presence of some toxin in the cutaneous blood-vessels or acting on their walls. It also remains to be explained why a certain food toxin may give rise to urticaria in one individual, and to an erythemato-bullous eruption in another. As a general rule, it may be said that the ordinary food toxins usually of a proteid nature tend to cause cutaneous reactions of an urticarial or erythematous type, while ptomaines or bacterial toxins from some infective focus are liable to give rise to more severe purpuric lesions. In this group may be included also certain cases of lupus erythematosus of the acute disseminated type in which the cutaneous lesions are associated with a more or less severe general toxæmia; the precise nature of the toxin is unknown, but it may be connected with some gross visceral lesion, such as organic disease of the kidneys or liver.

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[1] *Giorn. Ital. delle Mal. Ven. e della Pelle*, Milano, 1899, fasc. vi, p. 675.

[2] *Arch. f. Derm. u. Syph.*, Wien u. Leipzig, 1903, lxv, p. 21.

Dr. E. VON OFENHEIM: The facts which Mr. Lane has so clearly shown, and which form the well-defined picture of intestinal stasis and toxic absorption, are, in my opinion, beyond discussion, but still open to argument and controversy are the deductions which are drawn from these facts as to causation, consequences, and treatment. Amongst other arguments, it has been put forward in this discussion that the bacteria and not the stasis are the cause of toxæmia. Surely no one has ever contradicted this or maintained anything else; but the question is, what is the effect of the stasis on the bacteria, and, vice versa, the effect of the bacterial toxins on the stasis? That between them exists a vicious circle has repeatedly been proved. Stasis causes bacterial poisons to be formed—these poisons, again, have a paralysing effect on the intestines, and in this way add to the stasis.

Biologically, the internal absorbing surface of the intestine, the villi, corresponds, as Schottelius has shown, to the roots of the plant. That they are placed in the animal body to the inside of its external tegument, whilst in the plant they are externally situated, is only in accordance with the tendency of the animal body to enclose the greatest possible surface development into the smallest compass, whilst in vegetable life, which is chiefly based on a deoxidizing process, the widest possible external surface development is of vital importance for its welfare. If a plant is left for a long time in a flower-pot which is prevented from getting fresh earth and from getting contaminated with other organisms, this plant will, after a time, become ill and die, and even if one adds to the earth fresh, but sterilized, earth it will not effect the recovery of the plant; but if one adds fresh earth which is not sterilized and contains bacteria, or adds these bacteria separately, the plant will recover and thrive. Again, every bacteriologist knows that bacteria which have grown for a long time in the same medium change their appearance and undergo what is technically termed "involution," and after some time die off altogether, even if fresh broth, or whatever the medium is, is added to the old one. To keep organisms alive it is therefore essential to transplant them from time to time (the time varying, of course, according to the kind of organism). These two observations had to be mentioned to explain the facts which I am now going to put before you.

It is remarkable that the majority of the investigations on the intestinal flora of the constipated were carried out in the hope of finding some special species of microbes causing the symptoms of intoxication. Metchnikoff conducted his researches on those lines and was induced to try to increase the number of what he considered the "beneficial" organisms by administering certain strains of particularly resistant lactic acid organisms. What, of course, was overlooked was that it is an easy matter to introduce organisms into the intestines, but that it is by no means easy to acclimatize them, to make them grow under circumstances which are unfavourable for their species.

Other bacteriologists, again, attribute the pathological symptoms to an overwhelming amount of organisms, and suggest reducing their amount by disinfectants and cleansing measures, which are applied to the first part of the digestive tract—i.e., the mouth—as if any amount of cleaning and disinfecting could reduce the number of bacteria in a cavity of the living body.

Comparing the intestinal bacterial flora of a patient who is suffering from stasis with that of a healthy person, the striking difference between the two is not a *larger* amount of bacteria in the constipated, but a *smaller* one. The difference in the species in both cases is a very unimportant one, and sometimes one finds an exactly identical bacterial flora in constipated as in healthy individuals. But another feature is most marked, and that is that in the fæces of the constipated one finds countless involution forms, polar-staining organisms, and all kinds of mis-shapes, which show that the organisms present are living under conditions which are unfavourable for themselves, and which seem to cause them to undergo such changes as we find in old and overgrown cultures. We find that by cultivating emulsions made from the fæces of constipated persons only about one-fifteenth to one-twentieth of the number of colonies grow compared with those of a normal non-constipated individual. Dr. Distaso has, by different methods, found the same phenomenon. It seems that these organisms are undergoing the same changes in the intestinal tract of the constipated as we find in old stale cultures—that is to say, in cultures which have been overloaded with bacterial products by which the organisms themselves have gradually been damaged or killed off. Again, from cultural experiments we know that all these organisms do not give off their actual toxins at once, but that only after they have been damaged or destroyed their endotoxins are liberated into the surrounding medium. Only by the destruction of bacteria are those endotoxins liberated which are the cause of the toxæmic symptoms. As a matter of fact, Dr. Distaso has lately succeeded by some very ingenious experiments in producing a picture of constipation—at least the bacteriological picture—*in vitro;* that is to say, he transformed the bacterial flora of a normal non-constipated individual in his cultures into the flora which we find in the constipated. He hopes to publish his most remarkable results before long.

To avoid the mistake of dealing with the subject from a one-sided point of view, it is essential also in the investigation of toxæmia not to lose sight of the clinical aspect and to see how far the clinical symptoms can be explained by the bacteriological findings. A good deal has been written lately on disease carriers, that is to say, on the possibility of certain persons carrying pathogenic organisms in their bodies without deriving any harm from them and being able to transmit them to other healthy persons in whom the pathological symptoms develop. It is a well-known fact that typhoid, cholera, and dysentery bacteria can pass through the intestinal tract without doing any damage to their host, so long as these organisms are not broken up by lysis or autolysis. As a matter of fact, the bacteria found in the fæces of these carriers are always quite typical and show no signs of degeneration, as they do in those people who actually show the disease. It is only by the influence of antibodies, chiefly lysins, or by autolysins produced by the organisms themselves, that their bodies are broken up, their endotoxins set free, and the pathogenic condition produced. In other words, in those persons whose tissues have receptors for certain organisms the disease will develop; whilst others who have no such receptors, and therefore no influence on the organism, will remain healthy in spite of their infection. It may sound a paradox, but it actually comes to this, that *the presence of immune bodies is the cause of the disease.*

If we compare the clinical symptoms of these acute infections, as cholera, dysentery, &c., with the chronic condition of auto-intoxication, we must admit that the symptoms are practically identical, and that there is only a difference of quantity and not one of quality. Cold, clammy extremities, general malaise, sickness, furred tongue, foul breath, pigmentation, and erythemata; and last but not least, the effects on the central nervous system, showing themselves in the acute forms, as delirium and coma—in chronic toxæmia, as neurotic excitability and undue tiredness.

Assuming that our conclusions are correct, the therapy will resolve itself not into the question of fighting the one or the other species of organisms but in the problem of either *preventing* the *destruction* of organisms and their autolysis, or shortening the time during which the bacteria remain in the intestinal tract. The latter method is the one which has been so admirably adopted by Mr. Lane, and the wonderful successes he has obtained, many of which I have been fortunate enough to have had the opportunity of watching myself, are due to the fact that he shortened the passage of the fæcal matter through the intestinal tract, and particularly that he excluded the inert large intestine which acts as an incubator in the abdomen and provides the most favourable conditions possible for an overgrowth and, in consequence, for the autolysis of the bacteria.

In spite of the remarkable results obtained by this operation it is scarcely a method which can be employed in all cases, as nowadays practically ninety out of one hundred individuals in civilized countries are suffering from intestinal stasis, and it will more or less remain an ultima ratio to be reserved for extreme cases. One must therefore search for other means by which the breaking up of the organisms can be prevented and the deleterious effects of their endotoxins avoided, amongst which the most important one, perhaps, is the increase of the stasis. Experiments are in progress in that direction at the present moment, but it is yet too early to express any opinion as to their success or failure.

If we compare the clinical appearances with the results of bacteriological investigation and the pictures presenting themselves in the post-mortem room, there cannot be any doubt that the symptoms found and described by Mr. Lane are due to the absorption of toxins from the intestines; and whatever the treatment of it may be in the future, at the present moment there is no other way of dealing radically with the more severe cases than by an operation, by which the time during which the organisms remain in the intestines is shortened.

Mr. S. H. Browning: The difficulty of proving that disease of one of the special organs is due to alimentary toxæmia is very great, and we are reduced to the unsatisfactory procedure of making our diagnosis on the result of treatment. The only reason we have at the present time for stating that eye diseases are the result of alimentary toxæmia is that certain eye conditions improve or are cured when the diseased condition of the alimentary tract is improved or cured.

In our present state of knowledge the disease which gives rise to the most eye trouble is pyorrhœa alveolaris, and it is now recognized, thanks to the insistence of Mr. Lang, that this condition is quite a common cause of iritis and irido-cyclitis as well as of retinitis and choroiditis. When one considers the amount of pyorrhœa in the mouths of hospital patients it is surprising that such a small percentage suffer from eye disease, and I would like to suggest that the organism causing the pyorrhœa may have something to do with the association of pyorrhœa and eye disease. On looking through my cases of pyorrhœa associated with eye disease I have been impressed by the frequency of pneumococcal infections, which according to Eyre is the least common of the organisms found in pyorrhœa with the exception of the *Staphylococcus aureus*. The susceptibility of the eye to pneumococcal infections may perhaps account for the fact that pyorrhœa causing eye disease is more often associated with pneumococcal infection than pyorrhœa not causing eye disease. Treatment lies chiefly with the dentist, though the use of vaccines will considerably accelerate the cure, and will always alleviate the eye condition more rapidly than will treatment by the dentist alone.

A point that I would like to draw your attention to is the frequent association of asthenopia with pyorrhœa, and since I have been asking patients whom I have been vaccinating for pyorrhœa about this symptom I have come across quite a large number suffering from more or less severe asthenopia, and improvement has been most marked as the mouth has been got clean.

The next source of infection that I wish to mention is the tonsils, and in this connexion the eye condition has always been irido-cyclitis in the cases that I have seen. A case that Mr. Lawson sent me about two years ago is of interest. A married woman, aged about 25, was suffering from an acute attack of irido-cyclitis on a chronic irido-cyclitis, and as there was no obvious cause Mr. Lawson sent her to me for further examination. I examined her fæces and urine, her teeth, and the various nasal sinuses, as well as her ears, without finding anything abnormal;

she had no leucorrhœa. I did find, however, that she had enlarged and very septic tonsils, and quite a lot of pus and cheesy matter could be squeezed from them. I made a cultivation and found the pneumococcus and *Micrococcus catarrhalis*. A vaccine was made, but I suggested that if the tonsils were enucleated that that would probably be sufficient. The tonsils were removed, and Mr. Lawson tells me that there has been no recurrence of the eye trouble.

Suspecting that intestinal stasis and other pathological conditions of the bowel, and especially of the large bowel, might be the cause of eye disease, it has been my practice for some time past to examine the fæces and urine of all patients suffering from irido-cyclitis and other eye diseases of obscure origin, to see if there was any abnormality in the bacterial content. In a number of cases I have found this to be the case, and the bacteria have been abnormal either in total numbers or in relative numbers of the various organisms to the normal. Of course, the finding of abnormal bacterial contents of the fæces does not prove that the eye disease is due to alimentary toxæmia, and in the majority of the cases the result of treating the intestinal condition has been disappointing, but the results of some of them have been so satisfactory as to make the routine examination of the fæces in difficult cases desirable.

Some time ago I saw with Mr. Worth a young girl with severe irido-cyclitis and abundant keratitis profunda in whom the Wassermann and tuberculin reactions were negative; her teeth were good, her urine normal, and she had no leucorrhœa. Examination of her fæces gave a nearly pure culture of pneumococcus. A vaccine was prepared and the patient treated with it. The improvement was immediate and the patient is now quite well. The girl was constipated and suitable treatment was carried out at the same time.

Another case was that of a man who was invalided home from China with double irido-cyclitis, the right eye being almost useless. He had been under treatment for some long time without benefit. I examined the man for every source of infection and found that he had in his fæces a non-lactose-fermenting organism which was agglutinated by his blood serum in high dilution, and which proved to be a type of Flexner's dysentery bacillus. The man was reported to have had amœbic dysentery about two years previously while in China. After the seventh injection of a vaccine made from this organism his doctor wrote to me that his left eye was now quite well, and that the joint trouble that he was suffering from was also much improved. This was two years ago, but Mr. Lawson, whose case it was, tells me to-day that the patient had a relapse a few months ago.

Bacterial infection of the bladder is now taking its place as one of the causes of eye disease of a toxic nature, and I have seen several cases where the eye disease has improved with the treatment of the cystitis by means of vaccines.

I believe that these eye conditions are toxic in origin and are not due to the presence of any organism in the eye, and I have examined the fluid from the anterior chamber of the eye in many cases without finding any organism.

The Royal Society of Medicine.

May 7, 1913.

Sir Francis H. Champneys, Bt., President, in the Chair.

A DISCUSSION ON ALIMENTARY TOXÆMIA; ITS SOURCES, CONSEQUENCES, AND TREATMENT.[1]

Dr. H. D. Rolleston: As the uninjured skin is comparatively impervious, the other surface covering of the body or the mucous membranes constitutes the main inlet for microbes and poisons; and of the various mucous tracts, that of the alimentary is much the largest, and further, is much the most exposed to infection and intoxication. It is true that the exposed condition of the alimentary canal is specially protected by the acid gastric juice against microbic activity and by the liver against poisons, but these lines of defence may be overwhelmed and broken down. It is easy to argue that a very large number of the diseases to which flesh is heir are due to intestinal toxæmia, but the state of our present knowledge makes caution necessary. Still, although it may be difficult to prove the widespread influence of alimentary toxæmia, it is unwise to put aside possibilities until they can be definitely ruled out.

A difficulty about a wide assumption as to the existence of alimentary toxæmia in the sense in which it is employed in this discussion—namely, effects in other parts of the body due to poisons apart from micro-organisms—is that many of the effects assumed to be toxic may be due to the passage of micro-organisms in small numbers into the circulation and so into the tissues. The hypothesis of such an escape of micro-organisms has long been before us in Adami's sub-infection with *Bacillus coli*, which he suggests plays some part in the production of cirrhosis, hæmochromatosis, and pernicious anæmia. With the improvements in our methods of blood culture, bacteriæmia is now more commonly recognized, and in the future transient and comparatively non-virulent forms may be found to be more common than is generally realized. It is quite reasonable to believe that toxæmia and bacteriæmia of intestinal origin may occur simultaneously; this is illustrated by the frequent association of chronic pancreatitis with portal cirrhosis, for chronic pancreatitis is most readily explained as due to an ascending infection from the intestine, whereas hepatic cirrhosis appears to be the result of poisons absorbed from the portal system.

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[1] Adjourned from May 5.

Another point to be borne in mind in this discussion is that the same result—for example, nephritis, neuritis, or arthritis—may be brought about in different ways—by different forms of toxæmia, of which alimentary toxæmia is one. Therefore, in considering the influence of alimentary toxæmia it is not necessary to argue that it is the exclusive cause of certain conditions: to take an extreme instance, we need not join Mr. Arbuthnot Lane[1] in the *obiter dictum* that gynæcologists are solely due to intestinal stasis; and further the title of alimentary toxæmia to ætiological importance in a morbid condition is not necessarily rendered invalid because in some instances other factors are proved to exert a causal influence. Thus, exogenous oxaluria, due to the consumption of rhubarb, is not disproved because oxaluria may also result from metabolic disturbances which eventually result in diabetes.

The large subject of alimentary toxæmia may be divided in the first place into the results due to (1) alterations in the quality and quantity of the foodstuffs introduced into the intestine; (2) the activity of micro-organisms in the intestine.

ALTERATIONS IN THE QUALITY AND QUANTITY OF THE FOODSTUFFS.

To refer in the first place to idiosyncrasies to certain foods, it is true that the hypersensitiveness may depend on changes in the blood and tissues, the intestine being merely the passive transmitter; but on the other hand, the digestive processes may be congenitally so modified or the circumstances so altered by a change of the intestinal flora as to produce poisons out of ordinary food—e.g., an abnormal cleavage of proteins and other bodies. Thus, family periodic paralysis might conceivably be due to a congenital metabolic defect, which in the presence of unsuitable food gives rise to the formation of poisons in the intestine (Gardner[1]). This subject has, I find, been discussed by Dr. Vaughan Harley,[2] who suggests that the idiosyncrasy of some persons to eggs, which is now commonly quoted as an example of anaphylaxis, is due to the cleavage of lecithin into cholin, which may eventually yield the poisonous body neurin. In this connexion I may mention that a medical man with neurasthenic symptoms and a belief during the morning that he was ruined, recovered after he left off taking an egg for breakfast.

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[1] *Vide* Discussion (part i), p. 101.

Apart from any active toxic properties, alteration in the quality and quantity of the foodstuffs may so modify metabolism that morbid changes and symptoms are produced. Our knowledge of "the nutritional importance of the presence in dietaries of minute amounts of certain accessory substances"[3] which may possibly activate metabolism is only in its infancy. It has recently been shown that tropical beri-beri is due to feeding with polished rice, which is deficient in a nitrogenous substance, small amounts of which are essential for the metabolism of the nervous system, and that beri-beri, thus experimentally produced, can be cured by supplying the aqueous extract of rice polishings. Scurvy and infantile scurvy are also due to an analogous deficiency in the food; and it has been shown that dietaries composed of the proteins, carbohydrates, fats, and salts capable of keeping adult rats in good health entirely fail to promote growth in young rats, although they remain healthy. The addition to the diet of the milk from which the protein had been eliminated was followed by normal growth (Osborne and Mendel). These experimental results justify hesitation in employing for lengthy periods artificial milks made by the combination of the constituents of milk in appropriate proportions (percentage system) for infants who cannot take ordinary milk. To turn to the more commonplace results of excessive quantities of food, an excess in the intake of proteins is commonly regarded as one of the causes of arterio-sclerosis by raising the blood-pressure either (1) through toxic constriction of the peripheral field, or (2)—and this hypothesis will be referred to later—by primarily acting on the suprarenals or the thyroid. It has recently been suggested that an excessive protein diet is specially prone to produce change in the gastro-intestinal vessels, so causing angina abdominis.

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[1] Gardner, *Brain*, Lond., 1913, xxxv, p. 243.

[2] *Vide* Discussion (part i), p. 33.

[3] *Vide* review of the subject by A. E. Cooper, *Brit. Med. Journ.*, 1913, i, p. 722, which is fully utilized here.

In rare instances common salt in excessive quantities has produced œdema in healthy persons, just as it is known to do in nephritis. Rodriguez[1] has recently argued that an excessive intake of salt may, by its influence on metabolism, cause some cases of diabetes. An excess of carbohydrate food favours fermentation, and in children may cause fever. How far an habitual excess of proteins or carbohydrates can, by wearing out the assimilative powers of the tissues, lead to the metabolic disorders underlying gout and diabetes is a question which is perhaps hardly within the limits of a discussion on alimentary toxæmia.

RESULTS DUE TO TOXÆMIA DEPENDING ON THE ACTIVITY OF MICRO-ORGANISMS IN THE INTESTINE.

In connexion with this, which is the most obvious aspect of the subject, I shall only refer to some rather debatable problems. The condition of intestinal infantilism due to chronic diarrhœa might be explained merely as the result of malnutrition. Herter and Kendall, however, have found that the condition is associated with large numbers of a micro-organism—*Bacillus infantilis*—in the stools. The suggestion that toxæmia of intestinal origin may produce widespread effects, such as neurasthenia and liability to other infections, is in harmony with views put forward by Mr. Lane and others. It is an attractive hypothesis that changes in the ductless glands, either in the direction of proliferative reaction, as in exophthalmic goitre, or of degeneration, as in myxœdema and pluri-glandular insufficiency, may in certain cases be due to toxins absorbed from the intestine. Personally, I quite anticipate that proof will eventually be forthcoming. There are some points compatible with the view that exophthalmic goitre may in some instances be the result of alimentary toxæmia. Digestive disturbances are very frequent, a protein diet usually aggravates the symptoms, indicanuria has in my experience been common. The evidence recently brought by McCarrison[2] to prove that endemic goitre is due to alimentary toxæmia may well suggest that some other form of intestinal toxæmia may induce exophthalmic goitre. In both these diseases the blood shows a relative lymphocytosis, a change which may be due to intestinal disturbance.

The relation between synovitis and other forms of arthritis on the one hand, and the alimentary canal on the other hand, provides scope for considerable discussion. A. E. Garrod[1] argues that the transient joint manifestations accompanying erythemas, purpura, and urticaria are, like the cutaneous changes, toxic. The influence of alimentary toxæmia in the production of skin lesions has been fully discussed, and it does not seem unreasonable to suppose that transient attacks of synovitis may be similarly explained. The synovitis of acute rheumatism is so transient, and leaves so little or no change, that in the presence of the conflicting accounts as to the sterile or infected condition of the joints it may be toxic, and due to the products of streptococci in the alimentary canal. The condition of chronic rheumatoid arthritis may be secondary to focal infection in various parts of the alimentary canal, and pending more convincing evidence that micro-organisms are constantly present in the articular and peri-articular tissues, the lesions may be referred to here. Pemberton's[2] preliminary report on the metabolism of such cases when treated by colon lavage and a restricted diet, at first consisting mainly of buttermilk, is of considerable interest from several points of view. Cases which improve on this treatment may show an increased output of fæcal bacteria and of ethereal sulphates in the urine. The increase in the fæcal bacteria was thought to be due to bacilli of the lactic acid type. The increase in the ethereal sulphates is puzzling, but as it coincided with improvement it suggests that intestinal putrefaction of proteins is not an important factor in the disease, in which indeed Pemberton believes that carbohydrates play an important part. The retraction of the head and other symptoms of meningism, sometimes seen in cases of severe infective diarrhœa in young children, appear to be due to the action of poisons absorbed from the alimentary canal. A form of intestinal toxæmia in the newborn, due to bacterial infection of the meconium within the first forty-eight hours, has recently been described by Morse.[3] Incomplete evacuation favours absorption of putrefactive products from the meconium. The infant rather suddenly becomes seriously ill about the third to the fifth day, has moderate fever, and commonly cyanosis, twitching, general rigidity, and even convulsions. If the bowels are thoroughly opened, all food stopped for a time, and water freely given, recovery is usually rapid and complete.

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[1] Rodriguez, *Rev. de Méd.*, Par., 1913, xxxiii, p. 136.

[2] McCarrison, "The Etiology of Endemic Goitre," 1913.

The Alimentary Origin of Blood Diseases.—That the intestinal tract is primarily at fault in enterogenous cyanosis seems clear;

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[1] Garrod, A. E., *Lancet*, 1911, i, p. 1411.

[2] Pemberton, *Amer. Journ. Med. Sc.*, Philad., 1912, cxliv, p. 474.

[3] Morse, *Amer. Journ. Dis. Child.*, Chicago, 1912, x, p. 229.

but whether or not it is invariably a toxæmia only is not established. For in G. A. Gibson and Douglas's case, which was described as "microbic cyanosis," *Bacillus coli* was found in the blood when the cyanosis was well marked. In bothriocephalus anæmia, and in some cases of pernicious anæmia, poisons absorbed from the alimentary canal can be reasonably invoked. Clinically it is difficult to avoid the conclusion that in young children intestinal toxæmia is sometimes the cause of anæmia; but whether by hæmolysis or by inhibiting the formation of red blood corpuscles there is not sufficient evidence to show. In leukæmia and polycythæmia, in which there is violent stimulation of the leucoblastic and erythroblastic elements respectively of the bone marrow, the cause is unknown, but alimentary toxæmia has been suggested in polycythæmia, and by parity of reasoning might be invoked in leukæmia. It is most dangerous to argue from isolated cases, but J. H. Drysdale's remarkable case of acute myeloid leukæmia, in which a cure for two years followed administration of naphthalene tetrachloride given with this view, has a bearing on this point; and I have seen improvement in cases of leukæmia treated on these lines. Needless to say, great caution must be maintained with regard to the influence of alimentary toxæmia. Years ago Sir Andrew Clark urged that chlorosis was a copræmia due to constipation, but the absence of any evidence of excessive putrefaction in the intestine and of hæmolysis in the tissues has rendered this untenable. Again, the view that hæmochromatosis is the result of chronic but widespread hæmolysis, probably of intestinal origin, has recently been challenged on the ground that examination of the blood fails to show anæmia, hæmolysins, or evidence of hæmolysis, and it has been suggested that the disease is a metabolic disorder causing pigmentary degeneration of the parenchymatous cells (Sprunt).[1] As hæmolytic jaundice now seems to be established as a definite disease, the absence of jaundice in hæmochromatosis is an argument against the view that hæmolysis is the underlying factor.

INTESTINAL TOXÆMIA AND METABOLIC DISORDERS.

So far the results of alimentary toxæmia referred to have been those directly due to the action of poisons absorbed from the alimentary canal, for example, hepatic cirrhosis. But they may be indirect only, or depend on disturbances of metabolism originally caused by poisons derived from the alimentary tract. The part played by intestinal toxæmia in the production of metabolic diseases such as gout and diabetes is difficult to estimate accurately. While any bald statement that they are direct manifestations of alimentary toxæmia would be an exaggeration rightly calling for criticism; it would be equally unwise to assert that they are entirely independent of it, for it is obvious that when these disorders of metabolism are established their progress is intimately related to variations in the dietary. In some instances, especially in gout, the metabolic disorders appear to be hereditary, in other cases the derangement is acquired and in these instances, or in some of them, errors in the quantity of food or in its digestion may, when persistent for long periods, play a causal part. The methods by which intestinal toxæmia may disturb metabolism are various, and as illustrating the complexity of the subject, Woolley and Newburgh's[1] explanation of the ætiology of diabetes may be quoted; as they found that the injection of indol produced hypertrophy of the medulla of the suprarenals, they suggest that such hypertrophy, if lasting for a considerable time, may, by causing a change in the chromaffin-pancreas equilibrium, set up diabetes. That there is an inter-relation between the pancreas and adrenals is shown experimentally by (1) Pemberton and Sweet's[2] observations, that intravenous injections of adrenin inhibit the flow of pancreatic juice, that removal of the adrenals induces a flow of pancreatic juice which can be inhibited by injections of adrenin, and (2) the production of glycosuria by painting the pancreas with adrenin, and also, though not so certainly, by its injection into the circulation. Reference may be made here to the possibility that changes in the suprarenals due to alimentary toxæmia may be instrumental in the production of arterio-sclerosis. It has long been an article of faith that continued indiscretions in diet and over-eating cause chronic arterial and renal disease; more recently it has been noted that the suprarenals are large in granular kidney and arterio-sclerosis, and it might again be suggested that indol and other bodies absorbed from the intestine cause hypertrophy of the suprarenal medulla, and so high blood-pressure, and ultimately arterio-sclerosis and granular kidneys. Metchnikoff, as the result of the experimental administration of paracresol, phenol, and indol, products of the activity of *Bacillus coli*, induced arterial changes. Schur and Weisel stated that they identified adrenin in the blood serum of patients with chronic nephritis by means of the Meltzer-Ehrmann frog pupil reaction, but other observers, and quite recently Janeway and Park[1] have failed to find any evidence of adrenin or other pressor substances in the blood of persons with high blood-pressure. This suggestion as to the mechanism by which alimentary toxæmia causes arterio-sclerosis, is tempting, but as yet non-proven.

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[1] Sprunt, *Arch. Int. Med.*, Chicago, 1911, viii, p. 75; *Journ. Exper. Med.*, New York, 1912, xvi, p. 607.

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[1] Woolley and Newburgh, *Journ. Amer. Med. Assoc.*, Chicago, 1911, lvi, p. 1796.

[2] Pemberton and Sweet, *Arch. Int. Med.*, Chicago, 1912, x, p. 169.

Another remote way in which alimentary toxæmia may conceivably set up arterio-sclerosis is conceivable—namely, by inducing thyroid insufficiency. If the normal blood-pressure is maintained by a balance between the pressor and the depressor internal secretions, and the activity of the thyroid, which is the chief depressor ductless gland, be diminished, the blood-pressure should rise, and as a result of this arterio-sclerosis would eventually follow.

Dr. H. DOUGLAS WILSON: The study of alimentary toxæmia is a subject fraught with difficulty at every turn, so complex and of such divergent nature are the problems presented for examination. Its effects are so varying, and differ so widely in different individuals, that it leads one to the opinion that many ætiological factors, especially as to the nature of the offending toxin, must be at work.

It is to the French school and to Bouchard in particular that we owe much for originating lines of thought, investigation and treatment with regard to toxic absorption from the intestinal tract and its allied affections. While Continental and American writers continually refer to it, English medical literature on the subject is comparatively meagre, although, thanks largely to the stimulating influence of Mr. Arbuthnot Lane, it would appear to be about to receive the adequate and widespread attention which is certainly its due.

Of the alimentary tract as a whole, one would naturally suppose that the part most likely to produce toxic effects of more than a passing nature, such as might be described as chronic constitutional disturbance, is the lower digestive apparatus, and the colon in particular. Here are prone to occur conditions such as intestinal stasis which encourage the growth and propagation of flora, the effects of which may well give rise to many symptoms.

What I have to say is based on the results of observation and treatment of some 800 cases, in which I believed the colon was the seat of toxic absorption, and which have passed through my hands at Harrogate during the past nine years. Practically all were treated by colon irrigations of mineral water in combination with other spa methods.

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[1] Janeway and Park, *Journ. Exper. Med.*, New York, 1912, xvi, p. 541.

Many theories have been advanced as to its ætiology, the discussion of which does not come within the scope of this paper. It may be said, however, that several factors seem to contribute to its cause. Both sexes seem equally liable to its development, but the majority of cases seen by the writer have been females, in the proportion of 43 per cent. males to 57 per cent. females. There is no doubt it tends to run in families, and frequently one has seen it in two and sometimes three generations.

Sources of infection, such as bad teeth, or septic discharge from the post-nasal region or tonsils, are apt to develop and aggravate the condition. The appendix, too, may be a factor which must be considered, as it may be a source of infection or reinfection to the large bowel. One has seen this demonstrated in several cases which failed to maintain improvement from treatment until after its removal.

The age at which chronic intestinal auto-intoxication is most likely to occur is somewhat difficult to ascertain, for frequently its onset is very insidious, and it may have existed for a considerable time before the patient seeks advice. One has noticed, however, that patients often begin to show troublesome manifestations about the age of 40, just when the wear and tear of life is beginning to tell. In my series of cases the average at which the patients came under my care has worked out at 43 years for males and 44 years for females.

There can be no question that constipation, or at all events inefficient evacuation of the large bowel, is present in the vast majority of cases, though not in all. Frequently the medical attendant is apt to be misled in this direction by the patient's statement that a daily action is obtained without the necessity of resorting to an aperient, and there too often the matter is allowed to rest. Or an aperient may be ordered with which the physician is satisfied on the patient's statement that it has produced a good result, while all the time the bowel may be only partially clearing itself.

All one's experience goes to show that in these very cases where efficient clearance is necessary and essential for the alleviation of symptoms it is most difficult to obtain by ordinary laxatives. This is frequently easy enough to prove by administering colon irrigations after a previous course of laxatives, when they often show old hard scybala, mucus, and retained fæces. The explanation seems to lie in the fact that very many of these subjects have a spastic state of the sigmoid which is made more irritable by laxatives, and it seems to stop further evacuation after a certain amount of material has been allowed to pass.

Another symptom common to cases of chronic auto-intoxication is the slack state of the abdominal walls. This may exist to a marked degree in patients otherwise of excellent muscular condition and development, and its disappearance is synchronous with improvement in the state of the bowel and the general condition of the sufferer. Here, I believe, may be the starting point of the many stages of visceral ptosis into which patients in most instances should not be allowed to drift if taken in hand at a sufficiently early stage and appropriately treated.

No doubt there seems to be an intimate association between colon toxæmia and floating kidney. The former, in addition to its malign influence on abdominal muscles, seems to have ill-effects on the supporting structures of the internal organs, and the symptoms which have by some writers been ascribed to undue renal mobility might, more often than not, be said to be toxic in origin. As already noted, it is extremely common to find the sigmoid spasmodically contracted and very easily palpable.

The effect of toxic absorption from the lower alimentary canal on the rest of the digestive apparatus may be profound. Functions of the stomach, liver, and pancreas may be much disturbed. Various forms of dyspepsia seem to be secondary to it, and prove most intractable as to treatment unless the originating cause is first attended to. It is no unusual thing for these patients to pass pale stools, the want of colour being due to a deficient amount of bile, or to undigested fat. If the former exists cholagogues have little effect, and help is best and most quickly obtained by colon irrigations. The stools of those suffering from lower bowel toxæmia frequently show mucus in excess of the normal, but this is by no means a constant factor. It may vary greatly in amount and character, even in the same individual, and is often only revealed in colon washings. Certain types of patient state that they feel better in themselves when they note its presence, and its apparent disappearance may be accompanied by a recrudescence of symptoms.

The nervous system is particularly prone to be affected in some way by toxic absorption from the colon. It is very remarkable how rare it is to find the complaint in patients of a phlegmatic or bucolic temperament, and it seems to be a fertile source of neurasthenia and high-strung nerves. Too frequently are neurasthenic patients subjected to "rest cures," and the usual routine treatment of such cases, without first having a thorough overhaul of their digestive apparatus. It is rare, too, to find the subjects of colon intoxication without nerve symptoms of some sort or kind. Commonly enough these may be of minor degree, but none the less distressing. Such patients often complain of depression and irritability; trifles worry them; they have difficulties in mental concentration, and as to memory. They lose their sense of well-being, are often restless, and are apt to get morbidly introspective.

It is a not uncommon cause of insomnia, and one which should be more widely recognized, as it often escapes the attention of the medical attendant. Nerve symptoms, however, may be of great severity. For example, I have seen success follow treatment of the colon in intractable hemicrania of many years' standing, where previously all ordinary treatment had failed, and the patient had had to be given morphia, under medical advice, for attacks of increasing intensity and frequency. Acute neuralgias, and some forms of neuritis, are associated with it, and I have found it the cause in several instances of tender scalp. Da Costa, writing in 1871, recognized the importance of inquiring as to the state of the large bowel in cases of anomalous nervous disease; and were this more frequently borne in mind much benefit would accrue to individual sufferers.

The effects of colon auto-intoxication on the circulation differ in the same way as the trouble produces different symptoms in different subjects. In some cases blood-pressure is abnormally low—this is often found to be so where there is marked mental depression. On the other hand, it would seem that in many instances it is a source of abnormally high pressure. Tachycardia or bradycardia may exist, or the pulse may exhibit marked irregularity in time and force. Poor circulation, as is evidenced by coldness of the extremities, is common, and is a very frequent symptom. I have seen it associated in more than one case with Raynaud's disease.

Diseases of the locomotor system not uncommonly find their origin in chronic intestinal auto-intoxication. It is a more than probable cause in some cases of rheumatoid arthritis, one of prime importance in gout, especially of the acute type. In quite a number of cases I have seen efficient lavage of the colon cut short an attack of this disease where previously all the more usual methods of treatment had failed to bring or maintain relief. Fibrositis, too, may find its origin in absorption of toxin from the digestive apparatus, and it is now recognized by many authorities as a direct cause of this painful and troublesome affection. The state of the skin in these chronic toxic cases is often peculiarly dry and harsh. Affections such as some forms of eczema, psoriasis, and chronic urticaria may arise from a similar cause. I have seen success follow treatment of the colon in obstinate conditions of herpes and dermatitis herpetiformis and also of septic state of the nails.

As to treatment: Diet is of the greatest importance, and must be suited to the peculiar needs of the individual. Speaking broadly, it would appear necessary to consider it as much from a mechanical as from a chemical standpoint. Foodstuffs calculated to leave a residue of undigested particles are apt to set up further colon irritability, and in the many cases where a tendency to spasm exists this can be a source of further trouble. Food must be masticated thoroughly, and eaten slowly. Meals should be "dry" and large quantities of fluid taken between meals.

In conclusion, I would most strongly urge the utmost importance of routine, thorough examination of the abdomen in all cases where there can be a possible doubt as to the origin of symptoms however remotely removed from that region. Too often such a thing begins and ends with an inquiry as to the state of the bowels. In cases where there can still be a doubt, and these are frequently enough met with, lavage of the colon may clear up a diagnosis. In regard to intestinal irrigations, my own experience has been almost wholly confined to those in which the Harrogate sulphur waters have been used as an irrigant, but in some instances where this was not available I have recommended normal saline solution. The fluid employed must be bland in type, and of proper temperature and pressure. The use of antiseptic solutions as a method of treatment is to be deprecated, as their prolonged administration is frequently attended by disastrous results owing to their tendency to produce further irritation.

Mr. FRANCIS BROOK: Neurasthenia has long been suspected to be due in a majority of cases to a toxic condition of the blood originating in an alimentary toxæmia, but hitherto no definite proof of that theory has been brought forward. I venture to think that, on considering the evidence I am about to put before you, you will agree that this theory is no longer mere conjecture, but rests on solid fact. I base this statement on three grounds: Firstly, the clinical symptoms;

secondly, the bacteriological evidence; thirdly, the successful results obtained from treatment on these lines.

Clinically, what are the symptoms of a chronic toxæmia other than alimentary—for example, chronic alcoholism? Are they not prostration, restlessness, headache, irritability, nervousness, disturbed sleep, hyperæsthesia and pains, irritable temper, easy fatiguability, loss of concentration, and loss of memory: and do not these symptoms correspond exactly with the symptoms of neurasthenia?

We now come to the bacteriological evidence. For a long period it has been my practice to examine bacteriologically the fæces in every case of neurasthenia which has come under my care. At Liverpool, last year, at the annual meeting of the British Medical Association, I gave the details of fifty-four consecutive cases of neurasthenia.[1] Every one of these showed an abnormal condition of the flora. At the present time my cases number 132, and I can only say that each successive case confirms me in my belief that chronic intestinal toxæmia is at the root of almost every case of neurasthenic trouble. In considering the bacteriological evidence the first thing to do is, I think, to define what one means by normal intestinal flora, and I would here state that the anaerobes have not been dealt with, and that the medium employed was nutrose-lactose agar with litmus as an indicator. The usual bacteriological picture presented by a culture plate of the above medium, sown with an emulsion of fæces from an average normal healthy individual, shows about 90 per cent. of a mixture of *Bacillus coli communis* and *Bacillus coli communior* in about equal numbers, together with about 10 per cent. of a saprophytic *Streptococcus brevis*.

One hundred and thirty-two cases of neurasthenia in all were investigated, and the bacterioscopic examination gave the following results: *Bacillus coli*, twenty-seven cases; *Streptococcus pyogenes longus* in practically pure culture, sixty-two cases. Here I have not recognized the multitudinous varieties of streptococcus differentiated by some observers upon minor differences of biological characters, and which are undoubtedly due to variations of environmental conditions, but have adhered to the original classification of Lingelsheim into the pathogenic *Streptococcus pyogenes longus*, and the saprophytic *Streptococcus brevis*. *Bacillus coli* associated with *Streptococcus pyogenes longus*, four cases; *Coliform bacilli* in practically pure culture, twenty-five cases. This coliform bacillus (probably an atypical strain of *Bacillus coli*), which produces blue colonies on nutrose-agar plates, and only ferments the monosaccharides, has been recognized by Distaso and Eyre as causally associated with mucomembranous colitis; indeed, the former observer regards it as practically specific in this connexion. At least two varieties of it may be present, a discrete and a spreading type. *Coliform bacilli* associated with *Streptococcus pyogenes longus*, seven cases; *Bacillus lactis aërogenes* in more or less pure culture, seven cases. Serological observations (agglutination reaction and opsonin estimation) were made in all the cases, except some of the streptococcal ones, and formed a most valuable guide, especially in those cases where the infection was a mixed one. It is interesting to note that of five cases on the lunacy border line, not one showed any change at all from the normal. A point of much interest is that in a large number of the streptococcal cases pyorrhœa was either present or, if not present, a definite history of it could be obtained. And here I think it cannot be too strongly emphasized that the pyorrhœa is the cause and not the result of the intestinal trouble, as some writers appear to infer—the same *Streptococcus pyogenes longus* being isolated in these cases from the pyorrhœa and from the fæces. Another point of interest is that the majority of the coli cases were in women in the neighbourhood of the menopause.

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[1] *Brit. Med. Journ.*, 1912, ii, p. 964.

I think it will be agreed that the above analysis also throws much light on the well-recognized connexion of neurasthenia with colitis, a fact which has long been admitted, and also with various forms of chronic arthritis and neuritis. A further point worth recording is that, acting on Schrenk-Notzing's dictum that impotence may be the only symptom of neurasthenia, two cases whose fæces were examined showed very definite infection by streptococcus. One of these patients is quite cured, is now married, and leading a normal sexual life.

Upon the bacteriological results my cases may be classified as follows:—

Class I.—Those in which the bacteriological flora appeared normal as far as could be judged by the cultivations obtained upon nutrient media, and where subsequent investigation by serological methods was needed in order to select such strains of *Bacillus coli* as had undoubtedly acquired pathogenic properties towards their host.

Class II.—Those in which the *Bacillus coli* normally present had partially or wholly disappeared from the intestinal flora, and was replaced by (*a*) *Streptococcus pyogenes longus*; (*b*) *Coliform bacillus*; (*c*) *Bacillus lactis aërogenes*.

[I should here like to record my thanks to Dr. Eyre for the work he has done, and the advice he has given me in making the bacteriological investigations.]

We now come to the treatment and its results. Autogenous vaccines were given in all cases. The dose was always made small at the beginning, and so any constitutional reaction was avoided. Injections were given as a rule once a week, although sometimes at 5-day intervals. Sour milk was given to certain cases, especially those whose fæces were alkaline immediately on evacuation, and I may here say that patients usually feel better when the motions are acid, and not so well when they are alkaline. I have recently employed a preparation of *Bacillus glycobacter* for this purpose, and have found it useful. Intestinal antiseptics were given from time to time, but, except perhaps beta-naphthol and sulphur, I cannot say that I found them of much service. High colon lavage was sometimes prescribed twice weekly, and was certainly of benefit to some cases. Diet did, I think, make some difference to the results. The adoption of a vegetarian diet certainly assisted in rendering alkaline stools acid. Successful results were obtained in the majority of the cases, and in many where previous treatment by other methods had completely failed. The improvement, both physical and mental, always coincided with the disappearance of the abnormal flora from the intestinal canal, and those cases which were not cured were those where nothing that one could do caused a return of the normal flora. These cases were almost invariably streptococcal in type. The treatment occupies in most cases about three months, the majority of them getting well in that time. The rapidity of the improvement in some cases is such as to be almost startling. With regard to the permanence of the cure, some of the cases have been under observation over two years, and they remain perfectly well to-day. From the patient's point of view, one of the greatest advantages of this mode of treatment is that it can be quite well carried out at home, and does not necessitate absence from his duties if he is able to work.

In Class I, where the plate cultivations appeared normal, evidence of infection by *Bacillus coli* was obtained in every case by means of serological tests—for example, agglutination reactions and opsonin estimations, and improvement followed upon the use of the appropriate autogenous vaccine. In Class II (*a*) the *Streptococcus pyogenes longus* was present, and under treatment the improvement manifested clinically was coincident with the disappearance of the streptococci from the fæces and the reappearance of the typical *Bacillus coli*. In Class II (*b*) the same remark applies to the clinical improvement as the *coliform bacilli* disappeared. In Class II (*c*) (*Bacillus lactis aerogenes*), two of the cases were seen once only and were not treated; the five which were treated made excellent recoveries.

One of the most striking results was the rapid improvement in the physical appearance; the pallor, muddy complexion, dark circles under the eyes, and blueness of the lips so often present, rapidly gave place to a healthy colour, the eyes became brighter and lost their dull heavy look, and the face lost its appearance of premature age. The indigestion, aching pains, lassitude, and irritability disappeared, and the change in the mental condition was equally marked. The memory and power of concentration improved, the depression and insomnia passed away, and the whole outlook on life became quite changed, and in fact normal, and marked increase in weight often occurred.

Neurasthenia has been classified according to whether the blood-pressure is high, normal, or low. Whether this classification is of any very great importance I do not venture an opinion, but I wish to call attention to the fact that in several cases where the blood-pressure was high it fell steadily during the course of the treatment.

TRAUMATIC NEURASTHENIA.

With regard to traumatic neurasthenia, in a recent address given at Milan by Professor Murri, published in the *Universal Medical Record*,[1] he points out that "cause" only exists as a complex of determining conditions, and that most frequently the accident is but the occasion that reveals pre-existing disorder, or that gives effect to it when the conditions are already ripe. The effects, therefore, are not specific to the occasion, but to the precedent condition. Of the two cases of traumatic neurasthenia I have so far investigated, one showed definite evidence of *Bacillus coli* infection, and the other gave a pure cultivation of *Bacillus lactis aërogenes*.

SUMMARY.

I would summarize my observations by advancing the opinion that the bacteria referred to bore a direct causal relationship to these cases of neurasthenia, for the following reasons:—

(1) An abnormal condition of the intestinal flora was present in more than half the cases.

(2) Serological tests yielded evidence of infection by the abnormal bacteria, and by some of the strains of colon bacilli in each of those cases where the intestinal flora appeared at first to be normal.

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[1] *Universal Med. Record*, 1912, ii, pp. 10, 97.

(3) Most important, the improvement in the patient's condition, both physical and mental, under treatment synchronized with the reappearance of the normal intestinal saprophytes in the fæces, and the disappearance of the abnormal organisms.

(4) Of course, one realizes that the personal element and the factor of suggestion cannot be eliminated, yet many of the patients who were cured had previously tried other recognized forms of treatment—rest cures, high frequency, Weir Mitchell hydropathy, &c.—where these same factors were at work; hence I do not think that they can fairly be said to have had much influence on the results.

I will now give you some illustrative cases, dividing them into (*a*) those which were successful, (*b*) those which were not; in these latter the intestinal flora remained practically unaltered in spite of the treatment. I may here point out that neither the symptoms complained of nor the clinical appearance of the patient give any indication as to the variety of bacterial infection which will be found on examination of the fæces. The following notes have been taken from my case-book just as they were entered. In choosing these cases for illustration, I have purposely taken those of least recent date as giving a better idea of the permanence of the results.

SUCCESSFUL CASES.

Class I.—Bacillus coli.

Case I.—Mrs. A. C., aged 46, married, no children. Healthy till an attack of influenza three years ago; then anæmia, depression, chronic rheumatic pains, and some neuritis. In spite of treatment, rest cures, various kinds of baths both here and abroad, the condition, with the exception of the neuritis, remained unchanged. She complained of great depression, loss of memory, lack of concentration, irritability, and bad rheumatic pains (the joints appeared normal), and also of the ease with which she became fatigued. Complexion was muddy, and the eyes dull and heavy. The fæces, culturally and microscopically, appeared normal, but *Bacillus coli* gave an opsonic index of 1·5. Treatment commenced July, 1911. After the eighth injection the depression had disappeared. She stated that she felt well and had plenty of energy, and could walk without fatigue. After the twelfth injection the index was 1·09. The will and mind were clear and the memory good. In April, 1913, she reports herself perfectly well, and the opsonic index is 1·2.

Case II.—Mrs. F. C., aged 40, married, no children. History of an attack of what was apparently ptomaine poisoning one year ago, since when she has never been well. Has suffered from a feeling of heaviness in the appendix region for four years. Complains of inability to walk more than a few yards without intense fatigue. Pains in the limbs, especially in the legs. She is constipated and never has an action without drugs. Acidity, feeling of fullness after meals, palpitation, sleep and appetite good, anæmic, the complexion muddy, skin dry, dark circles under the eyes. Fæces apparently normal; gave an index to *Bacillus coli* of 1·5. Treatment began November, 1911. She said she felt better after the first dose. Improvement was rapid, and after a month she could walk some distance without fatigue. Complexion became normal, and the face much fuller. When seen fifteen months later she was well, and in a recent letter states that she has remained so.

Class II (a).—Streptococcus pyogenes longus.

Case I.—A. S., male, aged 36, married, schoolmaster. Neurasthenia three years, which latterly became so bad that he had to resign his position as headmaster of a public school. The first symptom noticed was a burning feeling of the face, hand, and back of the neck; then followed lassitude, depression, and great irritability. He had no constipation, but was always worrying about the action of the bowels, the stools being always light-coloured. The blood-pressure was 200 mm. The reflexes slightly exaggerated, marked insomnia, frequent micturition, great physical weakness. *Streptococcus longus* was present in pure culture. Treatment commenced March, 1911. After the second injection he walked 20 miles, and was much more cheerful. At the beginning of May the *Bacillus coli* equalled the streptococci in numbers. Head much better. Blood-pressure 170. In July, much better generally, blood-pressure 150, streptococci diminishing, insomnia only causes one bad night in five. In November, no insomnia for three months, no depression, blood-pressure 160. Fæces have resumed normal type, culturally and microscopically. He has taken up cross-country running, and in January, 1913, writes that he is perfectly fit.

Case II.—Mrs. T., aged 37, married, three children, youngest aged 7. Colitis in India seven years ago. Six years' history of neurasthenia. Complained of great lassitude, depression, insomnia, loss of power of concentration, pains in legs, and chronic abdominal dragging pain, previously supposed ovarian in origin, but probably coming from the colon. No constipation, complexion muddy, skin dry, dark circles under the eyes, very anæmic. Fæces showed almost pure cultivation of *Streptococcus longus*. Treatment commenced April, 1911. The symptoms rapidly cleared up, especially the abdominal pain. Sleep and spirits became good, and the complexion normal. In September the fæces were microscopically and culturally normal. In a recent letter she reports that she is in excellent health.

Class II (b).—Coliform bacillus.

Case I.—G. W., aged 45, married, railway manager. Two years ago he suffered with a constant feeling of vertigo; he used to perspire very freely, at first only at night, then in the day also. Complains of a feeling of dread, and undefined haunting fear of doom. No cause at all. Sleeps very heavily, but always dreaming. Great lassitude. Occipital headache, and burning feeling on the top of the head. Bowels open daily with salines. Twitchings of arm and face; horror of noise; great depression; very easily tired. Has tried several forms of treatment. On cultivation of fæces, coliform colonies appeared, the opsonic index to which was 1·91, while to the normal coli it was 1·0. Treatment commenced in February, and there was rapid improvement from that date. In a fortnight the depression had disappeared, and dreams and fatigues were less. At the end of three months he was well, and now reports that his condition, both physical and mental, remains excellent, and that he enjoys his work, which had previously become a burden to him.

Case II.—W. P., aged 29, an accountant. Was quite well till nine months ago, when the present symptoms became manifest. He complains of worrying greatly about things (the condition becoming rapidly worse), lack of concentration, heavy sleep with bad dreams. He goes over and over his additions, because he thinks there may be an error. Has a dragging feeling on the top of the head, and sensation of swimming in the head when reading or doing any head work. Occasionally, when walking, he feels as though he reels, but does not actually do so. He never feels fit. Fæces showed coliform bacilli, index 1·4. Treatment was commenced in December. The symptoms gradually abated. He was able to add easily without going over the work more than once. The swimming feeling passed away, and in April he reported that he was absolutely well, and just going to get married. The fæces had then resumed normal type.

Class II (c).—Bacillus lactis aërogenes.

Mrs. N. G., aged 60, married, no children. Ten years' history of neurasthenia, in the course of which she had tried many forms of treatment without effect. She complained of a feeling of absolute exhaustion, as though dying, and that to do the slightest thing was a "mountainous" effort. Frightful depression. Very highly strung, keeps going till she drops. Flatulence, acidity after every meal, pain and tenderness over colon on right side. Sleep broken by bad dreams; constant ringing in ears. Head always feels too heavy, and as if falling backwards. Mucus in stools, which are usually constipated. The patient was very anæmic, complexion pasty and yellow, skin dry, sclerotics yellow; blood-pressure 240, pulse 56. *Bacillus lactis aërogenes* in pure culture in the fæces. Treatment was followed by rapid improvement, digestion became good, the depression disappeared, and also the abnormal feeling of fatigue. Complexion cleared, and after ten injections the patient was quite well. The blood-pressure in the meantime had fallen to 170, and the pulse had risen to 68. After twelve months she has remained quite well.

We now come to the unsuccessful cases. These were quite small numerically, and usually streptococcal. They were not absolute failures, but they were not cures.

UNSUCCESSFUL CASES.

Case I.—E. E., aged 46, musician. Ill eight years. When he entered my consulting-room he announced that I was the twenty-eighth doctor by whom he had been treated. Operation on left kidney eight years ago for supposed renal colic. Nothing was found, and I have no doubt the pain had its origin in the colon. Twenty-eight weeks in bed last year. Complains of great lassitude, weakness, irritability, and depression. Every molehill appears a mountain. Periodical attacks of pain, very acute in left side of abdomen, running down to legs. Diarrhœa and unformed stools. Fæces showed streptococci in nearly pure culture. Treatment commenced February, 1911. He improved steadily in spirits, energy, and colour, which had been very pasty. In May the streptococci equalled the *Bacilli coli* in number. Stools now formed. In November, after a lapse of three months, the streptococcus to coli ratio was back to 10 to 1. In February the numbers were equal, and there he remains for the present. Twelve months later he reports his condition slowly improving. He is immensely better and looks a different man, and he has only had one day away from work in eighteen months, but I cannot say he is cured. I have discussed removal of his colon with him, but this he has refused.

Case II.—G. D. C., aged 49, male. Ill six months. Complained of vertigo and a feeling of reeling, although he never did so. Depression, lassitude, pains in legs, irritability. Sclerotics yellow, skin dry, eczema of old standing. Blood-pressure 180. Fæces showed streptococci in almost pure culture. Treatment begun in July, 1911. Gradual improvement took place in his condition, and in October the streptococci and coli were in equal numbers; blood-pressure 130. In January, although his condition remained about the same, the culture showed nearly all streptococci again. In February they were again equal, blood-pressure 126. He was much better, but not well, and then went to Switzerland. This case was one of those in which I tried a vegetarian diet for a time, but gave it up as it did not seem to be benefiting the patient.

Dr. WILLIAM ARMSTRONG: I should like to record the results of sixteen years' study of the many problems of alimentary toxæmia, and the lessons I have learnt regarding its treatment. Since I first wrote on this subject some fifteen years ago, great strides have been made in the scientific study of the intestinal ferments and flora, and their behaviour. But I doubt if similar progress can be claimed for the therapy.

(I) DENTAL SEPSIS.

I have repeatedly found that pyorrhœa alveolaris and other septic oral conditions have originated, or kept up, gastric disturbances; and in some cases arthritic mischief in the joints. Latterly I am convinced that there has been a tendency to overrate the proportion of arthritic cases due to this cause; but the excellent work of Goadby has, to my personal knowledge, given, in properly selected cases, brilliant results.

(II) GASTRIC STASIS AND ATONY.

The collection of symptoms comprising recurrent migrainous headaches, stiffened and swollen finger-joints; morning malaise; "never feeling really well" until after midday; the condition described as "livery" or "suppressed gout": these, even when of very long standing and refractory to treatment, completely clear up when the stomach is regularly emptied, and the fermentation of the delayed food with its formation of butyric acid and poisonous gas is checked.

It is difficult to persuade the laity that the "sluggish liver," which they so strenuously condemn and so pitilessly drive, is not the culprit, but really the victim of overwork, in its gallant attempt to destroy the digestive toxins, so much larger in quantity and so much more poisonous in quality owing to over-prolonged food retention in the stomach. The treatment divides itself into three sections: (1) Methods of emptying the stomach; (2) measures to strengthen the atonic gastric wall, and its weakened nerve supply; (3) remedies to control poisonous fermentation.

(1) The administration of a weak, hot solution of Carlsbad salt on van Ziemssen's plan daily. That is, 1 pint in wineglassful doses spread over three-quarters of an hour, fasting; or, this failing, gastric lavage two or three times weekly should be used.

(2) Mechanical treatment, chiefly of the electrical or vibratory order (with or without massage) over the atonic stomach, over the cervical and splanchnic ganglia; and also over the solar plexus.

(3) Bouchard's most valuable but neglected remedy: fuming hydrochloric acid 5 to 10 minims, mixed *freshly at the time of administration* with 6 to 8 oz. of water, and taken with every meal. This is of exceptional value, and greatly reinforces the antiseptic power of the gastric juice.

(III) DUODENAL FERMENTATION AND PUTREFACTION.

When the urine contains a large excess of indican, skatol, and cresol, patients show some one or more of the following symptoms: Feeling of general malaise; dry, muddy-looking skin; sallow complexion; general irritability and restlessness; enlargement of joints, especially those of the fingers; neuritis; discomfort and gaseous distension, especially of the transverse colon, two or three hours after food; and other symptoms of putrefactive fermentation of the carbohydrates and of poisoning by resulting starchy toxins; a tendency to obesity of an intractable type; and especially a marked rise of blood-pressure. I am convinced that a large number of the presclerotic type of high-tension cases are caused by duodenal poisoning. When pyrocatechin is present its poisonous effect upon the spinal cord probably accounts for the large proportion of arthritic cases observed.

The most effective treatment was calomel 1 gr. once or twice a week, followed by a sulphate of soda morning aperient; and either the petroleum and bismuth emulsion as suggested by Dr. Bezly Thorne, or resorcin, bismuth-salicylate, and phenol in tablets.

It will be remembered that the late Dr. Salisbury attributed all the ills that flesh is heir to, to starchy toxins; and although I cannot travel with him so far along that road two points have impressed themselves upon me. One that there are many cases of starchy toxæmia; the other that the red meat and hot water dietary suggested by Salisbury is of distinct value in many severe examples of this condition.

(IV) INTESTINAL POISONING.

In seeking a reliable working indication for the need of active treatment of this condition I found that a considerable excess of aromatic sulphates in the urine supplied such a guide. Some eight years ago in the arthritis number of the *Practitioner*, I published, with full details, a case of severe rheumatoid arthritis involving many joints. So great was the excess of aromatic sulphates that I took it as a test case, and gave up all treatment except very large intestinal douches carefully and very slowly administered. As much as 12 pints was administered at one time without discomfort; the case steadily improved and eventually made a complete recovery. Since then I have treated a considerable number of arthritic cases on the same lines. With the advent of the Plombières douche, that fashionable form of intestinal lavage, many more arthritic cases have been treated by this means; but its use without proper differentiation has caused disappointment, just as a similar misuse of the "sour milk" and "Salisbury" treatments has greatly damaged those valuable methods in the eyes of our profession. Those somewhat rare but most intractable cases of recurrent acute erysipelatous eczema have invariably responded to thorough intestinal lavage. Many chronic and refractory cases of various kinds, all however possessing one feature—viz., marked excess of aromatic sulphates in the urine—have responded readily to this treatment.

I would draw attention to three general methods which I have found of the utmost value in all forms of alimentary toxæmia:—

(1) The administration of a strong solution of radium emanation in water. Lazarus, Saubermann, Deutelmozer, Berger, Bickel, and many others have shown that this (*a*) greatly increases the activity of the various ferments, especially the peptonic and diastatic; (*b*) markedly improves nerve vitality, especially of the sympathetic system; (*c*) stimulates the output of the body waste: increasing the carbon dioxide from the lungs by 20 to 60 per cent.: the sweat toxins by 40 to 70 per cent.: and the urinary poisons by 10 to 15 per cent.; (*d*) has a destructive influence, *in large doses*, over low forms of bacterial and spore life.

(2) The use of oxygen, either inhaled in association with radium emanation, or given in the bath by the double powder of sodium perborate and manganese borate.

(3) Professor Bergonié's method of strong, rapid, painless, simultaneous contraction of practically the whole of the muscles, which are weighted to increase the contractions. I have found that this method, originally introduced for the rational treatment of obesity (in which it has proved most effective) most helpful in alimentary toxæmia. It braces up the abdominal walls, and also the atonic intestinal canal; empties the lymph spaces between the muscles, and quickens the often stagnant lymph circulation; stimulates the whole nervous system, and speedily dissipates those feelings of malaise and misery which make the life of the alimentary toxæmic so wretched; paving the way, if allowed to go unchecked, for that most intractable form of neurasthenia which is at once the despair of, and a reproach to, our profession.

Dr. W. Knowsley Sibley: A very large number of skin eruptions are the external manifestations of a morbid condition of the general system, directly due to a toxæmia of the blood, the result of the absorption of morbid products from the gastro-intestinal tract. It follows that in all of these conditions proper attention not only to the diet or "input," but also to the excreta or "output," is of primary importance for the satisfactory treatment of the skin complaint. A system saturated with the toxins absorbed from an overloaded intestine is by no means an uncommon cause of a large number of apparently very different skin diseases. In fact, it would be difficult, in the present state of our knowledge, to name many manifestations (excluding, of course, the various parasitic diseases, both animal and vegetable) which are not, in many cases, the external expression of a general unhealthy condition of the blood supplying the region, the result of a defective assimilation of the digestive processes, or more generally of a delayed excretion of the waste products of the digestive metabolism.

The secret of a good complexion is usually to be found in a properly regulated diet in accordance with the requirements of the individual case, and one so regulated as to ensure an efficient evacuation of all the organs of excretion, especially of the bowels, thus not permitting the by-products of digestion to remain long enough in the body to be in any way absorbed into the general system. The first sign of a want of balance between assimilation and excretion is usually to be found in the colour and condition of the skin, especially in certain regions of the body. A pasty or muddy complexion is often one of the signs of mal-assimilation, or of absorption from the contents of the large bowel, or both. The results of so-called hepatic derangements show themselves very quickly in the colour of the skin, and point to digestive disturbances of various degrees. A large group of skin phenomena under the heading of urticarias, or nettle-rashes, has long been recognized as the direct result of gastro-intestinal influences and are designated "stomach rashes." Many other common diseases, both acute and chronic, are also determined by similar processes. Consider in the first place eczema, one of the commonest diseases the dermatologist is called upon to treat There is no doubt that the condition of the gastro-intestinal tract plays a leading part in a large number of these cases, and that proper attention to the regulation of the diet, and of the bowels is necessary for the satisfactory treatment. The popular expression of a "gouty eczema," used to denote an eczema in a gouty subject, has much to commend it, for most of the phenomena associated with the term gout or goutiness, are but the varied expressions of a want of balance between the input and the output, or of a mal-assimilation and delayed excretion of the by-products of digestion, giving rise to various systemic disturbances, and among them numerous dermatoses.

Psoriasis, again, is often undoubtedly the result of toxic influences, derived from the gastro-intestinal tract, and a complete change in the diet from a too nitrogenous to a vegetarian one, will often cure an old-standing case. So again, acne, in its various forms, is generally associated with an overloaded bowel, and free purgation is often the most efficient treatment: the same may be said of many cases of purpura. Even in many diseases obviously due to the invasion of micro-organisms such as staphylococci, streptococci, or perhaps even the acne bacillus, a toxic condition of the blood, due to a systemic poisoning from fæcal or other morbid absorption, is necessary for the growth and development of these organisms. From my experience I should say that, speaking generally, the immunity of the skin to micro-organisms is directly impaired by an obstructed drainage system. This list of skin phenomena would be incomplete without mentioning those remarkable and very common pigmentary changes which occur, sometimes locally, more frequently generally, to which Arbuthnot Lane has drawn attention.

Alcohol plays a very important rôle in the production and keeping up of many morbid skin conditions, especially those of an obviously inflammatory nature, such as erythemas, urticarias, eczemas, and numerous other irritable dermatoses, not omitting to mention some of the more inflammatory types of psoriasis. The immediate effect of taking alcohol internally is to produce an external flushing of the superficial vessels, and so to increase the skin congestion. Tea, coffee, cocoa, and tobacco, are often harmful in much the same way. Complete abstinence from alcohol is of primary importance for the satisfactory treatment of many of these cases. Many people do not drink enough liquids, and these should take copious draughts of water, which should be drunk as far as possible on an empty stomach. A too generous nitrogenous diet often plays an important part in some cases, especially in eczema, acne and psoriasis, and a change to a more or less vegetarian one will often bring about a cure. In some patients a diet of rice and water, in others a pure milk diet, will often clear up an old-standing and very obstinate skin lesion, especially if of an inflammatory or irritable type.

The researches of Metchnikoff have shown how the various lactic ferments arrest and are destructive to many deleterious fermentative processes going on in the intestinal tract. The ingestion of sour milk in various preparations produces a much healthier and more satisfactory condition of the intestinal contents. Many cases of skin eruptions due to gastro-intestinal toxæmias may therefore be satisfactorily treated by this method.

It is evident that in a large number of cases of general skin diseases it is the mucous membrane rather than the skin which requires the chief attention. The skin lesion may be controlled and rendered more comfortable by suitable local, more or less sedative, protective or bland applications, but it is to the gastro-intestinal tract that the main treatment should be directed. In every case of skin eruption a systematic examination of this should be made, before determining the special line of treatment. Commencing with the mouth, the whole systemic disturbance may be due to a neglected pyorrhœa alveolaris, absorption from which, either locally, or more frequently from the products of this disease being perpetually swallowed into the stomach, setting up a gastro-intestinal catarrh, absorption taking place from this organ.

I discussed the relation between pyorrhœa alveolaris and certain diseases of the skin in a paper read before the Odontological Section of this Society[1] two years ago, in which I enumerated a large number of different eruptions which could often be traced to pyorrhœa, and the part it played in alimentary toxæmia, and the treatment for which eruptions was to be directed mainly to the cure of the condition of pyorrhœa. Some of the diseases I then enumerated were acne, vulgaris and rosacea, alopecia, especially alopecia areata, eczema, erythema, herpes, œdema, seborrhœa, and seborrhœic eczema. To quote from my paper above referred to, I then said, "Many cases of eczema at all ages are due to gastro-intestinal causes, urticaria invariably so. Many erythemas, again, are traced directly to stomach derangements. Even psoriasis is considered by some dermatologists to be an external expression of the same internal troubles, and the most efficacious treatment is one which regulates the diet of the individual, or in other words, the gastro-intestinal absorption."

Leaving the oral cavity and passing on to examine the stomach, there may be a chronic gastritis due to errors of diet, especially from the abuse of alcohol, producing a catarrhal condition affecting not only the stomach but also the hepatic system, and extending its baneful influences on into the duodenum and small intestines. More frequently still, the colon is found to be loaded with old-standing fæcal accumulations, gradual absorption from which produces a general systemic poisoning.

To sum up: The treatment of a large number of general skin eruptions resolves itself into the scientific treatment of chronic constipation. With regard to this most important fact in dermatology, it has to be realized that because a patient has a daily evacuation of the bowels it by no means necessarily follows that he does not suffer from all the morbid effects of toxic absorption from an overloaded colon. It is not the frequency of evacuation that is of importance but the quantity, that is to say, that the contents of the large bowel are systematically completely removed, and that there is not an ever-increasing residue left behind. In other words, it is necessary to be sure that the patient is not passing to-day what he ought to have got rid of a week, or perhaps a month ago.

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[1] *Proceedings*, 1911, iv (Odont. Sect.), pp. 71-84.

I have often remarked that most, if not all, of the drugs which have been found of benefit in the treatment of general diseases of the skin act chiefly, if not entirely, as laxatives, aperients, or disinfectants of the gastro-intestinal tract, and I believe their beneficial effect is due to their direct local action on the gastro-intestinal contents, rather than to any specific action of the preparations themselves, giving rise to the improvement, or cure of the skin condition. The most popular drug with the older school of dermatologists is arsenic, which is often prescribed as a specific for most of the more or less chronic forms of skin diseases. Arsenical preparations are among the most powerful bactericidal agents known to chemists, and apart from their irritating and therefore laxative effects on the mucous membrane, it is to this property that their so-called specific action is probably due. Mercury, a specific for other skin diseases than those produced by syphilis, is essentially a most powerful germ destroyer. Liquor hydrarg. perchlor.—corrosive sublimate—even in very dilute solution, has strong bactericidal properties, and acts as a powerful and far-reaching intestinal cleanser. Quinine in solution is also a powerful bactericidal agent and disinfectant, and is equally efficacious for this purpose when administered into the stomach, as when used for washing out the bladder or other viscus. Perhaps the next most popular drug, and one which still holds its place, is iodide of potassium. Here again, iodine is now acknowledged to be one of the most effective bactericidal agents known, and hence its utility in the cure of a large number of very different skin diseases, especially when prescribed in comparatively small doses, which permit of the iodine being liberated, and so acting directly on the gastro-intestinal contents. The more recent introduction of drugs of the coal-tar series such as the salicylates, salol, carbolic acid, resorcin, β naphthol, methyl violet, &c., which are being more and more extensively prescribed by dermatologists, are all of them bactericidal agents, acting directly on the gastro-intestinal mucosa and its contents. The old adage to prescribe aperients in acute, and diuretics in chronic, skin diseases is as true to-day as ever it was. The most popular prescription in the St. John's Hospital pharmacopœia is the laxative [illegible], the most important ingredient of which I believe to be sulphate of magnesia, and it is extraordinary in how many skin complaints this mixture acts as a specific.

I maintain, then, that with the exception of a few specifics for skin diseases, which are themselves the result of specific organisms, either local or general, practically all the other old and modern drugs prescribed by dermatologists act primarily and essentially as gastro-intestinal disinfectants or laxatives, or both. Moreover, all drugs which do not act in this direction are deleterious and positively injurious in the majority of cases. For instance, opium and its derivatives, which, with the possible exception of very occasionally in old people, should never be prescribed by dermatologists. Although this drug may temporarily relieve the distress of such skin conditions as urticaria or pruritus, as soon as its immediate sedative effects have passed off it will almost inevitably produce far worse symptoms and greatly aggravate the complaint.

Dr. H. D. McCulloch said: My interest in this discussion is from the physiological and biological standpoint. It is intended in some measure to link up with the bacteriological, pathological, and anatomical aspects so ably submitted by previous speakers. In the first place, I desire to draw attention to the absolutely sterile condition of the alimentary tract at birth and to the pre-natal[1] existence of ptyalin in the buccal secretion, a ferment which in infantile life is mainly concerned with the control of oral sepsis. The sudden delivery of the infant from a normally sterile into a notoriously microbic environment is anticipated by its possession of a comparatively developed lymphoid and lymphatic system, the highly cytogenous glands of which, eminently equipped as they are for the purpose, bear the brunt of the heterogeneous and overwhelming microbic invasions that must necessarily ensue at and during the first few months of life. The modus operandi, and the remarkable reactive properties of these glands, pending the disappearance of the thymus, and before the assumption by the bone-marrow of its hæmopoietic function, is a fascinating and instructive study. The lymphatics are in no sense of the term mere filters. I will venture to say that by overlooking their mechanism Metchnikoff has, by his more recent surgical recommendations, vitiated the fertility of his first great discovery. These operations are extremely futile. It will be admitted that the peculiar anatomical disposition of the lymphoid glands in particular, and of the lymphatic glands in general, strictly corresponds with those regions of the alimentary tract that have the greatest microbic habitation, a fact that may be more noticeable in adult than in later life. The aged are known to acquire strong heterogeneous immunities, though their pulmonary immunities are of a fleeting kind. The relative term "health" is in the main conditioned by the functional equilibrium of the lymphoid and lymphatic systems of glands. And since now one, now another, of these glands takes the lead, and evokes the antagonism of the others, as the rhythm of growth and development proceeds, from the biochemical aspect a series of intoxications of exogenous origin, followed by slowly acquired immunities—that is to say, of mild illnesses and recoveries—is the normal course of events. These immunities depend, doubtless, upon the extent of those already inherited. The glycogenic and hæmolytic liver is remotely protective, and this by virtue of its accessory lymphoid and lymphatic glands. The blood in the portal vein is as rich in excretory complexes as it is in those of the carbohydrates and fats. Bacteriological investigation of the vast majority of microbic diseases indicates the existence of a closer relationship between them than hitherto assigned by clinicians, who have devoted more consideration to the chemistry of the conditions than to the biological processes and the reactive mechanisms, vaguely termed inflammations, which have initiated and gradually produced them. The earliest stages of an inflammation are invisible, the later stages are obscured by redness. We are no further than Celsus was.

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[1] "Intestinal Pathology and Hygiene."—Hutchison.

I next beg to draw attention to the existence and better appreciation of symbiosis in its evolutionary aspect—that is to say, to the functional association of the lowest unicellular forms of life with higher mammalian forms, as is to be seen in the nitrogen-fixing microbes of leguminous and other farinaceous plants, an essentially physiological capacity. In mammals the nipples from the outset teem with milk-clotting organisms, which in the earlier periods of evolution were probably pathogenic to the suckling. Those of them that favoured an acid medium seem to have since become adapted, and their peculiar qualities are utilized in certain situations to mutual advantage. They have thus become physiological necessities in the last stages of digestion. For reasons that will suggest themselves this feature is most noticeable in the Tropics,[1] less in the temperate zones, and least in the inhabitants of the Arctic Regions. The last-mentioned are obliged to live upon fats and oils; they do not, therefore, suffer from intestinal stasis; if they drank beer and tea they probably in the course of time would. Abdominal surgeons are rare in Spitzbergen. Let us deplore the time-honoured periodic administration of castor oil, the cold-drawn variety—*ad baculum*—having become obsolete. The palate is pampered at the expense of medicinal virtue. Experiments have proved the folly of supposing an absolutely sterile intestinal canal to be consistent with function. Dr. Hale White drew attention to the uses of the *Bacillus coli*, Mr. Sargent and Mr. Dudgeon have drawn attention to the uses of the intestinal *Staphylococcus albus*, and Metchnikoff rightly ascribes the highest place to the *Bacillus lacticus*. In regard to the resulting intestinal ferments that are protective, Sir Lauder Brunton and Abderhalden have indicated the capacity of these micro-organisms under varying conditions of acidity and alkalinity, of producing the desired end-products for ingestion. Indeed, the recent recognition of the biological expression "carrier" lends support to this evolutionary view of symbiosis as a natural and environmental necessity.

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[1] The large admixture of spices and condiments used in tropical dietaries is scarcely appreciated in European cooking. The majority are anti-microbic.

The further point that I would raise—one that a few physiologists are already prepared to admit—is the desirability of considering the extra-vascular lymph or plasma to be the sole nutrient fluid, *the* fluid that most needs protection from toxic contamination. The blood being more a respiratory and an excretory provision, I contend that upon this view alone is it possible to account adequately for the production of peri-arteritis, sclerosis, and fibrosis, which result from exogenous toxæmia. The very intimate relationship between these fluids is not denied, but the swift movement of the blood is more consistent with the rapid distribution of oxygen and the removal of excretory products than it is with the slow process of protoplasmic synthesis and growth. It is necessary to bear in mind the pre-mesodermic origin of the lymph systems and the later mesodermic origin of the arterio-venous system a fact that indicates the interposition of the latter between the lymph on the one hand and the ultimate tissue cells on the other. The blood does not bathe the ultimate cells; indeed, the plasmic as distinguished from the axial stream of the arterioles terminates at the arteriolar bifurcation, from which point erythrocytic function in the ultimate capillaries is alone capable of being performed. Upon this subject a communication was submitted by me to the Physiological Society last March, and experimental proof was first shown in this hall on April 29 by Dr. Monod in the pulmonary circulation of the frog.

Permit me to criticize the misuse of the term "auto-intoxication" as applied by Mr. Arbuthnot Lane and others. Adami, in his treatise on General Pathology, emphasizes this point by saying that laxity in regard to the use of the term "auto-intoxication" has caused great confusion, and has rendered a term capable of high value particularly useless.[1] It is suggested that the term "absorption," in the present state of knowledge of biochemical problems, should be replaced by the term "ingestion." Adami also lays stress upon the fact that the human organism is a closed corporation, strictly speaking. He represents it by a figure of the simplified individual, which speaks for itself. The tempting notion of the easy accidental ingress into the circulating blood of microbes not gifted with intrinsic powers of penetration is a delusion. Their insertion by trauma into the venules is another matter—a remark that applies to the occasional ingress through abraded or degenerated mucous membrane into the meningo-faucial lymphatic sheaths. Imperfect phagocytosis and subsequent disgorgement in the deeper tissue interspaces is a more common and consequential occurrence. Such liberated organisms would set up thickenings in those situations according to their toxicity, without necessarily leaving any trace of their existence. They could not be recovered and cultivated. Toxins that are ingested into the portal blood may be discharged in the bile or there be modified, and, if not discharged by the lungs or the skin, would eventually be discharged by the kidneys. Failing these emunctory channels, and depending upon the relative viscosity of the fluids within and without the blood-vessels, certain toxic molecular complexes are constantly escaping by diffusion into the lymph. It is to meet this danger that the inception of endarteritis, periarteritis, and sclerosis takes place. It is owing to this liability to escape from the blood during excretory transit and to no other cause, I have endeavoured to fasten attention on this question of the lymphatics during the past few years. The more I have studied them the more is this truth borne in upon me, and the more I am concerned to champion in my humble way their important rôle in the mechanisms of disease. Like the indispensable foreign secretary, how many wars has he nipped in the bud by his successful diplomacy carried on over a long period of years, and of which the public are left, by his very success, in entire ignorance? *Credidi quod propter locutus sum.*

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[1] Adami, "General Pathology," p. 300.

Dr. J. F. Briscoe: Seventeen years ago I read a paper on "Constipation in the Insane" before the Psychological Association of Great Britain and Ireland, and went closely into details, especially on the baneful effects of intestinal stasis from the mental side. I alluded to the physical consequences of constipation and included them all under one categorical heading—toxic.

It is obvious that constipation is only a link in the series of causes and sequences arising from the disturbed abdominal viscera, and on the mental side encourages and accentuates such symptoms as apathy, irritability, perverted moral feelings, melancholia, mania—nay, suicide; while on the bodily side it produces very marked emaciation, rheumatism, cachexia, and a long string of diseases.

The animal economy is the theatre of toxic evolution and elimination—phenomena which are brought about by putrefactive processes, and which are always normally present in the fæcal contents of the bowels. The first point then to be noted is that every person who voluntarily constipates his bowels exposes himself to the risk of being morbidly constipated; and he, on the other hand, who regularly obeys the call of Nature, thereby prevents the accident of constipation.

It is the business of medical men to study, not only the influence of the mind on the body, but also that of the body on the mind. That constipation plays an important part in producing disease cannot be denied. Who, on the one hand, has not seen a prodigious evacuation of the bowels at the hands of the physician terminate a case of insanity, and, on the other hand, has not observed the skill of the surgeon unfold the mechanical obstruction which has led to the deadly consequences of intestinal putrefaction.

The physicians of a former age laid great stress on the congestive states of the abdominal viscera, and were most heroic with their bleedings and purgations. As in everything else "fashion changes," and nowadays the student of medicine rarely performs the operation of venesection, yet a bleeding judiciously and timely resorted to does undoubtedly, in some forms of disease, end beneficially an impending physical catastrophe. From humoralism we have passed to solidism, from solidism we return again to humoralism, and the microbe is the only connecting link. Investigation is now in the direction of the animal alkaloids and their effects. If the animal economy is not purely a chemical apparatus, it is far less a fermenting vat. Thus we see the school of the past and the school of the present will be found mutually to support each other. The cesspool of the human economy, now under the inspection of the Fellows of this Society, demands more than ordinary parlance, for it has opened up a matter of the very deepest concern if preventive medicine is a reality of to-day. The human sewer is a difficult subject to handle, for is not its normal state dependent upon the habits and methods of living, and which, moreover, does to the hygienist seem almost insurmountable when we consider the ideal state of affairs necessary for the most perfect results? Until dental and intestinal drilling are thoroughly grasped and understood by the people, alimentary toxæmia must continue as a cause for a large number of diseases.

The toilette of the digestive system has always been a difficult problem of solution. The state of the tongue may be a good index of intestinal health, but a foul condition of the breath speaks volumes of what may be suspected lower down, although the patient may not be constipated. I was talking to a nurse yesterday, and she stated she had been operated upon for appendicitis, unaccompanied by constipation. But as I faced her in conversation an unpleasant odour was projected from her mouth. It is obvious that it is not always the stress of the stay of the fæces in the alimentary canal that causes alimentary toxæmia, as it is the kind of food and quantity that has been partaken of, and in what manner it has been masticated. There is no doubt whatever that the person who eats wisely and triturates the food carefully has a more favourable chance of escape from a bottled-up appendix than one who regards carelessly and ignorantly his intestinal toilette; of course, I am speaking of purely medical cases apart from physical kinks needing immediate surgical skill and ingenuity for their restoration.

To-day there are some signs of a whole crop of difficulties facing the general physician, and does he not often appear to be powerless without the aid of the surgeon's scalpel in the treatment of abdominal toxæmia? And yet this should not occur, provided that he could follow and practise that counsel of perfection in the care and management of the digestive track as is carried out in the private and public mental hospitals of these isles. Only last year, at the annual meeting of the Psychological Association of Great Britain and Ireland, I pointed out, in opening a discussion on appendicitis in private and public hospitals for the insane, that it was practically unknown. And I incorporated in my remarks the fact that this freedom from appendicitis, typhlitis, or perityphlitis was not only among the adult insane but also among the children of such institutions as Earlswood Asylum for Idiot

Children, and in other sanatoria of the kind, where feeble-minded and physical young cripples were under care and management. My paper, and the more important part following it, the *discussion*, appeared in the *Journal of Mental Science*, October, 1912.[1] I received a pleasant and unusual return for the time I had taken in preparing the matter as opener of the debate, for, without exception, I carried my point, "that appendicitis is very rare among the insane inmates of all the private and public asylums of England, Wales, Scotland, and Ireland." And, before concluding these fragmentary remarks touching on alimentary toxæmia, I cannot conclude until I recapitulate some figures from the *Journal of Mental Science* quoted above, to give greater weight to my statement regarding appendicitis.

In ten years from 1902 to 1911 there are recorded by the English Commissioners in Lunacy seventy-five deaths from appendicitis, typhlitis, or perityphlitis, ascertained in the majority of cases by post-mortem examination, as having occurred among the insane population of the County and County Borough Asylums, Asylums for Idiots, Mental Hospitals and Licensed Houses of England and Wales. And of the seventy-five deaths, it would be instructive to know how many of these had the relics of this disease upon them, or were suffering at the time of admission from acute or chronic symptoms. And in making the statement that appendicitis is a rare malady in private and public hospitals for the insane, I do so with the support of several practitioners of psychiatry, well known to this Society of Medicine. Not only do these clinical observers give me their assistance, but further I have the help of the Pathologist (Dr. Mott) to the London County Asylum at Claybury, who states: "I do not believe there has been a single case of appendicitis on the post-mortem table at Claybury since I have been Pathologist. Consequently, in more than 2,000 post-mortems there has been no case." In comparing the Claybury Asylum for the insane with two general hospitals, St. Bartholomew's and Guy's, we can make a contrast. For instance, at St. Bartholomew's Hospital 1,645 autopsies were made between 1909 and 1911, and of these, 69 were recognized as appendicitis. At Guy's Hospital in the year 1900, of the first 500 post-mortems twelve were recorded as cases of appendicular disease. As regards the proportion of cases of appendicitis to the number of admissions, I find that at the same Hospital in 1890 there were 8,588 admissions. Of these admissions, 306 were subjects of appendicitis, 187 being under the care of surgeons, while 119 were under the care of physicians. The death-rate from appendicitis recorded in the Registrar-General's Report for 1909 shows a slight gradual increase from 1901 to 1909. Referring to other parts of the Kingdom in this investigation, I find the statistical tables from the Lunacy Board of Scotland show that formerly deaths from appendicitis were usually returned as deaths from peritonitis, that appendicitis had no separate heading in the Board's tables of causes of death, and is not yet separately notified. If a case of appendicitis were returned it would be placed in the table of Diseases of the Digestive System; but it has been ascertained that no death in a Scottish asylum has been chronicled during the past five years as due to appendicitis. Again, the Blue Book of the Inspectors of Lunatics for Ireland, in the issue for 1911, gives typhlitis grouped among the causes of death, but the table gives no return of the complaint for that year. And in the table there are only eleven cases of peritonitis. Likewise the Secretary of the Office of Lunatic Asylums, Dublin Castle, sends me the returns for typhlitis for the period 1902 to 1910 as follows: 1902, 0; 1903, 1 male; 1904, 0; 1905, 1 female; 1906, 0; 1907, 1 male; 1908, 2, male and female; 1909, 0: 1910, 0—viz., five in all. And the cause was ascertained, except in one case, by post-mortem examination. With these figures for your guidance I would remark that in experimental research and statistical calculations, extreme care and accuracy should survive comment. But, with the greatest attention and thoroughness that any of us can spend on the subject-matter of a professional inquiry in medicine, exception will be sure to prove the rule.

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[1] And also in the *Brit. Med. Journ.*, 1912, ii, p. 118.

Dr. ROBERT BELL: I trust I may be pardoned if I refer to a misstatement which Dr. Saundby has unwittingly made when he said that Bouchard originated the doctrine of auto-intoxication, a remark which, I think, is hardly correct, for by referring to the *Lancet* of 1880, vol. i, there will be found an article contributed by me, in which I believe the subject of auto-intoxication was drawn attention to for the first time in medical literature. I therefore submit it is unfair not only to me, but to the medical profession of this country, to pass on to a foreigner the credit which is due to an Englishman.

When notice was given of this then impending discussion, my impression was that it would be chiefly concerned with *auto*-toxæmia, and it is upon this branch of the subject only that I propose to focus my remarks.

In Dr. Hale White's able address I observed that he is inclined to look upon pyorrhœa as a primary form of alimentary toxæmia. From this view of the subject I feel I must differ *in toto*, as my experience of the disease has invariably led me to the conclusion that it is, in every instance, secondary to auto-toxæmia, arising as it does in subjects whose diet has been at variance with their physiological necessities. One example will be sufficient to illustrate the correctness of my assertion. About two years ago a well-known public speaker came to me in great distress in consequence of the fear that his career, as a professional man, would be sadly handicapped were he to follow the course which had been mapped out for him with regard to his teeth, of which he had a most perfect set; every one of them, however, was in a precarious condition in consequence of pyorrhœa. This I attributed to a faulty diet, associated with intestinal stasis and consequent auto-toxæmia. The diet, therefore, was restricted to what I contend man was intended to confine himself to, if he wishes to obey those laws which are in conformity with his anatomical and physiological structure—viz., cereals, fruits and vegetables, eggs, milk, and cheese. The result, in this interesting and anxious case, was, that in eight weeks the pyorrhœa had completely disappeared, and, coincidently, all apprehension regarding the safety of the teeth. I therefore think we are beginning at the wrong end when we look upon pyorrhœa as a primary cause of auto-intoxication, and in this contention I am supported by a friend who has also had considerable experience in similar cases. Dr. Hale White has stated that "some individuals are perfectly well if the bowels are only opened once a week." This is certainly quite opposed to my ascertained facts on the subject, nor can I conceive that two such opposing sets of circumstances can possibly co-exist. We all know how tolerant Nature is, and how unwilling she is to visit the penalty of disobedience upon her children, but it is contrary to her idea of discipline that this can go on for an unduly lengthened period, and retaliation will most assuredly eventually overtake and, what is of greater importance, destroy the delinquent.

The limit of time that should elapse between the eating of a meal and its passage through the intestines should not exceed eighteen to twenty-four hours, if a person is to retain his resisting power over disease. And I have no hesitation in affirming that every hour which is in excess of this is bound to interfere *pro rata* with the health of the individual and his or her resisting power to disease. This is a point to which I have given particular and prolonged attention, hence I am, I think, justified in drawing this conclusion. There are some people, however, who can accommodate themselves to, and appear to be happy in, any amount of filth, but because they seem to get on, apparently, fairly well in the circumstances which custom has habituated them to, it will invariably be found, in the long run, that custom does not ensure immunity. The fact is undeniable that a sanitary condition of the colon is essential to healthy physiological activity.

I trust it may not be considered out of place if I say it seems to me a little short-sighted to occupy so much time in endeavouring to name and classify the various bacteria which invade the intestinal canal, for, after all, they are very protean in their character; their appearance and potency, either for good or evil, depending largely upon their pabulum and environment. It is essential, therefore, that the diet should be so arranged as to encourage only the innocent, and not the malevolent form of these useful or pernicious organisms, seeing the form they assume depends so much upon the soil upon which they are cultivated. Moreover, the resulting products of decomposition, for which they are responsible, and which we call toxins, without doubt depend entirely upon the nature of the food supplied. It therefore should be one's aim to admit only such articles of diet into one's mouth, that the unutilized portion, when it finds its way into the colon, will give rise to the most innocent form of the products of decomposition. Now whole wheat meal bread contains, if anything, rather more than an equal amount of food units which is contained in the flesh of dead animals, and no one can, with truth, gainsay the fact that it is, at all events, a more suitable food for human beings, and, moreover, is much cleaner, as far as its ultimate condition in the colon is concerned, than butcher's meat is, and, therefore, while affording an equal amount of nourishment is much more suitable for dietetic purposes.

In this connexion I would venture to remind my hearers that man is the only animal which destroys the nutritive value of his food by cooking, and not only this, but also its beneficial effect upon the cells of one's body, for this reason, that, to enable them to retain their healthy vigour and resisting power to disease, they demand that pabulum be supplied to them in its natural condition. Cooking not only renders it more indigestible than uncooked food but favours constipation and the consequent evils which arise from it. No animal is so constituted that it is possible for it to derive full benefit from its diet if the vital principle, the nuclein, and the organic salts it contains in its natural condition are destroyed by heat. Indeed, the latter are quite as essential to the integrity of the blood as the other component parts of the ingested material, and they are generally washed away or become so altered in composition as to be rendered practically useless. Baked bread—and it is only whole wheat meal bread which I think is fit for human food—may be looked upon as an exception to this rule, but I maintain this, even, is neither so wholesome nor so nutritious as unfired bread, nor is it so easy of digestion.

We have only to examine the excreta of those living upon butcher's meat, boiled vegetables, and cooked fruit—at the same time taking particular note of the unsatisfactory condition of the evacuating power of the bowel, which, I may suggest, is invariably at fault in the circumstances—and compare the result of our observations with the information we gather from those who have confined themselves to a natural diet, and it will not be difficult to decide which is most compatible with a vigorous, healthy standard and the prospects of the continuance of this being assured.

Not only have various experiments proved, beyond the shadow of a doubt, that those living upon a natural diet are physically more fit than those subsisting upon the conventional diet of the present day, but also that their mental vigour is more favourably influenced. And it stands to reason this should be the case when we consider that the one is in conformity with our physiological capabilities and requirements, while the other is opposed to these.

No doubt the secretion of the thyroid gland, for a time, will tend to keep auto-toxæmia in check, but the period arrives when *its* functional activity and, therefore, its inhibitory power cease in consequence of the gland itself being rendered incapable of performing its duty, this being due to the toxic condition of its blood supply. It is then the subjective symptoms, which are characteristic of auto-toxæmia will become most pronounced—viz., a feeling of malaise, sleep, disturbed by dreams, generally of an unpleasant nature, followed by little or no refreshment in the morning, cold feet, a shivery sensation across the lower spinal region, bad taste in the morning, frontal headache, depression of spirits, irritability of temper, an undue sense of fatigue both of mind and body, &c. The objective symptoms are also easy of recognition and consist chiefly of a dusky complexion, accentuated by dark rims round the eyes, and a furred tongue.

In conclusion, I should like to state, as a matter of a fairly extensive experience, that I have never come across a solitary case of cancer in which the patient had not been a victim of chronic constipation, and I feel confident that cancer is not the only disease upon which an insanitary condition of the colon acts as a potent predisposing cause. How very great, then, is the importance, before arriving at a diagnosis, of making a careful examination of the patient's alvine discharges, a precaution, I fear, which is too frequently overlooked.

Dr. RALPH VINCENT: My remarks will be confined to the bacteriology of alimentary toxæmia in the infant and child. My classification of the organisms is directly related to the conditions determining their growth in and their action upon milk.

(1) Bacteria producing lactic acid, but not producing gas. This group is represented by the *Streptococcus lacticus* and the *Bacillus lacticus*. These are the typical organisms growing in pure milk incubated at 38° C.

(2) Bacteria producing lactic acid, carbon dioxide, and alcohol. This group embraces the whole of the colon organisms and includes the *Bacillus acidi lactici* (Hueppe), *Bacterium acidi lactici* (Esten), *Bacillus lactis aërogenes* (Escherich), and the *Bacillus coli communis*. When milk is incubated at 38° C. the growth of this group is inhibited by the growth of the organisms belonging to the first group. At 19° C. the colon organisms grow with comparative rapidity, as the growth of their antagonists is severely retarded by the low temperature.

(3) Bacteria growing in "Pasteurized" milk—i.e., milk that has been subjected to a temperature between 65° and 100° C. The products of these organisms are of a highly irritant and dangerous character. The *Bacillus putrificus* and the *Bacillus enteritidis sporogenes* are examples of this group.

(4) Bacteria growing in milk after it has been raised to 100° C. owing to the fact that their spores have not been destroyed. These organisms are found in milk that has been boiled or "sterilized" and in condensed milk. The group is an extensive one, the chief representatives being the *Bacillus subtilis*, the *Bacillus mesentericus* and the *Bacillus mesentericus vulgatus*. These organisms growing in the intestine produce powerful alkaloidal poisons and are responsible for the most fatal type of "summer diarrhœa" in infants.

The *Bacillus proteus vulgaris* does not belong to this group since it does not possess spores and is destroyed by boiling; but it is a widely distributed organism and is not infrequently found growing in boiled milk, having reached the milk after it has been boiled. In common with the other putrefactive organisms it is unable to grow in raw milk.[1]

With regard to the bacteriological analysis of the intestinal dejecta, three classes of organisms are commonly observed:—

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[1] For a fuller account of the organisms and their action *see* "The Nutrition of the Infant," 4th ed., Lond., 1913; and "Acute Intestinal Toxæmia in Infants," Lond., 1911.

(1) *Obligate Fermentative Bacteria.*—These cannot attack the proteins: they produce lactic acid by their fermentative action on the carbohydrates, and they do not produce gas. The *Streptococcus lacticus* and the *Bacillus lacticus* are the types of these organisms.

(2) *Facultative Fermentative Bacteria.*—The whole of the colon organisms belong to this class. In the absence of carbohydrates or in conditions unfavourable to carbohydrate fermentation these organisms become proteolytic. In their proteolytic capacity they are much more powerful than in their fermentative capacity. They invariably grow much more freely in peptone broth than they do in milk.

(3) *Obligate Proteolytic Bacteria.*—These have no action on the carbohydrates, but act entirely on the proteins. They can only act in the absence of carbohydrate fermentation, as they cannot grow in the presence of acid. They are not infrequently found in association with the colon organisms acting proteolytically.

In the healthy infant the biochemical processes occurring in the alimentary canal are largely controlled and determined by the *Streptococcus lacticus*. This organism is essentially dependent upon milk, is absolutely non-proteolytic, produces a moderate degree of acidity, and by its growth exercises an extremely powerful action in preventing the growth and action of all other organisms. In the lower part of the small intestine it does not grow so freely as in the upper part, while in the large intestine it, as a rule, does not grow at all. Corresponding with the cessation of activity on the part of the streptococcus the colon organisms come into action, but in health their action is fermentative.

The natural diet of the infant is the ideal diet in regard to its determining influence on the nature and behaviour of the intestinal flora. On such a diet the *Streptococcus lacticus* flourishes, and in consequence the growth and behaviour of other organisms is strictly controlled. In all cases of alimentary disorder in infants and children under my care, it has been my practice for some years to make systematic bacteriological examinations of the intestinal dejecta. The conditions found are very various, but they may be definitely arranged in two groups—the acid and the non-acid. In a case of diarrhœa, for example, it is of immediate importance to determine the group to which the organism belongs, for the treatment of the acid condition is wholly different from that of the non-acid condition. In a marked case of diarrhœa due to the acid-forming organisms the bacteriological picture is quite distinct. Diplococci are massed together in groups or lactobacilli are found. The latter organisms are distinguished by their containing granular bodies which are stained a brilliant red by methyl blue. In such a case the normal organisms have become superdominant, and the production of acid becomes excessive, with the result of an exhausting and irritating diarrhœa. The condition is one of pure irritation and there is nothing in the shape of alimentary toxæmia. Indeed, the condition is the opposite of toxæmia, for the antitoxic organisms are acting much too powerfully.

When an infant or young child is fed, as is so common at the present time, on a diet consisting entirely or almost entirely of cooked food, toxic conditions frequently develop; for the lactic organisms are delicate and they soon die out unless the infant continually receives a fresh supply. When cooked milk is given to an infant that has received raw milk the organisms in the cooked milk fail to develop, for the same reason that they fail to develop in raw milk. But when the infant is systematically deprived of raw milk the danger of their development is always present. As a rule, the first change is seen in the colon organisms. These cease to act fermentatively and become proteolytic. Once this process is established the lactic organisms are rapidly overwhelmed. While the colon organisms are usually the first to establish themselves this is not always the case, and even when these organisms have become dominant they may soon give way to more virulent organisms.

In the limited time at my disposal it is impossible adequately to discuss the bacteriological conditions, but I have selected three typical cases to illustrate some of the chief factors:—

(1) A child, aged 6, suffering from acute colitis. The dejections contained much mucus and bright red blood. The whole colon was acutely tender. The constitutional condition, considering the severity of the local signs, was good. The child was bright; there was no languor or drowsiness—an important point. The smell of the dejections (butyric acid), the severity of the colitis, and the absence of serious constitutional symptoms at once led me to think of the *Bacillus enteritidis sporogenes.* In my laboratory this diagnosis was established with certainty, for the organism was found in nearly pure culture. The child was immediately treated with frequent doses of the *Streptococcus lacticus* 52. The child made a complete recovery and was entirely free from any symptoms of the chronic colitis which so commonly follow the acute attack.

(2) An infant, aged 8 months, suffering from acute enteritis. "Quite unable to take milk." The bacteriological examination showed the

Bacillus putrificus and the *Bacillus acidi lactici* (proteolytic and virulent) to be the predominant organisms. The infant was treated with a modified *raw* milk and with mixed cultures of *Streptococcus lacticus* 54 and *Bacillus lacticus* 27. With these cultures the infant was able to digest the milk and speedily progressed to complete recovery.

(3) A child, aged 3. Enlarged spleen; enlarged liver. No appetite; great languor and weakness; pronounced anæmia. Quite unable to walk and only able to stand when supported. She had been seen by many physicians, for she had been ill for over two years, and the diagnosis for some time had been "splenic anæmia." It was a typical case of chronic colon toxæmia. The child was ordered raw milk and mixed cultures of the *Streptococcus lacticus* 52 and *Bacillus lacticus* 41. In three days the child's appetite was fully restored and she steadily progressed till she became quite normal. This case is peculiarly interesting because a few months ago this child was brought to me as she had relapsed and the old symptoms were beginning to reappear. On inquiry it was found that the parents had come to London and had obtained the milk from a company that systematically pasteurizes all its milk, while carefully concealing this fact from their customers' knowledge.

In conclusion, I would emphasize one point: The organisms commonly producing alimentary toxæmia in the infant and child are not specific pathogenic organisms. They are normal inhabitants of the alimentary canal, but in healthy conditions they are inert. Only when abnormal and artificial conditions of diet are introduced are they capable of coming into action.

Summary and Reply.

By W. Hale White, M.D.

It is obviously impossible to present, in a quarter of an hour, any adequate summary of a debate in which sixty speakers have taken part, but the speeches of the pathologists, chemists and bacteriologists have probably interested the Society most, for in an inquiry as to what clinical symptoms can with justice be attributed to alimentary toxæmia, the first thing is to understand how the poisons are formed, and how it is that they affect the body. There has been much said to show that it is not the formation of poisons which chiefly matters. Probably, they are continually being formed from the contents of the alimentary canal, or they might even occur as endotoxins derived from dead bacteria, but no harm follows so long as the constantly active defences of the body against such poisons are in full play. But the liver may fail us, the thyroid may fail us, mechanisms in the intestinal wall may fail us, or other unknown defences may fail, and then poisoning symptoms will occur. It will be a great step forward when, with the help of experimental pathologists, we are able to say which cases of supposed alimentary toxæmia are really caused by disease of the liver, disease of thyroid, or disease of some other organ as the case may be, or which are due to the formation of abnormal poisons or to an excess of normal poisons in the alimentary canal. Then, sometimes, it may be that the symptoms ascribed to alimentary toxæmia are really due, not to the formation of more poison, but to the fact that the wall of the bowel being diseased the poison is absorbed more readily than in health, or again, some alteration in the intestinal content may render rapid solution and consequent absorption of the poison easy.

Considering next the bacteria in the intestine, it is necessary to point out that if a person is poisoned by absorption from the intestine it does not follow that the poison is produced by bacteria, it may be that it is one of the normal results of the splitting of proteins, but that in all healthy subjects the poison is destroyed by the liver, which, however, is not performing this normal function in the person suffering from poisoning. We only know for certain that definite micro-organisms lead to poisoning when specific micro-organisms introduced from without cause specific diseases—e.g., in the case of typhoid, dysentery, cholera, and Gaertner poisoning; but many of those who have taken part in this discussion strongly suspect that under certain circumstances the micro-organisms constantly present in the alimentary canal lead to poisoning; but there is, I think, a consensus of opinion that the phrase "alimentary toxæmia" should be limited to the absorption of chemical poisons and should not include the passage into the blood of bacteria themselves, although this probably occurs more often than is usually thought. Which micro-organisms originate symptoms that are commonly included under the term "alimentary toxæmia" we do not know; even in comparatively simple cases, for example, we do not know the micro-organism which is the cause of enterogenous cyanosis, nor do we know whether, if the symptoms are not due to failure of protection against poisons, they are due to excessive number of bacteria, unusual supply of material for their growth, or absence of bacteria which are antagonistic to them; nor do we know whether the bacteria form the poisons from their medium, from their intrinsic metabolism, or from endotoxins liberated at their death. Although it has clearly appeared that we have a considerable knowledge of the bacterial flora of the human alimentary canal it must be confessed that this discussion has shed very little light on the bacteriology of alimentary toxæmia originating in the gastro-intestinal tract, but we know more about that of the symptoms which follow pyorrhœa alveolaris, although even here we often only surmise which is the offending organism by the rather unsatisfactory method of the opsonic index. Probably among the intestinal bacteria the *Bacillus aminophilus intestinalis* and its allies will most repay research, for much work has been done on them, but it certainly appears that many of the statements of the French school on the intestinal flora need serious revision.

As the subject of this debate has been alimentary toxæmia it was to be hoped that from it we should learn something definite as to the toxins concerned. Unfortunately we have not. It is well known that the view of the Metchnikoff school is that poisons are manufactured in the intestine, especially the large, by micro-organisms, and these poisons produce the symptoms commonly comprehended in the term alimentary toxæmia, and that indol is one of the most important of these poisons, but although that view has been upheld by some speakers it has been severely criticized. More than one of the authorities on bacteriology think it gratuitous to regard indol as the main toxic product of putrefactive organisms, and tell us that the conclusion drawn by Distaso that the flora of constipation is an index of intoxication is entirely unsupported by the evidence he adduces. I must confess it seems to me that the case for indol has not been proved and that there are serious defects in the work of the French school on the bacteriological and biochemical properties of the intestinal flora. If the debate has done no other good, it has helped to make widely known that much of the bacteriological and biochemical work which has been done upon the flora of the large intestine and the poisons found there does not satisfy the standards of scientific accuracy. But there is a general impression that the poisons of alimentary toxæmia may, in many instances, be comparatively simple chemical bodies derived from the proteins of the food. It is known that the ammonia present in the portal vein is derived from putrefaction in the large intestine. Tyrosine and histidine may be converted into poisonous substances by putrefactive bacteria, and experiments have been quoted which suggest that when certain of these poisons are absorbed arterio-sclerotic changes in the vessels and disease of the kidney may follow. Confirmation of these experiments will be looked forward to with great interest. Of the poisons formed in the alimentary canal probably the study of amines of tyrosine, leucine and histidine will most repay further investigation.

In the clinical part of the discussion the dental surgeons who contributed to it seemed to be on surer ground than other speakers. Disease of the gums leading to periodontal disease is by far the most fruitful source of oral sepsis. Large numbers of people owe ill-health to it, and it is often overlooked, and especially we are inclined to forget that mouth-breathing in children leads to persistent marginal gingivitis and that this disease is more intractable and serious in its results in those who are mouth-breathers, from which it follows that not only must the teeth be treated, but often it will be necessary to treat nasal obstruction also. Different speakers have laid a varying amount of stress on the use of autogenous vaccines. Probably the most important part of the treatment is local, and here much judgment is required; some remove teeth needlessly, some do not remove enough of them, but in the more severe examples the intelligent use of autogenous vaccines may be of distinct help. The question has been raised as to whether in a few cases pyorrhœa alveolaris is not secondary to a toxæmia arising from some other parts of the alimentary tract. It is quite possible that this may be so or, at any rate, that such local pyorrhœa is increased by the fact that the general resistance of the body is lowered by an alimentary toxæmia of other origin, but obviously the question can only be settled by a discussion of each case in which it may be raised, and such suggestions may do harm if they lead to a widespread belief that the local treatment of the disease of the gums is unimportant. Mention has been made of the many ways in which it is conceivably possible that pyorrhœa alveolaris might affect the general health, and it is clear that the opinion of most speakers is that nearly always it is by direct absorption from the diseased gums.

It appears that in this country not much attention has been paid to the relationship between alimentary toxæmia and diseases of the eye, but this discussion has been of great use in directing attention to the question. Certainly some diseases of the eye may be due to pyorrhœa alveolaris; what other diseases of the eye are due to other forms of alimentary toxæmia can only be decided by future clinical observation, which avoids the error of ascribing changes to alimentary toxæmia.

because no other cause can be found; but the extremely interesting question as to whether failures of accommodative power may not often be due to intestinal toxæmia needs further investigation. If the remarkable figures quoted by one speaker are confirmed a great advance in our knowledge will have taken place.

It is generally allowed that the mere absorption of a poison—e.g., morphine—from the alimentary canal is not enough to constitute alimentary toxæmia, and the striking urticaria which follows the ingestion of shellfish, copaiba, &c., in some persons is most likely an example of anaphylactic phenomena following the absorption of foreign proteins and therefore not an instance of alimentary toxæmia. The purpura often associated with cirrhosis of the liver is probably an alimentary toxæmia in which the poison absorbed from the intestine is able to act because the protective influence of the liver is, owing to its disease, in abeyance. Henoch's purpura may be an example of alimentary toxæmia, but we really know nothing of its pathology. The dark blue tint of the skin in enterogenous cyanosis is almost certainly due to alimentary toxæmia, and so may be the brown cutaneous pigment so often seen in those who have disease of the intestines. It seems as though increase of knowledge will show that several abnormalities of the skin may be ascribed to toxins formed in the alimentary canal.

We now pass to the most controversial part of the subject. It has been urged by several speakers that in consequence of the erect posture of man the intestines tend to drop, that to overcome this peritoneal bands are evolved, that they produce kinks, that these lead to stagnation of the intestinal contents, this leads to increased putrefaction, this to the formation of poisons, and that these, passing into the general circulation, cause definite symptoms and predispose to many diseases. Observers are by no means agreed as to the frequency with which these bands are found, nor, it is said, are they evolved as a result of the erect posture of an individual, because they may be found in the human fœtus and are in reality mere expressions of a normal and healthy fœtal process. The question will, it seems to me, have to be solved by anatomists. Are these bands to be found in all animals in whom the body is usually vertical—e.g., apes, gibbons, and penguins? Are there evidences of them in animals whose bodies approach the vertical—e.g., giraffes? Are there what might be called reverse bands in bats that spend so much of their time head downwards? Here I might anticipate and point out that if these bands are present in all animals that adopt the erect attitude it would be of great interest to know whether such animals have intestinal stasis.

Passing to the question of kinks, it seems, from listening to this debate, that those who have laid great stress on an ileal kink as a cause of intestinal stasis have not estimated at its full value the strength and activity of the ileo-cæcal sphincter, and that much of the supposed holding back of the intestinal contents at the end of the ileum is normal and that when abnormal it is often due to spasm of the ileo-cæcal muscle.

Many of the statements about kinks and stasis claim support from X-ray examinations after bismuth or barium sulphate, but we have been told that sometimes food is given too soon after a bismuth meal when we wish to know if there is gastric stasis; that sometimes so much bismuth is given that its weight may invalidate our observations; that allowance is not made for normal variations in the average time taken in the passage of a bismuth meal, anything differing from the average being taken as abnormal; that sometimes the bismuth mixture lags in the stomach because it is nasty, although in the same patient it will leave the stomach normally if it is palatable; that the so-called writhing of the duodenum is nothing else than the normal peristalsis and segmentation rendered more clearly visible than usual by the great quantity of bismuth-containing chyme present; that the X-rays do not show the duodenum to be dilated in cases of duodenal ulcer, and that many of the kinks which the X-rays are supposed to show depend upon the fact that the X-ray picture is taken in a single plane. It is clear that as the X-ray work on this subject has been so severely criticized it will have to be repeated by independent observers before we can decide upon its value.

Those who believe that evolutionary peritoneal bands often lead to intestinal stasis because they produce kinks, especially a kink at the last part of the ileum, think that, as a further result a kink forms at the end of the duodenum, this and the stomach dilate and either may become ulcerated. This debate has not provided any statistical evidence to show in what proportion of sufferers from duodenal or gastric ulcers such kinks are found, nor has any experimental evidence been brought forward to show that in animals such ulcers form as a result of an artificially induced ileal kink, and we have heard one speaker say that intestinal stasis never leads to gastric stasis, nor to duodenal kinking, dilatation or ulceration, and that duodenal ulcers are associated with a usually rapid passage of chyme out of the stomach and through the whole of the small intestine. It seems that the onus of proving their contention lies with those who believe that the above-mentioned phenomena follow intestinal stasis.

I do not think that in a short summary such as this anything can with advantage be said about the more remote effects—e.g., tubercle, cancer, rheumatoid arthritis, arterio-sclerosis, disease of the heart and kidney which have been ascribed to intestinal stasis, for, up to the present, no statistical or experimental evidence has been brought forward for such a view. It may turn out to be correct. I hope it will, for I should like to see such an extremely brilliant suggestion shown to be true, but up to the present no adequate evidence has been brought forward for this supposition, which, if it is to be accepted, must have in its favour, not one or two isolated cases or a mere expression of opinion, but a large mass of incontrovertible experiments and statistics adduced by more than one observer. But whether or not future observation proves its truth, there is no doubt that we owe a debt to the author of it, for, if it is correct, it marks a new era in medical thought, and even if the evidence for it fails, it has made us think hard.

Nor do I think that time allows me to say much about treatment. We have seen that the ways by which intestinal poisons may be formed are so various, and the faults of our mechanism which may allow them to get into our bodies are so numerous, that proper treatment is in our present ignorance often impossible; indeed, almost our only treatment, surgical or medical, is the very primitive plan of keeping the bowels well cleared out. Sometimes, indeed, Nature does that for us, for, as has been pointed out, severe alimentary toxæmia may exist with a diarrhœa which keeps the intestines almost empty. On the other hand, many people whose bowels are rarely open have not alimentary toxæmia. It is agreed that in the vast majority of cases medical treatment suffices, and what has been said about treatment in this discussion has come chiefly from the surgeons. Removing the colon has been mentioned, but a surgeon of wide experience tells us he has never seen a case in which he considered it justifiable. Short-circuiting the ileum into the sigmoid, too, has been discussed. Some say it succeeds, but it is clear it often fails to cure, and some surgeons prefer the simpler operation of appendicostomy. But if the cases that now seem to some to justify surgical treatment had been treated in the first stages by proper medical means, surgical interference would not be necessary; so that when this is widely appreciated, cases ought never to become so severe that surgical treatment is contemplated, and we may hope that one result of this discussion will be that we shall keep "the drainage scheme" of our patients in sufficiently good order as to render surgical interference unnecessary.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Section of Anæsthetics.

November 1, 1912.

Dr. J. **Blumfeld**, President of the Section, in the Chair.

Death during Hedonal Infusion Anæsthesia.

By G. A. H. Barton, M.D.

In asking permission to report this case to the Section, I was impelled by a sense, not so much of the importance of the case itself as of the importance of thoroughly ventilating all fatalities and misadventures that may occur during the trial of a new anæsthetic. It is during this period of its career that the virtues of an anæsthetic are apt to be overestimated. One reads in the journals more or less lengthy lists of successful cases. Misadventures or partial failures are minimized. Deaths are not always reported, and if they are, do not loom so largely in the columns of the medical Press. One hears vague rumours of unreported fatalities. These considerations have led me, then, to report this case in the hope that during the discussion following something tangible may be elicited respecting the safety of hedonal as an anæsthetic.

So far as I can gather, from a somewhat cursory examination of the journals, this makes the third reported case of a fatality after hedonal in this country; the others being one reported to this Section by Mr. Barrington Ward,[1] and one in the *Lancet*[2] by Messrs. Upcott and Evans, of Hull. In addition, however, I find, in looking through Mr. Page's work, that in one of his cases death was attributed to diabetic coma, which may quite possibly have been precipitated by hedonal, and that he mentions a fatality after gastrostomy in an old man. These misadventures are few, but as they have occurred in a very short period, I think they should rather give pause to the enthusiasm of those who, having been more fortunate, extol hedonal as a perfectly safe and ideal anæsthetic.

The case I now report was the tenth and last of my series. In itself I may say that it proves nothing except that great care should be exercised in the selection of cases for this method. The other cases were fairly uneventful, except that one took twelve hours in recovering consciousness, and during that time gave rise to some anxiety owing to the extraordinary rapidity of the pulse; and another case exhibited, for two days, the rise of temperature which I believe is now associated with the injection of a saline solution contaminated with the toxins of dead bacteria.

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[1] *Proceedings*, 1912, v, p. 93.

[2] *Lancet*, 1912, i, p. 1668.

The patient was a male, aged 43, and was operated upon on June 4, 1912, by Mr. Hope at the Throat Hospital, Golden Square. The operation was for double frontal sinusitis. He was a tailor who had recently been on strike. He looked underfed, and his physique was below the average. He was admitted to hospital the day before operation; during the brief period he was under observation his temperature was normal, and he had no cough or dyspnœa, and made no complaint of the same. In the superficial examination of the heart and lungs that I made I observed nothing of importance. I particularly mention these facts in view of the post-mortem findings. Had the condition of his lungs been obvious I should not have employed hedonal as an anaesthetic, owing to its depressing influence on the respiratory centre. An hour previous to operation he had had an injection of morphia $\frac{1}{6}$ gr. combined with a little atropine. This had been ordered by the house surgeon under the impression that it was the usual routine in intravenous anaesthesia. He was not at all drowsy, and having regard to the fact that a previous alkaloidal injection had been deliberately resorted to by Mr. Page in a few cases, I saw no reason to withhold hedonal on this account. The usual 0·75 per cent. solution of hedonal was used, and after the infusion of something approaching 400 c.c. in three and a half minutes he appeared to be under. The corneal reflex was sluggish. The flow was slowed down, and by the end of five minutes he had had nearly 500 c.c. and the operation commenced. The induction was perfectly quiet and some little blueness was noted. Thereafter anaesthesia was maintained with the greatest ease by a drop flow of the slowest possible rate during an operation lasting one and a half hours, a further 500 c.c. being infused in this period. The patient was well relaxed, his pulse was good, about 100 to 120; respirations were those of deep sleep. His pupil was contracted, and his corneal reflex, so far as it could be observed during an operation of this nature, was very sluggish or absent. His colour never quite recovered from the slight blueness which is not uncommonly observed during the induction stage of hedonal anaesthesia. I felt a little anxious about this, and held the tongue forward most of the time. My opinion was, however, that it was due to the combination of morphia and hedonal, with the added embarrassment of respiration caused by a post-nasal plug and a folded towel, placed over the mouth to prevent the blood that trickled freely down his face from flowing into it. This towel was more or less saturated with blood during the whole operation, and had to be changed once or twice, but I am quite sure no blood entered the pharynx by either the nasal or oral route during the operation. At its termination the post-nasal plug was removed, and I administered a little oxygen, which quickly restored him to an excellent colour. I left the hospital shortly afterwards at 12.15, feeling not the slightest anxiety about him.

About 1.20 the house surgeon was called to him and noted that his pulse was somewhat weak. Instructions were given to swab out his throat in case any blood was trickling down through the posterior nares. The swabs were, however, reported to come back fairly clean. He was seen again at 1.45, and died at 2.30, having apparently slowly sunk with no very obvious symptoms and without regaining consciousness.

Dr. Trevor will read the notes of the post-mortem, but I may just say that I am informed there were certainly neither cough, râles, nor increased respiratory effort to indicate the aspiration of the large amount of blood which was found in his tubes post mortem. My opinion is that in moving him from the operation table to the stretcher, and thence to bed, some considerable hæmorrhage was started and continued perhaps slowly thereafter, and being aspirated into his lungs, already *hors de combat* with miliary tuberculosis, slowly asphyxiated him, death being contributed to by the depressing effect of hedonal on the respiration and laryngeal reflex.

The obvious lessons I learn from this case are: (1) Never to give hedonal in presence of lung disease; in this case, unfortunately, there was no indication of its presence previous to the autopsy. (2) Never to give hedonal when there is the remotest chance of blood finding its way into the air passages, either during or after operation; under hedonal it is aspirated apparently without the slightest resentment on the part of the patient.

I am conscious that there are many points about this case that may arouse criticism. That, however, I will not anticipate. I have reported the case in the hope of eliciting further information and some expression of opinion as to the safety of hedonal in general, and the contra-indications to its use.

I have to thank Mr. Hope for permission to report the case, and Dr. Trevor for kindly supplying the full notes of the autopsy.

POST-MORTEM REPORT BY DR. R. S. TREVOR.

The body is spare. There is a recent operation wound for operation on the frontal sinuses. The sinuses communicate with one another and with the nose. Drainage-tubes in situ.

On internal examination: There are adhesions all over the right lung and at the left apex.

Lungs: Right, 1 lb. 12 oz.; left, 1 lb. 8 oz.; both are riddled with miliary tubercle; in both there is old fibro-caseous tubercle with a fair-sized cavity at the right apex; both lungs slightly mottled as the result of the inspiration of blood. The bronchial and infratracheal glands are tuberculous.

Air passages: The trachea is almost filled with blood and mucus; the blood is not frothy and it extends into both main bronchi; blood could be expressed from some of the smaller bronchi within the lung. Larynx free from ulceration; thyroid natural; pericardium natural; heart 9 oz.; the muscle shows marked cloudiness and is soft; there is no valvular disease; no embolism; the aorta shows a few atheromatous patches.

Abdomen: The mesenteric glands show many caseating areas; liver (2 lb. 11 oz.), spleen (7 oz.), and kidneys (5 oz.) are deeply congested; no obvious tubercles visible to naked eye; pancreas and adrenals natural; bladder normal.

Alimentary canal: The stomach contains a small quantity of blood; the small intestine shows many tuberculous ulcers; large gut natural.

Cranium: Brain (3 lb. 9 oz.), natural; no thrombosis of veins at site of infection.

DISCUSSION.

DR. R. S. TREVOR said that in cases dying during anæsthesia post-mortem examinations were made under circumstances which made it somewhat difficult to arrive at a conclusion as to the real cause of death. In this case he attributed death to heart failure while the man was suffering from the toxæmia of tuberculosis, such death being hastened by the administration of hedonal, and by interference with respiration by bleeding into the trachea. As it turned out, from the post mortem findings, the man was anything but a fit subject for hedonal; he was very far gone with pulmonary tubercle, and had suffered from privations and lack of food during the strike. The effect of the anæsthetic was to annul his respiratory centre, and the accumulation of blood in his air passages produced slow asphyxia. The blood in the trachea was not frothy, and did not seem to be much churned up; some of it was probably of post-mortem origin. The lungs, however, contained inspired blood, and were mottled, presenting the appearances known as "tigroid" lung. With some hesitation he would say that if there was the slightest risk of bleeding into the air passages, hedonal was not an ideal anæsthetic, unless great care could be taken to watch the patient afterwards.

Dr. Z. MENNELL said he had had considerable experience in the use of the drug, having administered it 196 times. At St. Thomas's Hospital it had been used a great many times: Mr. Page's original cases numbered 200, since when there had been a further 300, and he had had 56 in addition for cerebral surgery. Of those cases there had not been one death within the first twelve hours of the operation being completed. Mr. Page and he had formulated what they believed to be contra-indications, and these he had described at the meeting of the Medical Society a few days ago. He believed Dr. Barton's case fell under one of those classes. The indications against the use of hedonal were, first, any operation which would result in bleeding into the larynx or trachea. The prolonged sleep after the operation when the drug was used was in many cases beneficial, but where there was blood in the mouth it was a most distinct danger, as the laryngeal reflex did not return until quite a late period. The second contra-indication was where there was high blood-pressure, as those cases required an enormous quantity of the drug to make them anæsthetic. The third class were cases in which anæsthesia could be obtained as safely and satisfactorily by other means. They did not now use hedonal as a routine anæsthetic at St. Thomas's Hospital. There were many cases in which hedonal was extremely useful, and especially cases of cerebral surgery, for which it was practically ideal. In these there were many points of benefit to surgeon, anæsthetist, and patient; the anæsthetist was out of the way, any posture in the patient could be used, provided there was a free air-way; there was no initial fall of blood-pressure, as in the case of chloroform, the operation could be continued longer, and the necessity of a second attempt was often avoided. There was no vomiting, which was important when the head had to be kept in sandbags. Finally, it was easy to give an additional saline infusion without any further apparatus or incision. Of 123 cases of intracranial surgery, hedonal was given in 43. The deaths within the first forty-eight hours were as follows: with chloroform 20 per cent., with ether 13·6 per cent., and with hedonal 3·2 per cent. There were more hedonal cases than ether cases, chloroform preponderating. He would not have given hedonal for Dr. Barton's case, because of the trouble in the air passages, and he thought that 400 c.c. in the first three and a half minutes was much too rapid a rate; he did not now exceed 100 c.c. every two or three minutes. The anæsthesia did not take long to develop, as consciousness was usually lost after 200 c.c. or 300 c.c. had been given. Rapid administration resulted in cyanosis, which he looked upon as the gravest danger-signal. He had given hedonal to a goat which took enormous quantities in proportion to its body-weight; it became cyanosed while the pulse remained quite good, and its death was obviously due to failure of the respiratory centre. Chest disease he did not regard as necessarily a contra-indication, and the corneal reflex was absolutely valueless; the only indications he went by were the skin reflex and the respiration. The skin reflex should never be abolished. If, as he did, one ran the infusion through the internal saphenous vein, it was easy to elicit the skin reflex by stroking the sole of the foot with the finger, or a pin: the result was a movement of the toe and a drawing up of the leg. If the case was not a cerebral one, another good reflex was, that on pinching the lobe of the ear the patient would slowly roll his head over to the opposite side. Dr. Barton mentioned that in his case the tongue fell back, and the jaw had to be pulled forward. At first this also occurred frequently, with cyanosis, in his own cases, but in the last thirty or forty cases it did not occur, as smaller doses of the drug were given. It was possible to maintain a brisk skin reflex and yet abolish all consciousness and all muscular rigidity. Although the patient moved so much when the skin incision was made (sometimes he had even to be held down on to the table) yet the abdominal muscles were quite lax. He could only compare that flaccidity with a satisfactory spinal anæsthesia. He could speak feelingly as to the necessity of avoiding hedonal in cases where blood ran into the larynx, as he had himself had a death within eight hours after an operation in one of his earlier cases. The patient was an elderly man, who had always been very ill after taking a general anæsthetic, and he asked for hedonal, as he had heard of it. His blood-pressure was high, and he took a large quantity of the drug, 900 c.c., before the surgeon could begin. Enucleation of the tongue was done for malignant disease, and he did not think due care was exercised in tying the vessels, as there was a great deal of hæmorrhage. Post mortem a clot 3 in. or 4 in. long was removed from the larynx. The cases in which hedonal was useful were: (1) Cerebral cases; (2) operations about the neck which did not result in opening the air passages; (3) cases where vomiting was a great disadvantage, for instance, a large ventral hernia, and cases of goitre and Graves's disease. He had had six most successful cases. In one he gave only 240 c.c., after two doses of ¼ gr. of morphia at intervals.

Dr. SILK wished again to record his opinion that the dose of hedonal used was much greater than was at all safe. He understood hedonal to be one of the urethane group, allied to veronal, and the dose of those drugs by the mouth was 15 gr. to 30 gr. As far as he could gather, the average amount of the solution given intravenously for an ordinary case was about 1,200 c.c. The strength of the solution employed was 0·75 per cent., and so 1,200 c.c. represented considerably over 2 dr.: anybody who gave a couple of drachms of veronal by the mouth would be considered a very rash person, and he was not sure that giving that amount via the vein was not equally rash. His own experiences with the drug did not compare in point of numbers with Dr. Mennell's, but in such cases as he had given it he had always found the same trouble, there was a difficulty in giving sufficient to get the patient under, while not giving too much. His patients who had hedonal had usually slept, not only the night through, but the following day also, and he had concluded, therefore, that it was a mistake to give such a large dose. The alternative was that suggested by Dr. Mennell—i.e., not to produce anæsthesia in the sense meant by the old-fashioned anæsthetist, but only such a degree that the patient could kick about. Dr. Silk certainly did not regard such a condition as satisfactory, and he was very doubtful whether the majority of operating surgeons would be very pleased with it. Dr. Mennell spoke of finding the abdominal muscles relaxed as though it were a novel experience, yet one could get them relaxed in the ordinary way, and he hoped, therefore, people would be a little careful in their use of hedonal. Dr. Barton referred to three cases of death under this anæsthetic, but he (Dr. Silk) felt sure there were more. He did not doubt that hedonal might be useful as an adjunct to intravenous ether, and he had frequently employed it himself for that purpose, but he did not consider that the drug given by itself in the way Dr. Mennell used it was at all desirable. Mr. Page read a paper before the Section last session, the gist of which was, as Dr. Silk understood it, that hedonal was a very desirable anæsthetic in nearly every class of case, whereas the present advocacy really limited its use to cerebral surgery, and put forward the view that it was contra-indicated "where another anæsthetic could be used with equal advantage," which, of course, would cover a large proportion of one's practice. Moreover, as cerebral surgery was often the final resort in desperate conditions, the use of hedonal was, perhaps, less open to objection.

Mr. J. D. MORTIMER reminded the Section that Dr. Barton published a short account of his case shortly after it occurred, and he thought it somewhat regrettable that such was not a more usual practice when a new method was on its trial. As Dr. Silk said, it was probable that there had been several deaths from the administration of the drug, which had not been reported; he was aware of two himself which were not included in those mentioned on the present occasion, nor, so far as he knew, had they been published, although they took place several months ago. What one wanted to know as soon as possible was, not so much the best about any new method as the worst about it.

Dr. BARTON, in reply, expressed his regret that his paper had not been more responded to by the relation of experiences. Dr. Trevor had rightly said the case was not one in which hedonal should have been given: and had he known the state of the lungs and the effect on the laryngeal reflex produced by the drug he would not have used it. He congratulated Dr. Mennell on the success with which he had used the drug: it was somewhat discounted, however, by the death which had occurred; he did not think it would suffice to tempt him to use hedonal with any frequency in the future. He agreed with Dr. Mennell's contra-indication—viz., where there was bleeding into the air passages—but he was not aware that it was not a suitable anæsthetic in cases of high blood-pressure. When he left the hospital after giving it in this case his rough note was, that this was the most perfect anæsthetic he had given, yet the result was far from justifying such an opinion. With regard to the rapidity of the administration during induction, it was not quite 400 c.c. which were given, as he always introduced the needle with the solution flowing through, and fully 50 c.c. were generally lost before getting the needle in. The rate was about 100 c.c. per minute, the rate originally laid down by Mr. Page. He did not agree with Dr. Mennell as to the corneal reflex; he considered it important, in this respect, that it should *never* be abolished: if one gave hedonal until the corneal reflex was abolished, the patient was in danger. If the skin reflex also must be present, he thought there would be many movements going on, and he agreed with Dr. Silk that the surgeon would not appreciate the light sort of anæsthesia resulting from this reduction of the dose. Among his other ten cases there were three or four in which the patients were so lively that it was necessary to give a little C.E. mixture to keep them reasonably quiet. One of them was a plethoric woman, weighing about 16 st., to whom he gave 1,050 c.c. in nineteen minutes, and she had not gone under then. He had always given as little as possible so long as it satisfied the surgeon; he turned off the tap until it all but stopped the flow. Possibly if, in this case, he had had some arrangement by which, when the patient was under, he could have been injecting saline instead of hedonal, the result might have been different. He quite agreed with Dr. Mortimer that all such deaths should be recorded.

Demonstration of New Apparatus for the Intratracheal Insufflation of Ether.

By H. E. G. Boyle and G. E. Gask, F.R.C.S.

Mr. Boyle: The subject of intratracheal insufflation of ether has recently been dealt with in this country by Dr. Ehrenfried and Dr. Kelly[1] at Liverpool, and previously to that papers appeared in the Studies from the Rockefeller Institute for Medical Research, by S. J. Meltzer[2] and John Auer, who had been using this method on animals. To Dr. Chas. A. Elsberg, however, belongs the credit for having first successfully adapted the method to the human subject.

I do not propose to-night to enter into an elaborate description of the method, or to discuss its merits; the number of cases that I have anæsthetized by this method up to the present is not enough to enable me to make any statements that would be of great value; and so I shall content myself with showing you our apparatus, and pointing out a few of the main features of the method. I hope later on when I have completed a larger series of cases to be permitted to give you an account of the results.

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[1] *Brit. Med. Journ.*, 1912, ii, pp. 616, 617.

[2] *Med. Record*, New York, 1910, lxxvii, p. 477; *Journ. Amer. Med. Assoc.*, Chicago, 1911, lvi, pp. 521-25, and Studies from Rockefeller Inst., New York, 1912, Reprints, No. 19.

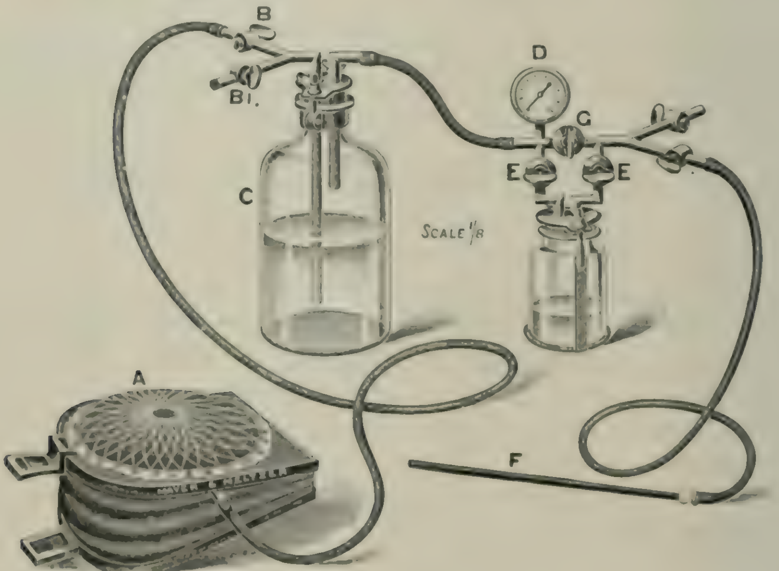
The principle of the method is the blowing of air laden with ether vapour into the trachea at about its bifurcation—and the expiration, or perhaps it would be better to say blowing out of the air, through the trachea. It is possible by doing this for enough ether to be absorbed to produce anæsthesia, and at the same time by regulating the rate of entry of the air to permit the lung to become collapsed, and subsequently at the finish of the operation by increasing the pressure to dilate the lung again whilst the pleura is sewn up.

My attention was first called to this method of maintaining anæsthesia by my colleague on the surgical staff of St. Bartholomew's Hospital, Mr. Gask, who early this year approached me on the subject, and asked if I was prepared to anæsthetize patients for intrathoracic operations. This appeared to me to be rather a new departure, but I soon found on reading the papers of Meltzer, Auer and Elsberg, that it was possible to maintain anæsthesia for these cases with safety, and so I determined to do what they had done.

My next discovery was that there was an Elsberg apparatus at King's College Hospital, and through the courtesy of my friend Dr. Silk we were enabled to see it, and Dr. Silk kindly explained its working to us. Mr. Gask and I were both agreed that the Elsberg apparatus was rather too large and complicated to take about, so we determined to devise a simpler and more portable apparatus which I have here to show you to-night. On March 7, of this year, I attempted my first case, and with your permission I will read you my note made at the end of the operation.

Woman, aged 50; exploratory laparatomy (by Mr. G. E. Gask). Operation: Removal of large multilocular ovarian cyst, which was adherent to intestines and anterior abdominal wall. Patient anæsthetized with gas and ether, ether continued with Clover's inhaler until the cyst was removed—twenty minutes. Mr. Harmer easily passed a No. 12 gum-elastic catheter into the trachea. The end of the catheter protruded 1 in. from the incisor teeth; to this end the exit tube of the insufflation apparatus was attached by rubber tubing, and air laden with ether vapour was pumped into the trachea. The breathing remained deep and automatic, the colour was pink, and the pupils of moderate size, no conjunctival or corneal reflex being present. Anæsthesia was continued by this method, all the air driven into the trachea being charged with ether for fifteen minutes, then the taps were so arranged that half the air passed over the ether and the other half straight to the trachea. This was continued for five minutes, then air free from ether was blown into the trachea for five minutes, when the patient coughed, and the catheter was withdrawn. The patient now had swallowing movements, and the corneal and conjunctival reflexes were both present and of a brisk character. Insufflation lasted for twenty minutes and the anaesthesia was very good. The pressure of air, as measured by the manometer, varied from 2 mm. to 15 mm. of mercury, and once, for a few seconds, it was 23 mm., owing to imperfect bellows.

Since then I have used this method in twenty cases. My chief difficulty has been the introduction of the catheter, but I am quite sure that this will be easily overcome with practice, for I have noticed on the occasions when I have been able to obtain the assistance of our Senior Throat Surgeon, Mr. Harmer, that he slips it in with the greatest ease. The anaesthesia is distinctly good, though comparatively light. The colour is rosy-pink. I have not so far observed that cessation of respiratory movement that has been spoken of by other observers; all my patients have continued to show respiratory movements, although these are less marked than when the ordinary methods of inhalation anaesthesia are being employed. The return to consciousness is fairly rapid, and can be hastened by blowing pure air into the trachea for a few minutes. Up to the present in my few cases there have been no unpleasant after-effects, and I have been rather surprised to find



that there have been no sore throats or bronchitis following the administration.

The apparatus as you see consists of a foot-bellows, two bottles, one for hot water and one for ether, a manometer, and some tubing. It has been made for us by Messrs. Mayer and Meltzer, to whom our thanks are due for the trouble they have taken to carry out our wishes. Finally, my thanks are due to those members of the staff of St. Bartholomew's Hospital who permitted me to use the method on their patients, and also to Mr. T. P. Legg for allowing me to try it in a case of goitre at the Royal Free Hospital.

Mr. GASK said his interest in the method was in that it enabled one to perform operations within the thoracic cavity. The first thing in operating on the thorax was to find a means of continuing aeration of the blood in cases where it was necessary to allow both lungs to collapse, such as in double pneumothorax. The apparatus was due to Auer and Meltzer, for they showed that in fully curarized dogs—i.e., dogs in which the muscular action was abolished—they could, by this method, maintain good aeration of the blood, although the respiratory movements were abolished. Then it seemed easy to attach a chamber for ether, to allow of the administration of an anæsthetic at the same time. Mr. Boyle had helped him to put the apparatus together. It had been used for a number of cases of all kinds, and a satisfactory anæsthesia could be induced by that means. It had been used where the thorax had been opened on one side only, but it had not yet been put to the supreme test in a case in which double pneumothorax was present; he had sufficient confidence in it to try it when a case demanding such an operation occurred.

Apparatus for the Intratracheal Insufflation of Ether.

By FRANCIS E. SHIPWAY, M.D.

I HAD the opportunity in August of this year of seeing Mr. R. E. Kelly use his first model, which he demonstrated at the Liverpool meeting of the British Medical Association.[1] He was anxious to have a portable apparatus made—I now show the improved apparatus which Messrs. Down Bros. have built, working on his lines.

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[1] *Brit. Med. Journ.*, 1912, ii, p. 617.

The apparatus consists essentially of two separate parts: (1) A one-eighth horse-power electric motor driving a "Hypress" blower at various speeds; (2) the air-ether segment. The motor and blower are contained in a closed box, which can be placed on the floor of the theatre or of an adjoining room, or on the lower shelf of an anæsthetist's table below the air-ether segment. There is ample reserve power with this motor, and it may be possible to substitute air with smaller horse-power, with the advantages of lessened weight and cost. There is a regulating handle on the inlet tube of the air-ether segment for the control of the pressure; it is a convenience in practice to be able to regulate the pressure independently of the motor, which can be set to run at a uniform speed. A foot-bellows is provided in case of a breakdown of the motor, or for use when electric current is not available. The air-ether part consists of: (1) A copper jacket containing a three-necked Wolff bottle, surrounded by a coil of lead tubing and a metal chamber for moistened gauze; (2) a mercury safety-valve; (3) a mercury manometer; (4) a thermometer. There is an attachment at the beginning of the air-inlet tube for connexion with an oxygen cylinder, and at the delivery end of the apparatus is a two-way tap by which the pressure can be reduced at intervals to zero. When the apparatus is to be used, the Wolff bottle is lifted out of the copper jacket, and partly filled with ether; hot water at about 140° F. is poured into the jacket, and a piece of moistened sterile gauze is placed in the metal chamber. By means of a tap gearing with two lateral taps, the air current can be made to miss the ether or pass over it in any proportion; the air, etherized or not, then passes through the circuit, and on its way is warmed, moistened, and filtered by the means described above. The pressure can be read, and the temperature (usually 90° to 92° F.) noted. The safety-valve is adjusted to blow off at a maximum pressure of 50 mm. Hg., and thus acts as a safeguard against any sudden and dangerous increase of intrathoracic pressure.

I have found some difficulty in introducing the tracheal catheter. I have tried the Cotton-Boothby introducer, but am not altogether satisfied that it is easy to use, and think that direct laryngoscopy is the best method unless some form of introducer can be devised which will render the passing of the catheter a simple operation.

This apparatus has so far been used in only a few cases, but it has proved to be in these efficient and simple.

Section of Anæsthetics.

December 6, 1912.

Dr. J. BLUMFELD, President of the Section, in the Chair.

Notes on the Administration of Anæsthetics in America, with Special Reference to the Practice at the Mayo Clinic.

By Mrs. DICKINSON BERRY, M.D.

I VENTURE to bring before you to-night a few observations on the administration of anæsthetics for various operations which I witnessed during a recent tour in America. I fear that I have little to present which will be new to the members of the Section, but I hope that my remarks will lead to a discussion in which others who have experience of the other side of the Atlantic will take part.

My notes may be divided into two parts—first, those referring to a cursory glimpse of some of the hospitals in New York, Boston, Chicago, and Minneapolis; and secondly, those made during a longer stay at Rochester, Minnesota, admitting of a more detailed study of the work in the Mayo clinic. My visit to the United States took place in July, consequently many of the principal surgeons were absent on vacation, but that did not greatly affect my special point of view, as hospital work and the administration of anæsthetics went on as usual. At New York I witnessed operations at several hospitals. The first was at the Skin and Cancer Hospital, where Dr. Gwathmey gave the anæsthetic for a severe abdominal operation, lasting an hour and a half. Dr. Gwathmey is President of a recently formed Society of Anæsthetists and is anæsthetist to the hospital. He used his own "Three Bottle Vapour Inhaler." The three bottles in the apparatus contain ether, chloroform, and warm water. By means of a stopcock air can be given alone, mixed with ether or chloroform, or both. The air is pumped in by a foot-pump and the vapour is warmed by passing through the warm water before reaching the facepiece. The anæsthesia on this occasion was light throughout, but was quite sufficient; the patient, a feeble subject, remained in an excellent condition through the whole operation. During the latter part oxygen was given instead of air, the oxygen cylinder being connected with the apparatus in place of the air-pump.

At the New York Hospital, one of the oldest institutions in New York, displaying in its hall a charter of George III, I witnessed an operation under ether given intratracheally. The apparatus used was Dr. Janeway's, which is driven by electricity. The working is much the same as that of the apparatus shown to the Section at its last meeting, but the machine is perhaps slightly less cumbrous. Induction was carried out with ether in the ordinary way, and the tube was then passed through the larynx without difficulty. The anæsthesia was quite satisfactory, and the patient remained an excellent colour throughout the operation. This is a method which, I gathered, is used a good deal at this hospital, but not much at any other in New York. At the New York Hospital there are two regular anæsthetists, both medical women. One of these is also anæsthetist to the Women's Infirmary—a hospital staffed by medical women. I saw there an abdominal operation under open ether, but was too late to see the induction. The anæsthesia was extremely good.

At the Roosevelt Hospital I was present at an operation for goitre under gas and oxygen supplied from a very cumbrous apparatus with four large glass cylinders. It seemed to work easily and the anæsthesia was satisfactory. This method is only used for special operations and not as a routine anæsthetic. The anæsthetist there was a nurse specially trained in the administration of anæsthetics.

At three other hospitals which I visited the anæsthetics were given by the internes, who correspond to our house surgeons. At the first hospital anæsthesia was induced by gas and ether with a Bennett's apparatus, which much resembles Clover's, but is fitted with an expiratory valve. Subsequent administration was by ether poured on to a sponge in a large cone. The anæsthesia was not very satisfactory—one man in particular gave a good deal of trouble by vomiting and retching during the operation. At the second hospital ether was used, preceded by ethyl chloride dropped on to the same gauze mask. The anæsthesia was fairly good, but there was some tendency to come round when the ethyl chloride was stopped. At the third—a babies' hospital—I saw a harelip operation, on an infant 5 weeks old, under ether administered by a Junker's apparatus and a tube passed through the nose. Oxygen was given with the ether during the latter part of the operation, the oxygen cylinder being connected with the Junker bottle in place of the air-bellows. This is the youngest patient I have ever seen anæsthetized with ether. The anæsthetic was taken perfectly well; there was no coughing or other trouble, and the anæsthesia was very satisfactory.

At Boston I saw only two operations at different hospitals, but with the same anæsthetist. Ether, preceded by gas, with Bennett's inhaler, was the method used for both. There was some cyanosis during induction in one case, that of a goitre, but otherwise the anæsthesia was satisfactory.

At Chicago the anæsthetics at all the hospitals I visited, with one exception, were given by internes. At the Augustana Hospital, where I saw a good many operations, induction was by gas and ether with Bennett's apparatus, and the administration was continued by ether poured into a cone. The anæsthesia required by the surgeon here was deeper than I had seen elsewhere in America. The patients were a good deal congested at times, and the breathing was not always kept free. One patient stopped breathing twice, but recovered fairly quickly on the tongue being drawn forward. At another hospital I saw operations done under scopolamine and morphia; the anæsthetist sat by and gave a little chloroform when required. The method was considered satisfactory because the patients were found to stand the operations well and to feel no after-effects. It certainly afforded the surgeon an excellent opportunity for demonstrating how insensitive the abdominal organs were unless the peritoneum was manipulated, but whenever this was done the operation had to be stopped while a little chloroform was given. The patient appeared quite conscious most of the time, but it was stated that there is never any recollection of pain during the operation and sometimes no recollection of any operation. At Chicago I also saw two operations under chloroform by Dr. Brophy for cleft palate. A modified Junker's apparatus was used. In neither was the anæsthesia very good; in one, especially, the administrator, an interne, was evidently unused to, and much afraid of, chloroform. The same was noticeable in the administration of chloroform by an interne at Minneapolis, also for cleft palate.

I now come to what was one of the main objects of my tour in America, the visit to Rochester, Minnesota. Like most people, I had heard and read much of the excellence of the anæsthetics at the Mayo clinic. In the last paper I read on the subject before leaving England the writer was inclined to attribute this partly to an inborn faculty for administering anæsthetics on the part of women, and partly to the aid of hypnotic suggestion. I was therefore very glad to have the opportunity of seeing them myself.

Our reception at St. Mary's Hospital, Rochester—the hospital where nearly all the Mayo patients are treated—was most cordial, as is, I believe, the experience of all visitors, and we were given every facility of seeing all we wished. I spent ten mornings at the hospital, where operations went on in four theatres simultaneously from 8 a.m. till nearly 1 p.m. I was able to stand close to the anæsthetists and move from one theatre to another as I liked. I watched chiefly the two anæsthetists who were working for Dr. W. and Dr. C. Mayo, but occasionally visited the other two theatres. The anæsthetists are all nurses who have been specially trained in the administration of anæsthetics. There are four full anæsthetists and at least one "understudy" to take the work in holiday time or during illness. The anæsthetists attend every morning, and are also liable to be called on to give anæsthetics outside during the afternoon. I gathered that they do no actual nursing, but have a certain amount of clerical work to do at the hospital besides their work as anæsthetists. During my stay at Rochester I saw only one anæsthetic and only one method used, and that was ether by the open method. I was told that other anæsthetics and other methods have been tried, but open ether certainly now reigns supreme. No preliminary drug is used except in stomach cases, in which $\frac{1}{6}$ gr. of morphia is usually given half an hour before operation.

METHOD OF ANÆSTHETIZING.

Before the anæsthetic is begun the patient is generally put into the position required—e.g., on the side for nephrectomy or prone for a Kraske's operation. Even the latter distinctly uncomfortable position seemed to trouble the patient very little. Further arrangement of the patient and preparation of the skin are carried on during induction. The patient is fixed fairly firmly to the table, usually by a strap above the knees, when in the dorsal position. One table was fitted with curved metal rods, attached to the table on either side, which were moved so as to fit down over the thighs and screwed into position.

Before commencing induction a piece of protective is laid across the eyes and above this is placed a pad of wool. A modified Esmarch's mask is used covered with a double layer of stockinet. Ether is dropped slowly on to this from the tin in which it is supplied by a pledget of wool fitted into the cork; the patient is talked to soothingly all the time. In about a minute a long piece of gauze is taken, attached by a twist to the handle and folded round the inhaler. Ether is now given more rapidly, but for a time the mask is not held down tightly and is occasionally raised to give the patient a free breath of air.

There was seldom any trouble during the first stage of anæsthesia, the anæsthetist continued to talk to the patient, describing in an encouraging manner the process of going under and assuring him he was taking the anæsthetic well. During the second stage there was frequently a little coughing and occasionally some excitement, but rarely any troublesome struggling. In about three or four minutes regular breathing was generally satisfactorily established. The anæsthetist then said the patient was ready and the surgeon was called. Usually another minute or two elapsed before the operation actually commenced, during which time the ether was steadily continued. Seven to eight minutes was the usual time between the commencement of the anæsthetic and the first incision. At the first incision there was generally some alteration in the respiration with often slight movement and sometimes inarticulate sounds on the part of the patient. As a rule the surgeon took no notice of this, but continued the operation without seeming inconvenience, and the anæsthesia rapidly deepened.

In the abdominal cases the anæsthesia was kept light throughout. In operations on the stomach, while the stomach itself was being operated on the anæsthetic was usually stopped altogether and the patient often came round enough to speak, but was only in a semiconscious condition. The anæsthesia was easily deepened when required for the later stages. Though the anæsthesia was often very light the anæsthetists always had the patients well in hand, and I never saw actual struggling or vomiting, though occasionally slight attempts at retching might occur. In goitre operations the anæsthesia was usually even lighter than in the abdominal ones. Ether was given in all goitre operations, except in a few exophthalmic cases, when local anæsthesia was used. Ether was also used for cleft palate operations and for tonsils and adenoids. The initial anæsthesia, which was obtained by open ether in the usual way, was fairly deep. In one cleft palate operation that I witnessed the operation was carried on for nearly ten minutes before the child showed signs of beginning to come round—ether was then administered by a Junker's inhaler and a satisfactory anæsthesia easily maintained. In cases of tonsils and adenoids the initial anæsthesia was generally amply sufficient for the operation, but there was a good deal more screaming during induction than is usually the case with ethyl chloride or chloroform owing to the longer time required to produce unconsciousness. I was told that ethyl chloride was used formerly but was given up. The same seems to be the case with regard to nitrous oxide; ether was used for quite small operative proceedings when gas would really have been sufficient.

DIFFICULTIES AND DANGERS.

During my visit I witnessed very few difficulties and still fewer dangers. Perhaps this was specially the case with regard to the work of Miss Henderson, the senior anæsthetist. Her anæsthesia was the the most uniformly light, especially in abdominal cases, but her patients always seemed in excellent condition. Her induction was nearly always quite smooth, there was little coughing and scarcely any mucus. High-pitched or irregular breathing often occurred during the operation and occasionally hiccoughing, but these did not disturb either the anæsthetist or the surgeon. The patients were always kept quiescent and there were never any symptoms of shock. The patients, some of whom were very old, stood what were often severe operations extraordinarily well.

The other anæsthetists whose work I watched had a few cases with somewhat troublesome coughing and secretion of mucus, and the respiration was not always quite satisfactory. In two cases, both nephrectomies, respiration became shallow and stopped, but in both was resumed almost immediately the tongue was drawn forward. In another case manipulation of the gall-bladder produced symptoms of slight shock, which, however, passed off without treatment. Beyond these instances I saw nothing to cause anxiety during any operation.

There has never, I was informed, been a death due to the anæsthetic at St. Mary's Hospital. After-sickness is generally slight, and ether bronchitis, I was assured, is unknown. That the method of anæsthesia is considered satisfactory there can, I think, be no doubt. At the Mayo clinic every department is thoroughly up to date, and in touch with all new improvements, and no expense is spared to have everything of the best, so if there were any cause for complaint other methods would certainly be substituted. Watching the anæsthetics at Rochester confirmed me in the belief to which I have always inclined, that light anæsthesia is safe anæsthesia and renders the patient less rather than more liable to shock. What disadvantages it possesses are from the side of the surgeon, not from that of the patient or anæsthetist. I certainly think the depth of anæsthesia frequently considered sufficient at the Mayo clinic would not satisfy a good many surgeons over here. I cannot believe that there can have been complete relaxation of muscles during many of the abdominal operations, especially in the early stages. Probably the broad abdominal retractors which were always used rendered complete relaxation comparatively unnecessary.

It is not fair, however, to regard the anæsthesia at Rochester as always light. For many operations, such as those on the perineal region and the kidneys, the anæsthesia was deep throughout, and so also, as mentioned before, was the anæsthesia induced in children for cleft palates and adenoids.

Accurate records are kept of all the cases anæsthetized at St. Mary's Hospital. The anæsthetist has a form by her side which she fills in as the case proceeds. On this is entered the time of starting the anæsthetic, beginning the operation, terminating the administration, and terminating the operation. The amount of ether used is also recorded, as well as some particulars about the patient and the operation.

The *amount of ether* consumed is certainly small. The usual amount for operations lasting from thirty to fifty minutes was 4 oz. In one goitre operation lasting forty-three minutes only 2 oz. were used. The largest quantities recorded on any of the records I saw were 7 oz. for a case of cholecystotomy and 6 oz. for one of double inguinal hernia lasting respectively thirty-five and forty minutes. These were both given by one of the junior anæsthetists.

I now turn from the Mayo clinic to consider shortly a few points in the administration of anæsthetics in America which strike the observer as differing from what we find over here.

First as to the anæsthetists. There are, I gathered, comparatively few medical men who practise anæsthetics as a specialty, but the number is increasing, especially in the Eastern cities. There appears to be a considerable tendency in America towards employing nurse-anæsthetists. It is not unusual for nurses to be sent as pupils to the anæsthetists at Rochester, in order to be trained for hospitals elsewhere. I inquired the reason why they had nurse-anæsthetists at

Rochester, and was told that they could not get medical ones. The system appears to work well at Rochester, but whether it would do so if generally adopted is doubtful, to say the least. A nurse's training certainly develops some of the qualities which go to make a good anæsthetist, such as quickness in observation and the power of recognizing whether a patient's condition is good or not, but for dealing with emergencies, and for scientific observation or investigation, a medical training is surely requisite. Where the choice lies, however, as it often does, between a specially trained nurse and a comparatively inexperienced house surgeon, it is not surprising that the former is often preferred.

Anæsthetics.—In America one is generally impressed by the comparatively little use made of chloroform. In some hospitals its use is almost prohibited—if used, a special report has to be made to the authorities. Ether is undoubtedly the usual anæsthetic, but as far as my observations go, not ether by the open method as practised at Rochester. When I saw open ether used elsewhere some other method was employed to aid in induction. This points to the conclusion that to give ether by the open method with the skill exhibited at Rochester requires much practice. The fact that chloroform is inadmissible as an adjunct no doubt helps largely to develop this skill. We find it is easy over here to get over difficulties in administering open ether by using a little chloroform; we get over the difficulties satisfactorily, but probably do not develop quite the same skill which we should do if we had to get on without the assistance of chloroform.

Another noticeable point in America is the complete neglect of the eye reflexes. Nearly everywhere the eyes are kept covered and never referred to. The general teaching is that the respiration and pulse alone are to be watched, and that the respiration is the main, if not the sole, guide to the depth of anæsthesia. Of course, most people will agree that the respiration is the most important, but to neglect the eyes entirely is surely to ignore aids which are of much value as adjuncts, especially so in giving chloroform. I saw more than one case when a knowledge and use of the eye phenomena would certainly have saved the administrator from going astray.

I think, also, that the importance of keeping an absolutely free airway is not so much emphasized as over here. I saw many instances of slightly obstructed breathing to which the anæsthetist paid no attention. No doubt the comparative safety of ether is the cause of this. I have heard it said that Americans consider that tongue forceps are used too much in London hospitals. I do not know whether this is so, but I am inclined to think that in America they use them rather too seldom. In all the cases I saw where respiration stopped there had been some preliminary obstruction to which the anæsthetist paid no attention. No doubt if the tongue had been drawn forward earlier the breathing would not have stopped, but I suppose it might then have been considered an unnecessary proceeding.

For myself, I can say that I learnt a great deal from the administrations I watched in America, and that everywhere I encountered the greatest kindness and consideration, for which I feel much gratitude.

DISCUSSION.

Mr. H. J. Paterson exhibited a number of slides illustrating what he had seen in America:—

(1) View of St. Mary's Hospital, Rochester.
(2) Portrait of Dr. W. J. and Dr. C. H. Mayo.
(3) Dr. W. J. Mayo at work in his theatre.
(4) Miss McGaw, the senior anæsthetist, giving open ether.
(5) Dr. C. H. Mayo in his theatre.
(6) The Pathological Laboratory at St. Mary's Hospital, with Dr. McCarty.
(7) Dr. Howard Kelly operating at Johns Hopkins Hospital.
(8) Dr. Howard Kelly operating in his private sanatorium.
(9) Dr. John B. Murphy operating at the Mercy Hospital.
(10) Dr. John B. Murphy operating at the Mercy Hospital.
(11) The School of Operative Surgery at Johns Hopkins University.

The President (Dr. J. Blumfeld) said Mrs. Berry's account had been most delightful and vivid. The paper suggested interesting comparisons with our own methods. The most striking difference appeared to him to be the slighter degree of narcosis which was considered necessary in America. The description of the return in stomach cases almost to consciousness, which the surgeon not only permitted but desired, showed what a difference there was in the practice of the two countries. Evidently in the States safety was the first consideration. Though Mrs. Berry alluded to the skill of the anæsthetists there in the matter of open ether administration, there surely was not a great deal of skill required to induce the light anæsthesia deemed sufficient. He asked how long a training the nurse-anæsthetists were required to have, and assumed that in the case of intelligent men or women not very much training;

was needed. He invited any surgeons present who had been to America to give their impressions as to the administration of anæsthetics there, as compared with our own country.

Dr. BARTON remarked that the amount of ether given in America seemed small, and asked whether the measurements were by weight or volume. Apparently it was sold by weight.

Mr. BELLAMY GARDNER said that in regard to the cleft palate operations, he was curious to know whether there was more hæmorrhage on account of ether being employed, and whether it interfered with the operation. In England it had been difficult to get the surgeon to agree to open ether for even removal of adenoids or extirpation of tonsils, because he was alarmed at the extra hæmorrhage in the first few moments of the operation. But this soon stopped, and under ether administration it was less likely to recur. In air passage operations of any other kind, however, it seemed quite inadmissible to use ether at all. Of course, the safety of the patient was of paramount importance in all operations, and he thought London surgeons had their own way too much, both as to the anæsthetic and the degree of abdominal relaxation demanded in order to facilitate their work. In some patients this relaxation should not be attempted; after the incision was made retractors should be used. In some cases the muscles to be relaxed were those of respiration, and then there was danger of stoppage of breathing. What the surgeon gained by being able to pull the muscles aside as easily as the lapels of one's coat the patient lost by the immediate incidence of a very great danger. He found ether very suitable in the case of children, and that it seldom resulted in difficulties with regard to the accumulation of mucus in the chest; when it arose in the upper air passages this could well be dealt with by sponging, and putting the patient into a proper posture. The tendency to struggling was very small, and, with the Skinner's mask, the patient was soon inattentive to outside sounds, and even to the conversation of the anæsthetist. The insertion of a gauze pad below the mask would be sufficient, in children up to 7 or 8 years of age, to induce deep anæsthesia eventually. It seemed evident from the paper that the anæsthetists in America in their use of ether had the whole of the profession at their back. Nothing could be done in the education of the general public on this matter unless the support of the profession was behind the opinion of the anæsthetists. So long as patients could answer one's recommendations by saying there were so many other kinds of anæsthetic in use, and would prefer one of the others, it would be impossible to press the use of this, the best and safest form of anæsthesia. If it could be stated by surgeons that ether given by modern methods was thirty times safer than other forms of anæsthesia, though it might cause more inconvenience during the operation, the result would be that many more proposed operations would receive the patient's consent.

Mr. FLEMMING (Bristol) said he differed from Mr. Bellamy Gardner's remarks in one respect—namely, that the most important point which the paper showed was that the surgeons in America backed up the anæsthetists. The most important conclusion from the paper, in his view, was that in the States light anæsthesia was adopted because of its safety. The belief in England that deep anæsthesia prevented shock was now dying out, but it had been the greatest handicap, and had caused more accidents in this country than anything else. He had used ether by the open method for quite young children since 1896, and found it very satisfactory.

Mr. G. E. GASK said that when going round the hospitals in America he saw operations at most of the large towns, and differed from Mrs. Berry on some points as to the excellency of the administration of anæsthetics there. He thought one should draw a line between the Mayo clinic, which was far above everything else in the States, and the rest. He saw anæsthetics given by nurses and by "internes" in all kinds of manners; and on the whole he would have said it was not done with anything like the same standard of excellency as one expected in London hospitals. At the Mayo clinic he saw only open ether being given, and it was administered extraordinarily well, better than he had ever seen open ether given in London. It was given in every kind of case without hesitancy, even in operations for exophthalmic goitre. He did not think there was much in the hypnotism of the patient by the anæsthetist. With regard to the question of ether bronchitis, he did not inquire to what extent this was met with in America, but in such a dry climate as they had over there bronchitis seemed little likely to occur. It must be remembered that at the Mayo clinic they did not get the very acute cases which were frequent in London hospitals: most of the patients had to travel at least 500 miles to get to the hospital, and this fact ruled out, very largely, such cases as acute peritonitis.

Dr. SILK said the only remark he intended to make on the paper had been partly led up to by Mr. Gask—namely, his mention of the line which must be drawn between the Mayo clinic and the other hospitals in America. The class of patient who went to the Mayo clinic and the conditions under which they were operated upon were totally different from those which obtained in London hospitals. He did not propose to enter the wide field of discussion as to the best anæsthetic, or whether it was better to have the patient only lightly under, or sufficiently under to satisfy the surgeon. He was somewhat mystified by the measurements, especially after seeing the bottle which Mrs. Berry passed round. How was the quantity given known? Was it weighed or measured?

Dr. LLEWELYN POWELL said the chief impression left on his mind by Mrs. Berry's admirable narrative and by Mr. Paterson's pictures was that the American surgeon got the best anæsthetist and the best anæsthetic he could in America, but he would probably prefer, if he could, to have a member of that Section, such as the President, to give the anæsthetic. There was surely something which American anæsthetists could learn from their English *confrères*. Though it seemed clear that there were cases of very satisfactory anæsthesia from ether in America, yet probably in many cases the English anæsthetist would get an induction more satisfactory to the surgeon by varying the kind of anæsthetic to suit the case. There was still something which the Old World might teach the New, and that was evidenced by the way in which medical men in America were taking up the subject of anæsthetics and making a speciality of it.

Mr. **Carter Braine** asked who was responsible for the anæsthetic in America if a fatality occurred. He asked this because, apparently, the anæsthetist was not a registered practitioner. He did not agree with the remark of Mr. Bellamy Gardner about muscular relaxation, for there were many operations in which it was essential to procure complete flaccidity, more especially of the recti abdominales. He alluded to such operations as those for malignant growths or papillomata in the bladder and prostatectomy. Given a stout person, and complete relaxation of the recti not obtained, then these operations could only be performed with the utmost difficulty, and might even have to be abandoned. He showed an open ether inhaler which had been given to him by Dr. Ferguson, of New Jersey, and regarded it as the best one of its kind he had seen. It was after the style of the Schimmelbusch, with the usual layers of gauze on it, and the whole being enclosed in a hood. The rim was made of copper, and it was easily adaptable to the face. The hood prevented so much of the ether being wasted, created a kind of atmosphere of ether vapour around the patient, and thus the amount of ether required was much reduced.

Mr. **H. J. Paterson** said that the reason the Americans had been so successful was that they had learned from us, while we, on our part, had not learned anything like so much from them. Nothing impressed him so much during his visit to America as the advantages of the open ether method as given at the Mayo clinic. Many years ago, in the exuberance of his surgical youth, he was rash enough to say, at the Medico-Chirurgical Society, that it was criminal for a surgeon to do an abdominal section under ether. Somebody in Rochester had read that remark, and when he went there he was asked to open a discussion on anæsthetics from the surgical point of view, in the expectation that he would be an advocate of chloroform. But he had been so impressed with the results of open ether that he thought he ought first to find out something of what went on behind the scenes. Therefore he spent a week in going round to all the patients who had had anæsthetics during the previous fortnight, examined their charts, made notes on them, asked questions about their sensations, and so on. When he came to open the discussion, he had to confess that he had been convinced of the superiority of open ether over chloroform for general surgery. Since he had returned home he had had all the more reason to remain assured that the opinion he had formed in Rochester as to the value of ether was correct. It was impossible now to touch on all the interesting questions which had been raised. He wished to make one remark on the giving of ether by the open method, but his remarks did not apply to any of the members present. It was this that many anæsthetists in this country talked about giving ether by the open method, but on watching them he could not recognize it as the American method. The essential feature of the open ether method in America was the absolutely continuous administration: a drop on the mask every two seconds was the rate. In fact, at Rochester it was called the "drop method." In this country the anæsthetist would often pour 2 dr. or 3 dr. on the mask, then stop for a few minutes and then pour a few more drachms on the mask. This was not open ether as practised in America. He was much interested in Mr. Gardner's remarks as to consideration for the safety of the patient, because he himself felt strongly on that point, and it was not right to subject patients to the additional risk associated with the giving of chloroform. Mr. Gardner also referred to the use of chloroform in mouth cases; in America they used Crile's apparatus in these cases. This, in his experience, was one of the most satisfactory methods of anæsthesia for tongue or mouth operations, and was certainly the safest method.

The PRESIDENT asked Mrs. Berry as to the preliminary use of morphia. He believed the opener said that at the Mayo clinic they did not use such preliminary injections, except in stomach cases. Had they given up the practice, or had there never been a routine use of atropine before the open method? Here it seemed to have been the general experience that when atropine had not been used before open ether, in a considerable proportion of the male patients the result had not been very satisfactory. Another point was as to the administration of ether by Junker's inhaler. Mrs. Berry saw it used in that way for cleft palate operations. Was this in children only, or did she see adults kept under satisfactorily by it?

Dr. SCHARLIEB, C.M.G., in reference to the President's remark, said he had given ether by Junker's inhaler very frequently at University College Hospital, including cases of abdominal section in adults, and he had found the method extremely successful.

Mrs. DICKINSON BERRY, in reply, said the amounts of ether given were by measurement and not by weight. One objection to the use of the tins was that one could not see the amount that had been used without pouring out the remainder, but she believed this was always done after an operation before the amount used was recorded. She witnessed only one operation for cleft palate under ether, and there was certainly no more trouble from hæmorrhage in this than in the others done elsewhere in the States under chloroform. She agreed with what Mr. Flemming said about light anæsthesia, the general lightness of the anæsthesia and its undoubted safety was what had specially impressed her at Rochester. She agreed with Mr. Gask that the anæsthetics at Rochester stood in a category apart from the rest of the United States as far as her observations went. Elsewhere, however, she had seen anæsthetics given but seldom by specialists; frequently they were administered by internes, and the administrations, though generally safe owing to the exclusive use of ether, were certainly not always smooth. With regard to the question of responsibility, the anæsthetists at Rochester considered that the surgeon undertook the responsibility, and that though anæsthetists, they were still in the position of nurses. The surgeons, however, interfered very little with the anæsthetists, less so than was sometimes the case in English hospitals. In reply to the President, she could not say whether atropine as a preliminary had been used formerly to any great extent and been given up. Both the present anæsthetists and the late senior anæsthetist, Miss McGaw, stated they preferred to give ether without any preliminary drug. In answer to Mr. Gask, she said that many patients who were very ill—e.g., with advanced malignant disease—did come to Rochester, but the kind of patient who often gives trouble to the anæsthetist in a London hospital, answering to the alcoholic drayman type, was seldom found there. In answer to the question of how a prostatectomy could be done without complete abdominal relaxation, Mrs. Berry said that in some cases there was deep anæsthesia, and in such an operation as that this would certainly have been obtained.

Mr. W. DE C. PRIDEAUX (Weymouth) sent for exhibition by Dr. Scharlieb, a facepiece designed for use by patients who had lost a good proportion of their teeth; it gave accurate coaptation, preventing leakage of air by two little air cushions.[1]

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[1] See *Proceedings* (1912), 1913, vi (Odont. Sect.), p. 8.

Section of Anæsthetics.

February 7, 1913.

Dr. J. Blumfeld, President of the Section, in the Chair.

Nitrous Oxide and Oxygen in Major Surgery.

By H. M. Page, F.R.C.S.

During the last few years there has been a revival in the interest taken by surgeons, anæsthetists and physiologists in the question of anæsthesia, and much valuable work on the subject has been done. This revival was principally due to the work of Dr. George Crile on "Shock," in which he showed that a very appreciable part of the cause of death after a grave operation is due to the anæsthetic, and he maintained that this average percentage could be lowered. He arrived at the conclusion that of all the general anæsthetics nitrous oxide and oxygen gave the smallest percentage attributable to the anæsthetic, and that therefore in some cases this would determine the question of life or death. I understand that he now uses these gases almost as a routine method.

Of late years much more work has been done in the use of nitrous oxide and oxygen for major surgery in America than here. Dr. Crile, Dr. Teter, Dr. Cotton, Dr. Boothby and others, have done valuable work on the subject, but that operations could be performed under nitrous oxide and oxygen was demonstrated many years ago, and to Sir Frederic Hewitt, a member of this Section, we owe the first practical apparatus for their administration, in fact I may say the only practical apparatus up till quite lately. I need not refer before this audience to his well-known experimental and clinical work.

My object in reading this paper is to suggest the further study and development of this method over here, as has been done in

America, and believing as I do in the importance of the matter, I am very glad to gather from one or two papers which have lately been published on this side, and conversations I have had with other anæsthetists, that a new interest is being taken in the administration of nitrous oxide for prolonged operations.

The drawbacks to the method are, that to give nitrous oxide successfully and safely for major operations much practice is necessary, and it must always therefore remain in the hands of the expert. *Successful* anæsthesia includes, of course, "ease" for the surgeon in operating, which is of importance to the patient as well as to the surgeon, in that it may affect the question of shock, and in some cases the complete success of the operation. The administration taxes the attention and endurance of the anæsthetist to a much greater degree than any other method. More or less cumbrous apparatus must be used. It is by far the most expensive method of producing anæsthesia. One must therefore try to make out a case for a more extended trial of these gases.

I do not think anyone will deny that nitrous oxide is in itself by far the least toxic and safest general anæsthetic we have at the present moment, and that *after*-results and discomforts are quite negligible in the large majority of cases, as compared with the effects of chloroform and ether. By the rapidity of its action and pleasantness, induction is robbed of most of its terrors, and many authorities look on that alone as of great importance. The element of fear can be further eliminated by a previous injection of a narcotic. If after injection of an opiate induction is done by any other method, the patient will be more thoroughly woken up, and so part of the beneficial action of the narcotic will be lost. When the nitrous oxide is withdrawn, the patient comes round either immediately or within a few minutes, and is perfectly himself. This has been objected to by some as likely to cause earlier and more acute feeling of pain, mental distress and fright. My experience is that the large majority of those who have had a previous injection of morphia or omnopon feel no great amount of pain; in a small minority there is pain, but nearly all of them go to sleep directly after they are back in bed again. If there is pain, I believe myself it is better for the patient to have more morphia or omnopon, than that the tissues should have slowly and laboriously to eliminate the more lethal poisons, ether and chloroform. As to fright from waking up so quickly, none will certainly be felt if the nitrous oxide is continued until the dressings are on. If you do not continue the administration till then the patient should be allowed to come completely round and thoroughly understand that everything is finished before being disturbed. If ether has had to be given along with the nitrous oxide, recovery will be delayed for a few minutes longer and the question will not arise. *After*-vomiting, as usually meant, practically does not occur. In a few cases the stomach may be emptied once with no distressing or lasting nausea. Occasionally you may meet with the patient who vomits after a narcotic, but I am beginning to think that these cases may be found to occur less often after omnopon than morphia. The method in my experience does not produce lung troubles, or increase any that may be previously present. I have given gas and oxygen with and without a little ether when bronchitis, unresolved pneumonia and advanced tuberculosis has been present without the mischief being increased. The sitting position can be adopted at once, and water, food, stimulants and medicines can be given very soon if desired.

As to what is gained apart from comfort and immediate safety to life when the administration is in skilled hands, there can be no question, whatever may be the reason, that after a long and severe and shock-producing operation under nitrous oxide and oxygen, with or without the addition of some ether, the clinical condition of the patient can only be compared to that following a successful spinal anæsthesia. Shock from trauma caused under nitrous oxide and oxygen compared with all other general anæsthetics is much less. Crile considers it four times less. To account for this, there is the fact that the gas is much less toxic to the tissues and cells generally of the body. It is eliminated with astonishing rapidity, and even after two or more hours' administration leaves very little result of its action.

Dr. Crile has brought forward sections to show the effect on certain cortical cells of the brain of the administration of chloroform, ether, and nitrous oxide respectively; the changes in the cells were most marked after chloroform, less after ether, and very slight indeed after nitrous oxide. I do not know if these observations of his have been confirmed, but I have not seen them contradicted, and it is what one would expect.

Taking the view most generally held that anaesthesia is due to the tissues being deprived of their power to use oxygen, in the case of the use of nitrous oxide and oxygen that power apparently must return immediately. Some recent work and writings seem to suggest that some of the beneficial effect of this rapid return of the nervous and other tissues to their normal condition may be due to less interference with the actions of the ductless glands, perhaps more particularly with the adrenals.

As to the expense, it can be considerably lowered by using a certain amount of rebreathing; and on the question of rebreathing, account must be taken of the work that has been done on the elimination of carbonic dioxide by Pembrey, Haldane, Priestley, Boothby and others, and especially of the most interesting and suggestive work of Yandell Henderson. I believe that all physiologists are agreed that exaggerated respiration lowers the normal CO_2 content of the blood, and that a condition of diminished CO_2 content of the blood must have a marked deleterious effect on the cells and their chemical functions, and that CO_2 is the principal stimulant to the respiratory centre. In the induction period of anæsthesia there is invariably, if not at other times, exaggerated respiration. It has been shown that this loss of CO_2 will be much accentuated in abdominal operations with exposure of the viscera, and in relation to this it has occurred to me that the comparatively small amount of shock met with in some severe pelvic operations, in the Trendelenburg position, under various anæsthetics, may be partly due to the fact that the intestines are not exposed and CO_2 not lost. In complete colectomies when the intestines have had to be left outside the abdomen, Arbuthnot Lane carefully wraps them in silk or other tissue, boiled in vaseline, thus making an artificial omentum, which according to Crile would stop the elimination of CO_2, and in my experience shock has been less in these cases than I should have expected, no matter what method of anæsthesia one has used.

Besides Henderson's valuable experimental work on the question of carbonic dioxide and its relation to shock, Dr. Crookshank in his book "Flatulence and Shock" pointed out that the control of the processes that lead to the giving off of CO_2 is probably invested in the adrenal bodies and the chromaffine system generally, and that there is therefore an intimate connexion between the vasomotor processes concerned in shock and loss of CO_2 via the adrenals. The recent work of Van Anrep and Itami appears to go far towards confirming this view. This may perhaps partly explain the lessening of shock when the adrenals are thrown out of play by spinal anæsthesia. Anyhow, whatever may be the ultimate position of this recent physiological work, it cannot be doubted that this loss of CO_2 may have a considerable bearing on the question of shock. Whether ultimately all the conclusions drawn by

Henderson from his valuable experimental work are accepted or not, and without minimizing the part played by afferent nervous impulses, this loss should be taken into account.

My clinical experience is that a certain amount of rebreathing is beneficial to the patient, and I therefore agree with the American authors who have advised rebreathing as a means of replacing an undue loss of CO_2, and that therefore there is fortunately a more important reason for using rebreathing than the question of expense. I also find that the use of rebreathing makes the anæsthetist's work easier. I like to have at least 300 gallons of nitrous oxide and 90 gallons of oxygen available for a long operation.

I have used nitrous oxide and oxygen for producing anæsthesia, with or without the addition of ether, in ninety-four undoubted major operations, most of them having some additional cause adding to the gravity, or likely to produce much shock, also in very many minor operations, and in a good many others on the borderline between the two. My longest administration was one hour and fifty-five minutes. The following list includes the more important cases in which I have used this method :—

- General septic peritonitis.
- Appendix abscess.
- Appendicectomy.
- Colectomy.
- Short-circuiting.
- Volvulus.
- Combined abdominal and rectal excision of growth.
- Gastro-jejunostomy.
- Colotomy.
- Gastrostomy.
- Pancreatic growth—exploratory operation.
- Carcinoma uteri—Wertheim's operation.
- Subphrenic abscess.
- Suprapubic cystotomy.
- Nephrectomy.
- Nephrolithotomy.
- Vesico-vaginal fistula.
- Amputations of—
  - Hip
  - Thigh } in diabetes.
  - Leg
  - Arm
- Plating of fractures.
- Fractured patella.
- Amputation of breast.
- Whitehead's operation for piles.
- Fistula in ano—in diabetes.
- Osteomyelitis.
- Varicose veins.
- Radical cure of hernia.

Fifty-one of these operations were intraperitoneal, and in support of the possibilities of this method I can say that in only one of these did the surgeon complain of the anæsthesia, though in some cases he may have been more long-suffering than others would be. In a few cases I have been told, on inquiring, that the anæsthesia was good, and the surgeon has then been surprised to hear that $N_2O + O_2$ had been given all the time. Some administrations are easy and some very difficult, but more ether can be added if necessary in the difficult cases, or, as sometimes has to be done when using other drugs, a complete change of method can be made. The most difficult cases are met with in the stout, muscular, florid, and alcoholic patients.

I have only changed the method, after having started the administration, three times, and only one of these cases is included in the ninety-four major operations.

(1) I changed to open ether after about twenty minutes. The patient was in the habit of taking drugs, a fact which was not known at the time, and the comparatively small dose of morphia had only an exciting effect.

(2) Not included in this series. I changed to ether alone immediately after induction, as the case was obviously going to be very difficult. I was using Hewitt's apparatus and therefore could not use rebreathing.

(3) Also not included: the case was giving me much difficulty, and as there was no special indication for giving $N_2O + O_2$ I changed the method.

With greater experience and the improved apparatus I now have, I do not think it would have been necessary to change my method.

The most important contra-indications are advanced degeneration of the heart and vessels, a marked degree of emphysema, and the presence of dyspnœa or obstruction to the air-way.

Of the ninety-four patients nine died. They were all in a desperate condition. Four were cases of neglected general septic peritonitis. Two were cases of neglected intestinal obstruction, in which Paul's tube had to be used. One was a case of obstruction in which a resection was done in an emergency operation. All of these four cases had been neglected for days, and were more or less moribund. One was a case of amputation of the upper arm after an accident, the patient being bloodless, cold, with respiration of a sighing character. He had had an anæsthetic twice before during the day in attempts to save the arm. Lastly, a case of duodenal ulcer, completely blanched and exhausted from hæmorrhage. None of these patients died on the table—they died some hours or days after the operation. All of them recovered consciousness. In none of these desperate cases did the administration give me the same amount of anxiety I should have had if giving one of the other anæsthetics.

All the other patients recovered. In the series were six cases of general peritonitis with perforation of the appendix, the ages varying from 16 to 60. Appendicectomy, drainage, &c., was done, and they all recovered. In one of the cases of amputation of the breast with dissection of glands the patient was aged 80. Hæmorrhage was not increased, and the surgeon who operated told me, that not having operated on a patient anæsthetized by these gases before, he was surprised at her good condition when he saw her in the evening. My oldest case was that of a man aged 81. A subphrenic abscess was opened and drained. He was under the care of a physician for his heart; a few days before the operation his pulse had been about 80 at the wrist, and about 160 at the heart. He laid on his left side during the administration; no trouble was experienced, no ether given, and he was no worse or different at the end of the operation than before. The youngest patient with whom I have used this method was aged 5. It was an appendicectomy. No trouble was experienced in this case, but I do not recommend that this method should be used at such an early age without some special indication. Another case was that of a woman, aged 53, who a year before was under a physician for grave chronic heart disease. Peritonitis with no adhesions was present. Before operation the pulse was 130. A gangrenous appendix was removed. The fat on the abdominal wall was estimated by the surgeon at 5 in. Two or three minutes after the facepiece was removed she said she felt able to walk back to bed. Her condition was just the same as before the operation. No ether was given in this case—she recovered. A diabetic, in whom an amputation was done at the thigh, ate and enjoyed his tea an hour after the end of the operation.

I have administered N_2O+O_2 in conjunction with spinal anæsthesia, the patient being in the Trendelenburg position. I should think it must be very uncomfortable to be conscious during a pelvic operation where this position is used.

As to the indications for the use of this method, I consider that whatever the operation may be, a patient is fortunate whose anæsthesia is successfully produced by these agents, but that the special indications for the administration of nitrous oxide and oxygen are as follows:—

(1) Desperate cases, either from toxæmia or traumatism and hæmorrhage. The choice here, if available, should lie between this general agent and spinal anæsthesia, that is, if all the chances the patient can have of recovery are to be secured; and the same choice holds good, to perhaps a modified degree, in all operations likely to produce much shock. Lack of time prevents me from going fully into the question of choice between this method and spinal anæsthesia, but I am sure that nitrous oxide and oxygen is better than spinal anæsthesia in the nearly moribund class of case, as they do not stand the general toxic effect of the drugs used for spinal anæsthesia. My experience in the use of spinal anæsthesia in acute abdominal and highly toxic conditions in children

makes me agree with Mr. Tyrrell Gray that spinal anæsthesia is the best in these cases, and it is of course a very good method and less trouble in many abdominal cases in adults.

(2) For operations on diabetics nitrous oxide is certainly the best anæsthetic. All the forms of intravenous and spinal anæsthesia entail traumatism on vital parts, and both chloroform and ether have been followed by fatal coma. These operations are mostly amputations, and anæsthesia is therefore easy.

(3) I would also suggest its use in kidney and genito-urinary operations, if after-troubles, such as fever, suppression, lung trouble, &c., are feared, and spinal anæsthesia is not indicated for the purpose of relaxation, as in some bladder cases—e.g., prostatectomy.

An injection of morphia or omnopon and atropine should be given an hour before the operation, or if by accident or on account of some contra-indication an opiate has not been given, atropine should be injected alone.

Induction, I consider, is best done always with N_2O+O_2 and ether. The addition of ether during induction makes the administration easier all through, even when none is added afterwards.

Enough oxygen must be given to keep the patient pink. In actual practice there is no question of percentage of oxygen whatever instrument is used—the indicator is merely a guide for adding oxygen until the desired result is produced. If at any time there is difficulty in getting the depth of anæsthesia required, ether should at once be added, instead of pushing the N_2O by giving it under pressure.

If there is difficulty in maintaining a good colour, the cause must be found and removed. It may be due either to too little oxygen—mechanical obstruction—inadequate respiration, due either to too great a depth of anæsthesia, acapnia, or circulatory failure.

In my hands some ether has been used in about 66 per cent. of the major cases: in some cases during induction only, in others at the end as well, where parietal peritoneum has had to be sewn up; again in other cases there has been a return to ether for a minute or two at intervals, much more rarely a little ether has been given during the whole administration.

I am using ether more often than I did at first, and I do not find the condition of the patient appreciably altered at the end by the small quantity of ether inhaled. Of course, as little ether is used as possible. The following three cases give some idea of the amount of ether I have used:—

(1) Short-circuiting the ileum into the highest part of the rectum: N_2O+O_2 for one hour and a quarter; ether, 1½ oz. put into Clover's inhaler at the beginning—no more added.

(2) Laparotomy and excision of rectum, coccyx, &c., N_2O+O_2 for one hour and fifty-five minutes; ether, 4½ oz. This was the total quantity put into the inhaler. No opiate was given before the operation, only atropine, as an injection the previous evening had made her vomit and feel very unwell.

(3) Complete removal of breast and glands. N_2O+O_2 for one hour and a quarter; ether, 1 oz., before induction; no more added.

In an easy case, and the very grave cases are generally the easy ones, the rapidity with which you can deepen and produce anæsthesia by the nitrous oxide with the help of a little ether enables you very often to give very little N_2O, and, of course, no ether, for quite long periods if the particular stage of the operation is not painful.

Rebreathing for several breaths alternately with breathing out through an expiratory valve should be used. I find the expiratory valve and the bag near the facepiece of use in judging the condition of the respiration, and I prefer the English facepiece and Hewitt's ordinary three-way stopcock, not the one for gas and oxygen, to the American pattern of facepiece and expiratory valve. If mechanical improvement of the air-way is called for, a tubular mouth-prop with a stiff india-rubber tube generally answers the purpose. I have not, so far, had to use the tongue forceps or clips in any of these cases.

In a few of my cases there has been a tendency to cessation of respiration, which has occurred when the patients have not been rebreathing. They were a good colour at the time, with quite a satisfactory pulse, but a commencing pallor, rather than a cyanosis, quickly began to appear, and although they were fairly well under, I suspect that loss of CO_2 was the principal cause, and not nitrous oxide poisoning. I have, however, always shut off the nitrous oxide, given a breath or two of air, followed by rebreathing of oxygen without nitrous oxide, and, of course, attended to the air-way. One aims at maintaining an efficient respiration, a good colour, and, if possible, a weak corneal reflex. If the corneal reflex has to be abolished to get sufficient relaxation, it should be allowed to return at short intervals. This will not interfere with the operation. In the case of too much rebreathing, the respiratory action is increased; the stopcock is then turned to valvular action.

A toxic condition from nitrous oxide can be produced. This is more likely to come on if the gas be given at an increased pressure, and especially in susceptible patients. The respiration, according to American writers, is first "laboured, with accentuation of the expiratory phase, followed by cessation." I have met with this kind of respiration, and have no doubt it would be followed by apnœa if not attended to. Vomiting is said to occur occasionally, both in too light and too deep anæsthesia. I do not happen to have met with it in patients prepared for major operations.

That there are dangers to be avoided in this as in every method of anæsthesia is quite certain, as deaths acknowledged to be due to the anæsthetic have been reported. These dangers and deaths can, I believe, be prevented by the unremitting attention of a skilled anæsthetist, who has seen his case beforehand, and deliberately chosen this method. Under these circumstances, I am confident that nitrous oxide and oxygen, with or without ether, will in certain cases give the patient a greater chance of recovery than any other method of producing general anæsthesia.

My thanks are due to the members of the staffs of Guy's and the West London Hospitals, who have allowed me to try this method; especially to Mr. Arbuthnot Lane, Sir Alfred Fripp, Mr. Aslett Baldwin, and Mr. Donald Armour.

The apparatus required must, I am afraid, always be cumbersome. I have here Dr. Teter's apparatus. Without the cylinders it is fairly light and portable. I used it in its original state in some of my most grave cases, but personally I have done better with it since I added the bag near the facepiece. I have not found the flow of gases as even as I should wish; a good deal of manipulation is required to prevent snowing up of the valves. Dr. Teter claims that rebreathing can be used with it, but the bags are a long way from the patient, and I find that a bag nearer the facepiece is better. And it is easier to regulate the pressure of the gas with the bag in this position. I have not always been able to get a sufficiently strong ether vapour from his ether attachment, and have done better since I have introduced Clover's container between the bag and the facepiece. Without any proof of its advantage, my experience makes me prefer to heat the gases.

Dr. Boothby's apparatus, which he kindly demonstrated at the West London Medico-Chirurgical Society last year,[1] seems to me a great advance on any former apparatus, in that two of the bags are done away with, leaving only the one close to the face. He claims that the valves are not liable to snow up, and there are regulators and gauges to fix the pressure at which the gas is delivered. The gases flow through water, which enables you at a glance to judge approximately what mixture you are using, which I feel undoubtedly would be a great help, notwithstanding that, of course, you judge your procedure as in any other method of anæsthesia by the condition of the patient. It may also, to some extent, be purifying for the gases.

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[1] *West Lond. Med. Journ.*, 1912, xvii, p. 294.

Dr. Gwathmey and Dr. Wolsey have designed a nitrous oxide, oxygen, and ether apparatus much smaller than Dr. Boothby's, but on the same principle—namely, that there is only one bag and that the gases flow through water, and judging from a description on paper I should think it would answer the purpose.

In some of these major operations I have used Hewitt's apparatus with Clover's container between the stopcock and facepiece, but I have done better and easier work with Dr. Teter's apparatus. The drawbacks to Hewitt's apparatus are that it does not allow rebreathing, and that it is very difficult to regulate the amount of oxygen you are giving; nor can you cut off the gas and give pure oxygen.

The Dental Manufacturing Company have put together for me this cheap and portable combination, consisting of two bags in Trewby's springs, to help to regulate the pressure of the gases for nitrous oxide and oxygen respectively, a mixing chamber with oxygen regulator, and another bag close to the face. Clover's inhaler can, of course, be used with it. The cylinders are coupled up separately, and can be changed during the administration.

I have used this arrangement successfully in severe intraperitoneal operations, and in conservative dentistry have given gas and oxygen continuously by the nasal method with it for thirty-one minutes, the patient being in the dental chair, but of course, it does not fulfil all the conditions one would wish for, and I prefer the larger and more costly apparatus.

DISCUSSION.

The PRESIDENT (Dr. J. Blumfeld) thanked Mr. Page for his paper, in which he had made out a very good case for the use of gas and oxygen and rebreathing, at least in extreme and dangerous cases. His own experience was limited to a few cases in which he had employed no ether at all, only gas and oxygen: he had had no apparatus which permitted of rebreathing. When he had used the method described by Mr. Page in serious and very grave cases in which it was most important to avoid toxic effects, his experience agreed with that of the author—i.e., he had been able to bring people successfully through in conditions where it would have been very difficult with ordinary methods entailing the use of chloroform or ether. A particular example was that of a woman with a gangrenous gall-bladder, very fat and extremely ill, for whom it was necessary to open the abdomen. She would have probably succumbed under any other method. A number of the points brought forward by Mr. Page would appeal to the antagonistic spirit of some of the members, especially those who were staunch supporters of the open methods of administration. The different points of view from which shock was now regarded were very interesting to observe, and there could not be a wider difference of practice than that between those who employed open ether and those who advocated such methods as Mr. Page's, based upon the physiology of Yandell Henderson. And one found it difficult to reconcile the statement that one must avoid all degrees of asphyxial element, with the view that there must be no diminution of CO_2 in the blood and tissues. Such points would doubtless be discussed.

Mr. BELLAMY GARDNER joined in thanking Mr. Page for his most interesting paper on the subject which had engaged his (the speaker's) attention for many years. In the year 1897 he first brought forward the use of gas and oxygen for minor operations—i.e., those not involving abdominal sections, but including small varicose vein, adenoid, tonsil, and stricture operations. In the following year Mr. Herbert Paterson, who was the resident anæsthetist at St. Bartholomew's, wrote a paper detailing a series of operations in which this anæsthetic had been used,[1] in one case for one hour and five minutes. Later still he (Mr. Gardner)[2] wrote a further paper giving experiences with this anæsthetic for major surgery, including the radical cure of hernia, adenoma of the breast and longer varicose vein cases, but not for abdominal sections. It was a very expensive anæsthetic, and an American doctor had written him saying that over a three months' trial it was sixteen times more expensive than ether, so that on the whole it was not worth the trouble. Many of the patients were sick, and he could not promise that they would not be. As he (Mr. Gardner) said at the last meeting, a great deal depended on the general view of the profession at the moment, and it was difficult to get the patients properly prepared as for ether, though this was quite necessary. Also the environment was often a difficult one: the patient might be on a large, broad bed in an hotel, so that one had to crawl on the bed while the gas-bottle, fitted with foot-keys, remained on the floor: moreover, under its influence the patient was perhaps somewhat rigid, and the surgeon got on better with the more relaxing anæsthetics. At that time he had a remarkable experience in a case in which he was using an ether apparatus without valves, to which a gas-bag was attached. There was a breathing channel 2 in. in diameter. He found that his ether inhaler, which was supposed to contain 4 oz., had leaked, so that he had only $\frac{1}{2}$ oz. of ether, though plenty of gas. The operation was to occupy twenty minutes. He got a remarkable anæsthesia by keeping on with the gas, yet with the aid of this "farthing's worth" of ether the operation went off beautifully. Gas and oxygen alone was a difficult mixture to promise success with. He had been to cases where the surgeon promised matters would be satisfactory with it, but when the patient was an alcoholic it was almost impossible to render him unconscious with gas and oxygen. Still, it was satisfactory if other conditions were favourable, and where the patient was on a narrow table, which permitted proper use of the apparatus, with the foot-keys, &c. He soon saw that rebreathing was better than the use of valves, and he thought laboured respiration in the latter case was largely due to the working of the valves for a considerable time. In his own practice he reserved the method largely for diabetic patients, for whom he regarded it as the most valuable of all forms of anæsthetic. Three weeks ago he had a case of a gentleman who had had diabetes for eight or nine years and had a huge carbuncle on the back of his neck, which had been neglected, and was sloughing; the patient was passing 10 pints of urine per day. To give any general anæsthetic for an operation for relief of the pain seemed out of the question on account of the danger of coma supervening, but he administered gas and oxygen, and the patient, much relieved, recovered consciousness, but died two days afterwards from the effect of the general septic absorption. He did not think the rapid return of the pain after gas and oxygen counted for very much, for he had seen patients who had had Whitehead's operation done for excision of the rectum under ether, rapidly wake up to the most excruciating pain, even when they had previously been deeply under the anæsthetic. He did not believe ether warded off the pain for very long. When gas and oxygen were used alone, unless the patient was very carefully prepared, it was not uncommon for attempts at vomiting to come on after three or four minutes' anæsthesia. The moment when the patient was going to be sick was signalized by very low blood-pressure and intrinsic obstruction to the air passages, a moment at which added asphyxia tended to turn the balance against the heart, and this might cause secondary syncope. To the same degree this could scarcely arise under other anæsthetics. At such a moment there was nothing to be done except to remove the facepiece and allow the vomiting to proceed, and to clear the air-way. It was difficult to start again satisfied that there was no vomited material in the passages. If the anæsthetic now spoken of should come into general use he feared that deaths might occur from asphyxia during impending vomiting; and it required very careful tuition and the insertion of a mouth-prop in every case in order to avoid danger. It would be unwise to give gas and oxygen and rely on the nasal air-way only. The after-effects of ether, by the ordinary or the open method, were good. He doubted whether the brain cells were damaged to any extent as a rule. He went one day into the Prince of Wales's Hospital and saw five patients who had had abdominal section the previous day; they were sitting up and chatting, and apparently as well as if nothing had happened to them. It did not seem necessary to administer such an expensive anæsthetic as gas and oxygen when the after-effects from ether were so good.

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[1] *Brit. Med. Journ.*, 1898, i, p. 211.

[2] *Ibid.*, 1898, i, p. 1133.

Dr. Des Voeux said he had seen the method now described used in two cases. One was that of a sensitive lady, who had had an anæsthetic on four previous occasions, consisting of ordinary gas and ether, and who after each of them had vomited and suffered mentally and physically. When the present method was used she was under the anæsthetic for twenty minutes, but the change was extraordinary. She came round very quickly, and in an hour she was comfortable; although she felt sure before the operation that she would vomit on coming round, she did not do so. Recently he had a very serious case of illness, requiring operation, and thought of the method of oxygen and gas. The patient was a medical man who had suffered from severe tuberculosis for two years, whose right lung was almost completely blocked by quiescent tubercle, with adherent pleura. For thirteen months he had had severe abdominal tuberculosis, with a hectic temperature running up to 103 F. He wished to be operated upon, but as he was wasted to practically a skeleton, and was moribund, every method of dissuasion was tried, but he firmly believed that if he were short-circuited he would have a good lease of life. Several surgeons refused to operate, but eventually Mr. Lane was prevailed upon to do so. Tuberculosis was found in the cæcum and elsewhere. He was given gas and oxygen by Mr. Page, and the whole procedure went off well; there was no rigidity nor anything uncomfortable throughout the operation. His blood-pressure before the operation was the same as it had been during the three or four previous weeks namely, from 85 to 88. He was under the anæsthetic an hour and a quarter, and when he came round, while still on the table, he winked at him, and his blood-pressure was 100. Next day it had fallen again to 88. Even the same evening as the operation he expressed himself as feeling better. The operation relieved his pain, but of course not his general disease. He died five weeks after the operation, but to within a week of the end he was in comfort. Death was due to perforation of one of the ulcers, leading to septic peritonitis. The operation seemed to have been worth doing because of the relief it gave for two or three weeks.

Mr. H. E. S. Boyle said there were one or two questions which he would like to put to the author. Nothing had been said about the relaxation of the abdominal muscles when the anæsthetic was given for abdominal section; but when he had given gas and oxygen for lengthy operations, it had been difficult to secure such relaxation or to be able to promise that it would be obtained: of course he had not given the preliminary injection of a narcotic nor had he added ether to the gas and oxygen in his cases, and so he would like to know if when these things were done it was easy to get relaxation of the abdominal muscles. Mr. Page said more ether might be added in difficult cases, and that came as a surprise to him, and he had not realized that ether was used in the method described as "gas and oxygen for major surgery." He could confirm the remark that gas and oxygen was the best anæsthetic for diabetics, as he had given it in three such cases where amputation was done, and there were no toxic results. He had been surprised to hear of operations for piles and other rectal conditions, as well as those on the urethra by this anæsthetic, because he had always found that cases in which the sphincters were seriously interferred with were liable to get opisthotonos, which was distressing to both surgeon and anæsthetist.

Dr. LOOSELY said he had had occasion to use Teter's apparatus in six cases, all in children, the eldest being a boy aged 6. This boy had had ether twice before, and was very sick afterwards. For the operation on double talipes he was under half an hour, and was all right immediately following it, with no vomiting. Much value attached to the anæsthetic under discussion for that class of case. The youngest of his series was a child aged 14 months, who had umbilical hernia, and he thought there was some risk in using plus pressure in a child of this age, and so removed the spring from the valve in these cases. He could not keep this patient sufficiently relaxed for the comfort of the surgeon; but turning on the ether kept him quiet, and the open method had not to be resorted to. One patient, aged 18 months, with inguinal hernia, he was able to keep well under and quiet without using ether. In the Children's Hospital, Great Ormond Street, he had been pleased with the after-results. He had not had the opportunity of using the apparatus for adults.

Mr. ASLETT BALDWIN said Mr. Page had given gas and oxygen for him in a number of major operations, for patients who appeared to be in a desperate condition; and he had formed a very high opinion of the method. In some cases where the condition of the patient seemed to show the operation must be finished off in a hurry, gas and oxygen had enabled him to complete it in safety. In other cases he had got through all right where he would have hesitated to operate under any other anæsthetic. It was the ideal anæsthetic in cases where the patient was already partially poisoned by his disease, as in diabetes. He would relate a case to show the advantage of the method. A young woman was under the care of Dr. Beddard, at West London Hospital, on account of acute bronchitis, on which an acute abdominal state supervened, which Dr. Beddard considered urgently needed operation. Under those circumstances one dared not give chloroform or ether. He (the speaker) opened the abdomen, and found the lower part of it and the pelvis filled with blood. The region was carefully explored; the tubes were normal, and as no active hæmorrhage could be discovered, the wound was closed again after the blood had been rapidly mopped out. She seemed no worse after the operation, and made a perfect recovery. The relaxation of the abdomen was quite satisfactory during operation. She had nothing else besides gas and oxygen. In another case he believed ether was given at first. It was that of an elderly woman with a very weak heart, so bad that her doctor thought she would not bear lifting on to the operating table. A year previously a physician had given her only two months to live. She was very stout, and her abdomen was distended, and when he cut down he had to pass through about 5 in. of fat before coming to the abdominal muscles. He removed a gangrenous appendix and sewed her up without trouble. As soon as the inhaler was removed from her mouth she expressed her ability to get off the table and walk back to bed. Her recovery was uninterrupted. Vomiting, though not always absent, was not so troublesome as with other forms of anæsthetic, the recovery was always quicker, and food could be given earlier, which was very important. At St. Mark's Hospital he had done a number of rectal operations, for fissures, small piles, fistulæ, &c., under gas, and he prevented the opisthotonos or tendency to it by trussing the patient up in a jack-towel. The towel was sewn across a suitable distance from one end, the patient was usually placed on the side and the towel passed round the neck and the flexed knees, the thighs being flexed at the same time.

Mr. PAGE, in reply, thanked the President and other members present for the kind way in which they had received his paper. He suggested that the answer to the President's question as to the CO_2 content of the blood and asphyxia was that less N_2O was required after induction than before, especially if some ether had been used during induction, and that therefore more O_2 could be given. With regard to Mr. Bellamy Gardner's remarks as to the expense of the anæsthetic, that was freely admitted, but where rebreathing was adopted the expense was less. Though this fact might prevent its general employment, there were surely some cases in which expense should not be considered in the balance as compared with the safety of the patient. There certainly had been some rigidity of muscles in some cases, but some surgeons did not mind that as much as did others: a large number of his cases had been quite relaxed. Relaxation could not be promised without the addition of ether. He admitted that it was difficult to give gas and oxygen. He had been careful to say it should be in the hands of an expert. He recommended adding ether during the induction period. When gas and ether were given with the Clover's inhaler, in the old days, a prop was not always used, but no doubt it was good practice. If gas and oxygen were given alone a mouth-prop should always be used. He agreed with the fact being emphasized that deaths might occur if it came into general use. He agreed that the after-effects with open ether were good, and at Guy's Hospital they were among the first to employ it, but considered the results better after nitrous oxide and oxygen with ether. The case of the doctor mentioned by Dr. Des Vœux was certainly a bad one, and he (Mr. Page) was condoled with in having to give the anæsthetic for such a case, but he was able to tell the patient that he felt sure that he would get through the operation. He did not recommend gas and oxygen as a routine method for piles and other rectal operations. One fistula case was in a diabetic; he had no trouble with it. A beer-drinker might be given the anæsthetic and kept relaxed, but it would be difficult and would not always succeed. He did not recommend the method for children so young as 14 months. He did not recommend the method unti about the tenth year, and only then if there was some special indication. He had given it for an appendix case in a child aged 5, but he did not advise its use till a later age; but it did very well even at this age for such things as opening a septic joint or bone.

Demonstration of a Modified and Simplified Apparatus for administering Gas and Oxygen without Ether.

By A. L. FLEMMING, M.B.

MR. FLEMMING said that in Bristol for some time they had been using gas in combination with ether for major operations, also gas and oxygen without ether. He was not in a position to purchase a Teter apparatus, and therefore he resolved to get the best form he could. In the Hewitt apparatus he had noticed the absence of facilities for rebreathing. He had also noticed that respiratory depression was very apt to come on after ten minutes with gas and oxygen, which, in the light of the teaching of Mr. Yandell Henderson, might be due to some degree of acapnia. But it was obviated when an apparatus which permitted of rebreathing was used. The apparatus now exhibited by him embodied two or three principles not at present in the Hewitt apparatus. The gas and oxygen entered by separate channels, and were controlled by one valve. If the valve lifted at all, it opened both ports; there was no such thing as a gas valve open while the oxygen or air valve happened to be stuck. Any combination or rebreathing could be secured by these apparatus. The heavier apparatus was carried by a moveable bracket attached to the anæsthetist's table. Two tubes passed to the two bags, and for prolonged major operations he had attached the Clover. The lighter apparatus could be taken to pieces by turning a screw, and there was no complication. He had used this method for a three-hour operation, such as for excision of the rectum.

Demonstration of Elsberg's Apparatus.[1]

By J. F. W. Silk, M.D.

By the kindness of Sir Watson Cheyne, an Elsberg's apparatus for the intratracheal insufflation of ether was shown and its action explained by Dr. Silk.

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[1] Shown at the meeting of the Section on November 1, 1912.

Section of Anæsthetics.

March 7, 1913.

Dr. J. BLUMFELD, President of the Section, in the Chair.

A Brief Eulogy of Joseph Thomas Clover.

By G. BUCKSTON BROWNE.

CLOVER was born on February 28, 1825, and he died, aged 57, on September 27, 1882. His life is a splendid example of what can be accomplished by the power of the will—the gardener, as Shakespeare has it, who can produce from gardens or from human bodies desired results, however indifferent the gardens or bodies may be, for Clover's body was a frail one and his health poor. Some men circumstanced as he was would have made no effort, but he, like so many in our profession, which, whatever its faults, cannot be charged with selfishness, made of his very weakness a stimulus to exertion. He determined that the shorter his life was likely to be, the harder he would work while it lasted. Clover came from the Eastern Counties, a district which can boast of many distinguished medical and surgical names. On turning to the "Dictionary of National Biography," I find that Clover's great-uncle, the distinguished veterinary surgeon of Norwich, who flourished from 1725 to 1811, Mr. Joseph Clover, is judged worthy by the editor, the late Sir Leslie Stephen, of half a column of notice. This great uncle early in life followed his father's calling of blacksmith and farrier in Norwich, but by extraordinary application he so far mastered Latin and French as to be able to read in the original the best authors on veterinary surgery and medicine. He subsequently devoted his life to veterinary science; he invented an apparatus for the cure of broken tendons and bones in horses, and in 1753 he became the discoverer of the manner in which the larvæ of bots are conveyed from the coat of the horse into its stomach. We have thus a very interesting example of qualities appearing in one generation after another in the same family.

for the Clover of whom we speak to-night was in his very essence a creator, an inventor; and this quality of mechanical invention deserves, I think, to be called genius, for fundamentally genius means the begetting, the creating faculty. Clover was educated at University College Medical School and Hospital. He was a student there when anæsthesia first came before our profession as a practical matter (1845), and he at once ardently threw himself into its special study. His admirers can claim him as the pioneer anæsthetical specialist, for he immediately devoted himself as a practitioner entirely to the assistance of surgeons and dentists in their more painful operations. He was a man of perfect punctuality and method, patient and kind, not only to his patients, but to the younger professional men with whom he came in contact. He was generous with regard to fees, naming his fee, but always accepting the *bona fides* of his colleagues when they, as is often the case, have to plead mitigating circumstances. In presenting this frame to the Section containing his portrait and an important autograph letter, which I am so glad that you have accepted, I feel compelled to say that I think when Clover decided to throw in his lot entirely with the fortunes of anæsthesia, that anæsthesia's gain was general surgery's loss, for Clover was a very great surgical inventor, and one of his inventions in surgery must influence that art as long as the world endures. Benjamin Disraeli asks in one of his novels, What is a great man? and after dismissing gentlemen in Windsor uniforms, *et hoc genus omne*, as not necessarily deserving the name, decides that a great man is the man who influences the mind of his generation, and since Clover influenced the surgical practice of his generation, and probably that of all succeeding ones, I desire distinctly to claim for Clover the title of a great surgeon. I base his greatness upon his contribution to the surgery of stone in the bladder. One of the greatest boons conferred upon suffering humanity during the surgically glorious nineteenth century by our generous and unappreciated profession was the gift of lithotrity, or the art of removing a stone from the urinary bladder without the employment of the knife. Civiale, of Paris, was the father of the art of actually breaking up and crushing the stone as it lay in the bladder by instruments introduced by the natural passage, but Clover, of London, was undoubtedly the father of the art of completely and immediately removing from the bladder all the fragments so made. His evacuating tubes and rubber bottle alone rendered possible the modern operation of lithotrity at one sitting, and so conferred an inestimable boon upon mankind. And more even than this can be said, for while no single man can claim the modern lithotrite or stone crusher as his invention, Clover's evacuating apparatus is practically as useful to-day as it was when first made public in the pages of the *Lancet* of May 12, 1866[1]—for I should be content and happy to use it, just as it left his hands, if I had to perform lithotrity to-morrow. All modern evacuating bottles or aspirators are essentially Clover's. Great modifications have been suggested and employed by Sir Henry Thompson, and by Professor Bigelow, of Boston, and many others, but all have been abandoned, and the evacuator of to-day is essentially and practically Clover's. The same can be said of Clover's evacuating tubes; he urged that they should be short, not of large calibre, and furnished with stylets. Since his paper was published, long tubes, large tubes, and non-styleted tubes have been used and recommended by operators—and have all been abandoned. Clover proved that the smaller the tube the brisker the current of water in it, and that the shorter the tube the more rapidly the broken stone was withdrawn and caught by the bottle. Much might be said about Clover's minor surgical inventions and suggestions, but I have said all that I think is really necessary, for I am imbued with a sincere desire to be just, and nothing more. The last century was, surgically speaking, the most marvellous that the world has ever seen, and throughout that century the surgeons of the British Empire led the van of the army of progress. What I have said of Clover can only be said of some dozen or so of these British pioneers—namely, that he was an originator, and has left an abiding mark upon the practice of his profession. I am, therefore, justified in calling Clover a genius and a great surgeon.

Mr. H. BELLAMY GARDNER contributed the following brief biography: Mr. Joseph Thomas Clover, whose framed photograph, together with two autograph letters, have been presented to this Section of the Royal Society of Medicine through the kindly generosity of Mr. Buckston Browne, was born at Aylsham in Norfolk in 1825. He received a general education at Grey Friars Priory, Norwich, became a pupil of Mr. Gibson, a surgeon of large practice in that city, and, in 1842, a dresser at the Norfolk and Norwich Hospital. An attack of pulmonary disease, which subjected him to periods of disabling illness for the rest of his life, occurred at this time, and kept him away from his duties for four months. In 1844 he came to London and entered as a student at University College Hospital. He was regarded by the staff and his fellow-students as one of the most promising and prominent men of his time, and became house surgeon to Mr. Morton and then to Mr. Syme in 1848, and later in the year was elected

[1] *Lancet*, 1866, i, p. 515.

resident medical officer. Mr. Syme subsequently offered him a similar post at the Edinburgh Royal Infirmary, but this he was obliged to decline. The duties of his office, which he held for five years, included the administration of a large number of anæsthetics from the time of their introduction into surgical practice on December 19, 1846, onwards. In 1850 he obtained the Fellowship of the Royal College of Surgeons, and in 1853 he moved to No. 3, Cavendish Place, London, W., where he continued to live throughout his career until his death in 1882. The state of his health determined him to practise exclusively the administration of anæsthetics, for in this he had already become pre-eminent. He subsequently became administrator of anæsthetics at the Westminster and University College Hospitals, and at the Dental Hospital of London. His mind was markedly of a mathematical, constructive, and inventive order, and his knowledge of mechanics sound and thorough, so that, when the potency and dangers of chloroform first began to be realized, he set to work, and after many trials constructed a portable bag which could be filled with a mixture of 4 per cent. of chloroform vapour and air. This is described by him in an article written in 1868, but was employed in practice seven years before. Besides this bag, which has now gone out of use, anæsthetists owe to him the design of a *facepiece* as superseding a mouth-tube, which was the early method of administering nitrous oxide, also the *initial idea* of mixing nitrous oxide with ether during the induction of anæsthesia, and the invention and *construction* of both the large gas and ether inhaler and the portable regulating ether inhaler, which latter has been and remains in general use since his time. These are indeed a valuable legacy to our speciality, and in addition to his great work in instilling confidence into the profession as to the possibilities of safe anæsthesia during surgical operations, constitute a record by which his successors may still feel truly inspired.

Mr. Clover's contributions to medical literature are as follows:—

"On the Administration of Chloroform through the Nostrils." *Lancet*, 1868, i, p. 231.

"On the Administration of Nitrous Oxide" (introduces the Facepiece). A Paper read at the Annual Meeting at Oxford on August 22, 1868; *Brit. Med. Journ.*, 1868, ii, pp. 201, 491.

"Chloroform Accidents," *Brit. Med. Journ.*, 1871, ii, p. 33.

"On a Case of Fatal Syncope during the Administration of Chloroform" (containing a description of his apparatus for the delivery of 4 per cent. of chloroform vapour and air from a bag), *Med. Times and Gaz.*, 1874, i, p. 693; *Brit. Med. Journ.*, 1874, i, p. 817.

"On an Apparatus for Administering Nitrous Oxide Gas and Ether Singly or Combined," *Brit. Med. Journ.*, 1876, ii, p. 74.

"Portable Regulating Ether Inhaler," *Brit. Med. Journ.*, 1877, i, p. 69.

"Laryngotomy in Chloroform Asphyxia," *Brit. Med. Journ.*, 1877, i, p. 132.

"Death from the Administration of Nitrous Oxide Gas," *Brit. Med. Journ.*, 1877, i, p. 439.

Dr. DUDLEY BUXTON said that when Mr. Buckston Browne claimed that Mr. Clover was a genius, one definition of a "genius" passed through his mind. A genius was one who was capable of taking infinite pains. It was certainly true that Clover was essentially a man who was able to take pains; and this he did.

It had been his (the speaker's) privilege to look through the important papers which Mr. Clover had left unpublished, and on going through them he was greatly impressed by the extraordinary power of taking in details and attending to details which had culminated in the successes which Mr. Clover had been able to place before the world—successes which had impressed Mr. Buckston Browne to regard him as a great surgeon, and which enabled those in that Section to regard him as a great anæsthetist. Those who enjoyed the aftermath of his labours did well to render homage to that great man, and to recall the story of the progress which his efforts had made possible in the employment of anæsthetics. He (the speaker) feared that in these days men were too apt to accept as granted the work of those who laboured in the days gone by; there were many discoveries which were claimed to have been made to-day which in reality had been discovered before, but had become hidden in *Transactions* or other obscure places. Clover was a man who wrote comparatively little, and therefore a great deal of the merit of his work had sunk into oblivion. Yet it was preserved in the hearts of some of them, those who had had the privilege of knowing him, and those who had had the curiosity to search through the literature which remained dealing with his work. Still, Clover left behind him enough to show that not only was he a man who advanced his subject, but one who really had that prophetic power which some great men possessed, and the ability to anticipate discoveries. It would be tedious for him to labour the matter, but he might be allowed to devote a few moments to speaking of some of the obvious advances which Clover had effected, and which had left their impress upon our ideas to-day, and upon our medical work. Clover was merely a name to the student of to-day, the name of one who invented an inhaler. But he was far more than this. Clover, at the time of that very important Committee which owed its inception to the Royal Medico-Chirurgical Society, the Report of which was issued in 1864, though not sitting upon the Committee, was an assessor to it, and the scientific and technical adviser to it. Further, Clover, he believed, also supervised the experiments which were undertaken by that Committee. In this report it was interesting to remember it was said that chloroform undoubtedly was a dangerous drug, but that ether, although less dangerous, was not of such general use, owing to the long time which ether took to produce narcosis. He could not help thinking that this finding of the Committee rankled in the mind of Clover, and induced him to determine to amend the matter and demonstrate that not only was ether a safer drug, but that it should be as satisfactory, with scientific handling, for the purpose of anæsthetization, and as manageable as its younger claimant to popularity, chloroform. And so a little over ten years afterwards Clover produced his "smaller portable regulating ether inhaler," and that at once produced a revolution in the method of giving ether. The older plan of soaking a towel with quantities of ether, or using it in covered masks containing sponges or flannel, was brushed aside, and an attempt was made to use it scientifically. Another great advance was made when Clover indicated that the initial difficulty of giving ether, the feeling of suffocation and distress when the patient first drew breaths, retching, gagging, and so on, could be obviated if induction was brought about by means of nitrous oxide. This procedure overcame the initial difficulties and the dangers due to the holding of the breath, while struggling was diminished if not abrogated. Clover thus indicated that ether could be used, providing there were no conditions of the lungs or bronchi to contra-indicate its employment. But that did not cover all the work which Clover did for anæsthetics. He was imbued with the teaching of Snow, whose memory Dr. Dudley Buxton feared was not kept as green as it should be at the present time. Clover never lost sight of the value of dosimetric methods of giving chloroform. His well-known chloroform inhaler was devised to give exact percentages. An able article, which appeared posthumously in "Quain's Dictionary," placed the order of safety of the different methods of giving chloroform in the following sequence, the most dangerous being placed first: The folded towel, lint, then dropping upon a Skinner's mask, then the inhaler of Snow, which he extolled, and then finally his own inhaler, which he modestly said was not of universal application, owing to its cumbersome appearance and the necessity of acquiring a more difficult technique. He (the speaker) might remind the meeting that by means of that inhaler Clover was, next to Snow, the man who taught, or attempted to teach, the importance of administering chloroform in definite, known percentages. In his apparatus he mixed 33 minims of chloroform in 1,000 cubic inches of air, and thus got a mixture in the large bag which was of known percentage strength. He set forth in the most careful manner to elaborate his designs for instruments for giving ether in succession to nitrous oxide, the result of both careful study and close attention to details. He had seen Clover's diagrams, which revealed a gradual evolution from the simplest ideas, and culminated in the finished instrument, evidencing that Clover had looked at the matter from every point of view. In fact, he dealt with his problem as a genius, and nobody but a genius would have carried out what Clover did. He was a skilled mechanic, and a most capable man with his hands. But he was not a mere mechanician; all his work was based upon profound scientific knowledge of his art. He recognized that the practice of anæsthesia was an art based upon a science, and it was because he had this accurate science behind his art that Clover was what he was. Those interested in the matter, who could consult the few papers he had left behind, would find that a great deal of the teaching of to-day, which seemed to be modern, was really adumbrated by Clover in his writings. For example, he recognized the important mode of speaking of narcosis: he did not use the common term now employed, "degrees of anæsthesia, partial and light anæsthesia," but he spoke of narcosis, and a particular degree of narcosis he regarded as anæsthesia. Another important piece of work which Clover did was to investigate the effect of using electric currents in stimulating the heart which was failing owing to an overdose of chloroform. He demonstrated the danger of employing that method. For many years after Clover's death it was still the custom to carry round a small battery by which the last hopes of the patient's recovery were put to an end through the dangerous procedure of stimulating the region of the stellate ganglion. What more should one say of Clover? He could say many more things. Already Mr. Buckston Browne had spoken of his kindness and of his extreme modesty. It would be found that great men were modest, because they recognized how far below their ideals they themselves were, and must ever be. The small man believed he had attained to the top of the mountain; but the great mind saw other mountains soaring higher beyond. He congratulated the Section in doing homage to one who had achieved so much for the work upon which the Section was engaged. He believed it was good for them to pause awhile in their technical art, and to burn incense before the altar of men like Clover, and to hope that the burning of that incense might make many practitioners strive to advance their calling as Clover undoubtedly did.

Technique in General Anæsthesia for Intranasal Operations.

By H. Bellamy Gardner.

THE system for establishing suitable general anæsthesia for intranasal operations, which I now venture to describe, has been slowly evolved during the course of many years, and appears to produce the best conditions for the operator's manipulations, while protecting the patient from all risk of intercurrent asphyxial strain. It depends *primarily* upon the mechanical and physiological principle that while surgical work is being carried on in the nasal passages the patient's respiration should be entirely conducted through the mouth. This is effected by the insertion of a captive post-nasal sponge directly anæsthesia has been established.

The reasons for the observance of this first principle are, in the first place, to prevent blood and other fluids from draining backwards out of the nasal cavities into the pharynx, œsophagus, and trachea, and this is necessary because their presence in the upper air passages results in constant movements of swallowing and coughing in light anæsthesia, with irregularities in the respiratory rhythm, which interfere with the intake of the anæsthetic vapour and introduce an asphyxial factor which may depress the circulation. In the deeper stages of anæsthesia such fluids may pass the epiglottis and invade the trachea and bronchi, producing a partial or complete obstruction to the entry of air into the lungs and the risk of septic pulmonary infection. Secondly, by cutting off the nasal cavities from the respiratory tide, spraying of blood over the operation area from behind forwards at each act of expiration cannot occur; and thirdly, the vicious circle induced by free blood in the upper air passages, with coughing and swallowing, followed by venous turgescence, and therefore renewed oozing of blood from the mucous membranes of the operation area, is entirely avoided.

The second principle I desire to establish is the necessity for the proper employment of cocaine and adrenalin as an adjuvant to operations within the nasal passages, for when applied to the mucous membranes at a sufficient interval for its adequate absorption—namely, one hour before the operation—the combined solution of these drugs will cause ischæmia, and also by a local anæsthetic action blunt and retard the stimuli, which, in passing from this highly sensitive area, are apt otherwise to cause a certain degree of circulatory shock.

The third principle is that in the absence of certain pathological conditions of the circulation, and with the feet remaining horizontal, the upright or sitting position of the patient is permissible from the very induction of anæsthesia; provided that the patient be not shaken or moved about, his tongue not allowed to fall back against the pharynx, and that the early stages of the third degree of anæsthesia be not exceeded.

The technique of the surgeon and anæsthetist, after adopting these principles, takes the following form: Two nights before the day of operation the patient is given a suitable purgative, for if this be done the night before the operation a somewhat low blood-pressure and tendency to faintness supervening from its action may not have passed away. One hour before the time for operation, either the operator himself or a skilled assistant carefully packs the interior of the nose over the required area with pledgets of wool soaked in a mixture consisting of equal parts of 5 per cent. of cocaine and 1 in 1,000 adrenalin solutions, the strength of each drug in this final mixture being thus reduced to 2½ per cent. and 1 in 2,000 respectively. On his arrival the anæsthetist examines the patient's heart and circulation and in the absence of physical signs of aortic valvular disease, marked anæmia, very low blood-pressure, or history of fainting attacks, proceeds as follows: On a shelf by the side of the operating table he arranges his apparatus, which includes drop bottles for the C_2E_3 mixture and chloroform, a Junker's chloroform bottle with foot-bellows, a Skinner's mask, two honeycomb throat sponges wrung out of sterilized water, held in Rampley's sponge forceps, a tongue-clip, a dental mouth-prop, a Mason's gag, and a bowl of hot water. A stool for him to stand upon or a plank resting on the bars of two chairs is placed behind the head end of the operating table, and with the usual ablutions and aseptic sartorial adornments, he is now ready.

The patient is now brought in and seated upon the operating table, the head end of which is raised at an angle of 45° or 50° from the horizontal, his clothing over the neck and shoulders is arranged as the operator desires, he is covered with two thick blankets, and his head is wrapped and pinned up in a towel to protect the hair from contact with the surgeon. He is then propped into position with two pillows so that he will not, after this, require to be moved about in the very least. A small dental mouth-prop is now inserted between his teeth or gums on the right side, and the anæsthetist, standing on the stool behind, takes the Skinner's mask in his left hand and with the palmar margin supports the patient's lower jaw against the mouth-prop. He then administers either the C_2E_3 mixture for a few minutes first or chloroform *ab initio*, according to judgment. When the patient has arrived at the early stages of the third degree of anæsthesia, the corneal upper eyelid reflex having become only weakly active, the patient's mouth is opened wider with the Mason's gag, the tongue-clip inserted into the tongue and the latter drawn gently forward and retained in place by the left hand of the administrator which still supports the chin. The terminal tube from the Junker's bottle is passed into the left side of the patient's mouth and chloroform vapour pumped in at each inspiration by means of the foot-bellows. The surgeon now inserts either one large or two small sterilized honeycomb sponges into the post-nasal space, each tied to a piece of tape 15 in. long, which is left hanging out of the mouth. The mouth is kept open either with the Mason's gag or the mouth-prop, as may seem convenient, and if the stage of anæsthesia be now as desired, all is ready for the operator to begin.

If rightly and thoroughly carried out by this system, the anæsthesia remains perfect throughout, however long the work may take, and the loss of blood during the operation of submucous resection of the nasal septum is often less than is caused by the extraction of one ordinary tooth. I cannot truthfully say this of any other method known to me, and though till now I have only discreetly withheld approbation from the work of surgeons who make hurried plunges into the gory darknesses of partly asphyxiated patients not prepared upon this system, I now think that the latter ought not to be subjected to such risks, as they often otherwise have been, for the sake of the particular operations under discussion.

The same technique is capable of adoption for all intranasal operations excepting those requiring the insertion of the surgeon's index-finger through the mouth into the post-nasal space, such as for the removal of the posterior ends of enlarged turbinal bones. These are better accomplished with the patient lying down upon his side to provide drainage for the usual very free effusion of blood immediately resulting. The great advantage to the surgeon in being enabled to see the parts within the nose in their normal relations exactly as he did when examining them for the first time in his consulting room, so facilitates his difficult work that I consider the upright position is positively called for in intranasal work; and, although I do not maintain that it is at all wise to give chloroform to patients seated upright without a considerable personal experience of anæsthetic administration, yet, after carefully studying the subject in a large number of patients in which this position has been adopted, I do not think it produces so marked a lowering of blood-pressure and anæmia of the brain as is usually supposed, and I believe that it is far safer than raising the patient's head after he has been put to sleep lying flat down, for this is quite certain to produce faintness; a result which may also follow any other alterations of his posture while under chloroform.

Patients requiring radical operations upon the frontal or maxillary sinuses are also best prepared by this system, as there may be free fluid in the nasal cavities at any moment during either of them; but, as in the case of frontal sinus operations, there is no particular advantage to the surgeon in the upright position, and considerable circulatory shock, which cannot be locally obtunded, is often produced by hammering and chiselling the bones of the skull, these may be performed with the patient in the supine position. I am sure that the perfection of operative ischæmia cannot be attained, even by half an hour's previous packing of the nose with cocaine and adrenalin, and as for their insertion five minutes before the operation, as is the custom of some surgeons, I regard it in the same light as Sydney Smith, who, when he saw some one patting the shell of a tortoise in the Zoological Gardens, said if it was intended to make the creature happy, it was about as effectual as stroking the dome of St. Paul's to please the Dean and Chapter.

DISCUSSION.

Dr. BARTON said that as anaesthetist to a throat hospital for ten or twelve years he had listened with great pleasure to the paper, though he did not feel in entire agreement with all the views expressed. Mr. Gardner seemed to prefer the upright posture for cases in which he (the speaker) generally found surgeons did not require it, though with the patient on the table, the head of the table was not infrequently slightly raised. Almost the only operation for which he found the upright position used was turbinectomy, but for that Mr. Gardner recommended the lying-down posture. As it was a brief operation, gas sufficed for most surgeons. Immediately the operation was done the patient's head could be pushed forward, and there was no trouble from hæmorrhage. He had not noticed mention of the previous injection of scopolamine and morphine, from which for submucous resection he found the greatest benefit. The injection should be done at the same time as the nose was packed, and in an hour the patient was fully under its influence. In these cases there was very little bleeding, and so it was safe to give the alkaloids; but they were better avoided in frontal sinus operations and some others in which hæmorrhage was considerable. He agreed as to the usefulness of foot-bellows, as they saved a hand. A very good form was now made which fitted over the bellows of the Junker inhaler, and was inexpensive. He did not find that sponges in the post-nasal space were so generally used nowadays. Even if the bleeding should exceed what was expected, he found it easy to check its flow into the pharynx, his usual plan being to push up the soft palate with a sponge affixed to a holder: this could be at once removed or replaced as required. For frontal sinus operations, where there might be continual trickling into the post-nasal space, a sponge in the nasopharynx was useful.

Dr. McCARDIE wished to refer particularly to operations on the nasal septum. He believed that most septal operations were carried out under local anæsthesia; perhaps in only one in eight was a general anæsthetic used. The amount of general anæsthetic administered should be minimal. He and others had had cases in which more than a few drops of adrenalin and a weak solution of cocaine injected during anæsthesia had caused untoward symptoms. The lying-down posture was the safer. Mr. Seymour Jones, Assistant Surgeon to the Ear and Throat Hospital, Birmingham, had devised a simple curtain mask, which Dr. McCardie showed, for use during the operative anæsthesia. Anæsthesia was induced by a mixture of two parts ether to one of chloroform, and when the patient was lightly under, the little curtain of gauze was fixed by strapping across the upper lip and to the towel which covered the head. The curtain was thrown down over the mouth, and prevented contamination therefrom, and protected the surgeon from the vapour inhaled by the patient. Before the mask was fixed the face was vaselined, a Doyen's gag was inserted and half opened in the patient's mouth, and the anæsthetic was simply dropped on the part of the curtain over the mouth. In view of the sometimes dangerous effects caused by adrenalin, it was important to use a fair proportion of ether. He had seen faintness, and even syncope, caused by the sudden injection of adrenalin during moderately deep anæsthesia. Consequently he used only light anæsthesia, his object merely being to keep the patient immobile. That was contrary to what was stated by Dr. Levy at the last meeting of the British Medical Association—namely, that animals were only safe from syncope, after the injection of adrenalin, when they were well anæsthetized by chloroform. He (the speaker) could not explain the difference of experience. Under local anæsthesia variations in the circulation and in the vasomotor system, due to the adrenalin, were compensated for automatically: the inference was that during light general anæsthesia there was better compensation than during deeper anæsthesia.

Mr. H. E. G. BOYLE said that the technique of the surgeons with whom he worked differed from that just described. Mr. Harmer and other surgeons thought it better to have the patient lying on his side, putting a sandbag beneath the shoulders and head: then any bleeding ran down and collected in the right cheek. After adopting that posture for a time it was found unnecessary to use post-nasal sponges, to the use of which trouble in the ear had been attributed on one or two occasions. He agreed with what Mr. Bellamy Gardner said about the dental prop, because most of these patients came on the table to be anæsthetized with a nasal plug in position, and without a dental prop they clenched their teeth and would not breathe properly. He agreed that it was safer to anæsthetize patients in the sitting posture than to have them lying down and then raise them after they were anæsthetized, but he recommended Mr. Gardner to suggest to the surgeon that he should have the patient on his side, and sit down and do the operation in comfort. He wished he could say, as Dr. Barton did, that in his experience turbinectomy was a brief operation. He had had cases in which it occupied forty-five minutes. With regard to the apparatus with the curtain, he was surprised that the Junker tube was not used after the operation was started, because if the patient was anæsthetized with gas and ether, and the Junker inhaler was used afterwards, very little chloroform would keep the patient quiet. He often had to work with the whole face covered with gauze, and the tube of the Junker in the side of the mouth. The condition of the patient could be gauged by the regularity of the breathing: it was not always necessary to observe the pupils.

Mr. BELLAMY GARDNER, in reply, said he was glad to hear that Dr. Barton agreed that if the patient was sitting up he should not be in a chair. When Kraske's operation was first done, with the patient's legs hanging over the end of the table, anæmia of the brain and faintness resulted; whereas if the patient were on his side on a table severe rectal operations could be done without producing so much shock. He therefore did not agree to anæsthetize a patient with the feet hanging down. Turbinectomy took various forms, and if done with a spokeshave it was soon over: what he was referring to was removal of the enlarged end of turbinals, after which, when there was a good deal of bleeding, it was better to let the patient down flat to permit of the escape of the blood. With regard to morphine and scopolamine, he agreed with Dr. Barton that if it were used, the proper time was an hour beforehand. For most nasal operations he did not use this preliminary injection; the patient was duller on coming round, and he did not think much good was gained by it. The foot-bellows he used was simply a hand-bellows with a flat side made of lead. The question of injection of alkaloids under the mucous membrane was discussed at a meeting of the Section two years ago. Extraordinary faintness and three fatalities had been recorded, and he referred to it to call attention to the efficacy of laying on the mucous membrane a weak solution of cocaine and adrenalin: the effect of that was quite different and very marked. He agreed with Mr. Edmund Boyle that to have the patient lying on his side was the ideal position for anæsthetization, for then, even if the tongue became paralysed, asphyxia would not occur; but it would be difficult to persuade the abdominal surgeon to agree to this posture. He avoided the use of ether altogether in nasal operations, except in a mixture. If the patient was pale from fear, he flushed him first with a little C.E. mixture. The object of his paper had been to prove that with the method advocated there need be no movement or other untoward factor in the operation throughout.

Scopolamine-Morphine-Atropine as a General Anæsthetic.

By LIONEL E. C. NORBURY, F.R.C.S.

IN this short communication it is my wish to detail a few personal observations upon the use of a mixture of scopolamine, morphine and atropine when employed as an anæsthetic in certain cases where inhalation anæsthesia would appear to be contra-indicated, or at any rate inadvisable. My experience of the use of this combination of drugs for such a purpose is limited to six cases (admitted to St. Thomas's Hospital), but nevertheless this small amount of material has been sufficient to enable one to form an opinion regarding the usefulness of this variety of anæsthesia in certain circumstances.

"Scopolamine-morphine-atropine" as an anæsthetic is especially valuable in patients suffering from diabetes, phthisis, certain respiratory conditions, and in severe cardiac or arterial disease. The cases in point have all been amputations of the limbs, chiefly for senile or diabetic gangrene, and as in these latter chronic bronchitis or other lung trouble is frequently co-existent, the employment of inhalation anæsthesia has been thought inadvisable. Again, in cases of diabetes the danger of "coma" supervening is a very real one.

METHOD OF ADMINISTRATION.

A hypodermic injection of a solution containing *morphine tartrate* $\frac{1}{6}$ gr., *hyoscine hydrobromide* $\frac{1}{75}$ gr., and *atropine sulphate* $\frac{1}{150}$ gr. is usually given two hours before the operation. This quantity is often sufficient, but should it not be, a further injection of $\frac{1}{4}$ gr. of morphia alone may be given fifteen minutes before commencing the operation.

The patient should be inspected half an hour before the operation.

in order to judge whether the initial dose has been sufficient or not. A second injection of scopolamine-morphine may be given if thought necessary, but it would appear safer to administer an injection of morphia alone in cases in which the initial injection has failed to produce satisfactory anæsthesia. In three of the cases local infiltration of the skin and subcutaneous tissues with a 1 per cent. solution of novocain in the line of the incision was employed in addition, but this does not seem to be a necessary adjunct, since the multiple punctures with the injecting needle were well tolerated, and equally good results were obtained when scopolamine mixture was used alone. Nevertheless, local infiltration may be of value in cases in which anæsthesia is not sufficiently deep at the time of operation. In one case of amputation through the thigh for "diabetic gangrene" a hypodermic injection of morphine tartrate $\frac{1}{4}$ gr., hyoscine hydrobromide $\frac{1}{100}$ gr., and atropine sulphate $\frac{1}{150}$ gr., was given one hour before operation. The sciatic nerve was exposed between the tuberosity of the ischium and great trochanter, and was infiltrated with 1 per cent. novocain solution. The anterior crural nerve was similarly exposed below Poupart's ligament and infiltrated.

On commencing amputation through the middle of the thigh slight pain was felt, and so gas was given to complete the skin incision, and the rest of the operation conducted in comfort. There was no sign of shock, and no increase in the pulse-rate. The patient slept for some hours, and complained of no pain on waking.

In two cases, scopolamine-morphine-atropine mixture was employed alone to produce anæsthesia. One of these was an amputation through the thigh for senile gangrene, in a male, aged 85, who three and a half weeks previous to admission had suffered from an attack of cardiac failure. Complete anæsthesia was obtained two hours after the injection. There was no change in the pulse- or respiration-rate, and recovery was satisfactory. The other case was an amputation through the arm, just above the elbow-joint, in a very feeble woman, aged 75. The condition present was a septic tuberculous wrist. Anæsthesia was incomplete two hours after the initial injection, and so a further injection of $\frac{1}{4}$ gr. of morphia was given just before the operation. The respiration- and pulse-rate did not vary throughout, and the operation was carried out in comfort. Four hours after operation the respirations dropped to eight per minute, but the patient responded readily to a hypodermic injection of strychnine. No post-operative pain was complained of, and recovery was satisfactory.

Of the six cases operated upon (of which a short account of each is given later) there were five males and one female. The ages varied from 56 to 85; four were amputations through the thigh and two through the arm. The operations were performed for diabetic gangrene in two, senile gangrene in two, cellulitis of hand and gangrene of fingers in one, and septic tuberculous wrist and forearm in one. There were no deaths in this short series of cases. One diabetic patient developed gangrene of his remaining foot eleven weeks after operation, and died comatose a few days after amputation through the thigh, performed under spinal anæsthesia.

In successful cases where scopolamine-morphine-atropine is employed there is complete anæsthesia, and the patient has the appearance of being in a deep sleep. The pulse and respiration are normal and unchanged, the pupils are small, the conjunctival reflex is absent, the corneal reflex present but sluggish. In some cases the respiration is slightly stertorous, and the patients usually sleep soundly for some twelve hours after operation. In one case the respirations dropped to seven per minute immediately on completion of the operation, while in another case already mentioned the respirations dropped to eight per minute some four hours after operation; this was accompanied by Cheyne-Stokes breathing, but the pulse was unaffected.

In each of these cases the condition lasted for about half an hour, the patient responding well to a hypodermic injection of strychnine. These instances afford evidence that scopolamine-morphine acts principally on the respiratory centre. Of the deaths which have occurred from these drugs, the great majority have been due to respiratory failure with cyanosis. This condition does not appear to be merely a depression, but it is accompanied by Cheyne-Stokes breathing, suggestive of paralysis of the medullary centres. It is often difficult to secure complete muscular relaxation when employing scopolamine-morphine alone, and so it would not be an ideal anæsthetic in abdominal operations, &c.

In one case of amputation at the seat of election for chronic ulcer of the leg, not included in the above series, an injection of morphine tartrate $\frac{1}{5}$ gr., hyoscine hydrobromide $\frac{1}{50}$ gr., atropine sulphate $\frac{1}{100}$ gr., given two hours before operation failed to produce sufficient muscular relaxation. The patient was aged 47. At the time of operation he was comatose, respirations 16 per minute, pulse 80 per minute, but his limbs were in a spastic condition. It was necessary to administer a small amount of chloroform in order to overcome the spasm. There was no post-operative shock, no increase of pulse-rate; consciousness returned ten hours after injection, and recovery was satisfactory.

It is in operations upon diabetic patients that scopolamine-morphine would appear to be especially useful as an anæsthetic. Johnson, in an article dealing with "Amputation for Diabetic Gangrene,"[1] says, "The chief dangers attending amputations for diabetic gangrene are: (1) dangers from the anæsthetic, (2) shock, (3) sepsis."

With regard to the former, the chief danger is acidosis, due to the circulation of fatty acids in the blood, which are in some way responsible for the onset of diabetic coma. According to Pavy,[2] coma is the result of the accumulation of CO_2 in the tissues, the alkalies of the blood being neutralized by the fatty acids, and so there is little alkali left to convey the CO_2 from the tissues. Since acidosis is in some way responsible for the onset of diabetic coma, and since post-operative coma occurs in some 10·6 per cent. of cases, it is obviously important to take every precaution to prevent the occurrence of acidosis, and to avoid the use of anything that will increase the condition, if already present. A certain amount of disturbance of fat metabolism commonly occurs after the administration of anæsthetics, such as chloroform or ether, as shown by the presence of acetone and diacetic acid in the urine, and this is especially so after the use of chloroform, producing the condition of so-called delayed chloroform poisoning. Post-anæsthetic fatty acid intoxication has been specially noticed in conditions associated with fatty changes in the liver—e.g., diabetes, sepsis, rickets, starvation, &c.

(2) Shock is a danger to be carefully avoided in operations upon diabetic patients. In the cases in which we have employed scopolamine-morphine anæsthesia there has been no evidence of shock, and in this connexion it would appear to be a suitable anæsthetic to employ.

(3) The third danger—namely, sepsis—although a very important one, on account of the poorly nourished tissues of the patient and the liability for sloughing to occur, does not come within the scope of this paper.

Finally, while bearing in mind the very limited experience that one has had in the use of scopolamine-morphine-atropine as a general anæsthetic, I think that one may justly draw the following conclusions from the cases in point:—

That scopolamine-morphine-atropine mixture has a field of use as a general anæsthetic in operations on diabetic patients, and especially so in amputations for gangrene associated with diabetes.

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[1] *Brit. Med. Journ.*, 1910, i, p. 801.

[2] *Lancet*, 1908, ii, pp. 1727-40.

It is similarly useful in amputations upon very feeble patients, the subjects of chronic bronchitis, &c., or suffering from advanced disease of the circulatory system, and where it is of great importance to avoid "shock."

When employed alone it is of little use in cases where complete muscular relaxation is essential, but in this connexion it has its advantages as an adjunct to inhalation anæsthesia: the patient being semi-conscious at the time that the inhalation anæsthesia is commenced, less anæsthetic is required, shock is reduced to a minimum, and sleep is secured for some hours after the operation.

ABSTRACT OF CASES.

Case I.—T. D., male, aged 77; senile gangrene, right foot. No glycosuria; nocturnal delirium; advanced arterio-sclerosis. Injection of morphine tartrate $\frac{1}{6}$ gr., hyoscine hydrobromide $\frac{1}{62}$ gr., two hours before operation. Further injection of morphine tartrate $\frac{1}{8}$ gr., hyoscine hydrobromide $\frac{1}{75}$ gr., half an hour before operation. At operation patient semi-comatose; respirations 16; pulse 80 per minute; pupils small, conjunctival reflex absent; corneal reflex sluggish. Local infiltration of skin with 1 per cent. of novocain. Amputation lower third of thigh. No reaction on dividing main nerve-trunks. No shock. Slept for twelve hours. Good recovery.

Case II.—G. C., male, aged 85. Senile gangrene right foot. Attack of "heart failure" three and a half weeks previously. No glycosuria. Injection of morphine tartrate $\frac{1}{6}$ gr., hyoscine hydrobromide $\frac{1}{75}$, atropine sulphate $\frac{1}{150}$ gr., two hours before operation. Complete anæsthesia. Amputation lower third of thigh. No change in pulse- or respiration-rate. Recovery good.

Case III.—J. L., male, aged 68. Gangrene right foot; diabetes. Injection of morphine tartrate $\frac{1}{4}$ gr., hyoscine hydrobromide $\frac{1}{100}$ gr., atropine sulphate $\frac{1}{150}$ gr., one hour before operation. Patient drowsy and semi-conscious. Exposure of sciatic and anterior crural nerves. Infiltration of nerves with 1 per cent. novocain. On commencing incision slight pain felt, and so gas administered for skin incision. No shock. No increase in pulse-rate. Patient slept for some hours and complained of no pain on waking. Recovery.

Case IV.—C. W. P., male, aged 56. Gangrene left foot; diabetes. Injection morphine tartrate $\frac{1}{6}$ gr., hyoscine hydrobromide $\frac{1}{75}$ gr., atropine sulphate $\frac{1}{150}$ gr., two hours before operation. Subsequent injection of morphia $\frac{1}{4}$ gr., just before operation. Injection of 1 per cent. novocain in line of incision. Amputation at junction of middle and lower third of thigh. Patient talked incoherently during operation. No sensation till sciatic nerve was divided, when a slight reaction was noticed. Patient did not remember anything about operation. Eleven weeks later, gangrene of remaining foot. Large amount of sugar still in urine. Amputation lower third of thigh under "spinal anæsthesia." Coma and death.

Case V.—E. L., female, aged 75. Septic tuberculous wrist and forearm. Patient very feeble. Injection of morphine tartrate $\frac{1}{6}$ gr., hyoscine hydrobromide $\frac{1}{75}$ gr., atropine sulphate $\frac{1}{150}$ gr., two hours before operation, and injection of morphia $\frac{1}{4}$ gr. immediately before operation. Amputation lower third upper arm. Respiration and pulse did not vary throughout. Respirations, eight per minute four hours after operation, lasting for half an hour and responding to hypodermic injection of strychnine. No shock; no pain. Good recovery.

Case VI.—G. G., male, aged 66. Very feeble; cellulitis of hand and gangrene of fingers. No glycosuria. Injection of morphine tartrate $\frac{1}{6}$ gr., hyoscine hydrobromide $\frac{1}{75}$, atropine sulphate $\frac{1}{150}$ gr., two hours (?) before operation. Injection of morphia $\frac{1}{4}$ gr. immediately before operation. Injection of 1 per cent. novocain in line of incision (not felt). Amputation lower third of upper arm. Patient talked rather incoherently, but answered questions during operation. Pulse and respiration did not alter during operation. Convalescence satisfactory. Recovery.

Scopolamine-Morphine-Atropine as an Adjunct in Inhalation Anæsthesia.

By A. F. Morcom, M.B.

The object of this paper is to emphasize the advantages of a combination of a mixture of scopolamine-morphine-atropine with anæsthesia induced by the ordinary method of inhalation. It is not proposed to furnish lists of statistics to prove facts in connexion with this mixture, but merely to place on record a few observations that have been made during its employment previous to surgical operations for the last two and a half years or so in the wards of St. Thomas's Hospital and in the St. Thomas's Home adjoining it. The solution employed is a combination of three drugs: hyoscine hydrobromide, morphine tartrate, and atropine sulphate, made up into a solution with sterile water in such a proportion that 5 minims contain: hyoscine hydrobromide $\frac{1}{120}$ gr., morphine tartrate $\frac{1}{6}$ gr., atropine sulphate $\frac{1}{100}$ gr.

The method employed is for 5 minims of this solution to be hypodermically injected into the subcutaneous tissues three-quarters or half an hour before operation. The immediate effect produced on the patient varies. Violence and excitement are exhibited in about 1 per cent. of cases, in 30 per cent. of cases no apparent change is produced previous to operation—and this is especially so in the case of men—but in the majority of cases the patient becomes drowsy and falls into a quiet sleep. In those cases where no apparent effect is produced in the initial stage there is marked effect during and after operation, though to a smaller degree. Nicholson, of Chicago, in 1909, in a report on 650 cases so treated, stated that in 94 per cent. of patients there was a quiet condition of the nervous system, and in the remaining 6 per cent. there was no change whatever. In this series there were no cases in which exaltation of the nervous system was apparent.

The advantages of a preliminary injection of scopolamine-morphine-atropine as an aid to a general anæsthetic appear to be manifold. The drowsy condition into which the patients generally fall frequently enables the anæsthetic to be administered without the patient waking up. This seems to be particularly the case in highly nervous patients; a very satisfactory point, as it is these patients who feel most acutely the preliminaries of the operation and the administration of the anæsthetic. In this connexion it should be mentioned that the initial administration of ether is sometimes liable to awaken the patient, owing to its irritability to the bronchial passages, so that it is best to commence the induction with some less irritating anæsthetic—personally, I always use the ordinary chloroform-ether mixture—and then to continue with open ether when the patient begins to go under. If this is done, the initial stage of induction is almost ideal; the patients are absolutely tranquil, there is no struggling or excitement, the respiration is deep and regular, and the pulse is full and slow. It is found that much less anæsthetic is required to obtain surgical anæsthesia, and this is certainly very marked in the case of open ether. Nicholson, in 1909, estimated this reduction at 50 per cent.

When anæsthesia has been obtained the pupils remain contracted and react faintly to light, the corneal reflex being hardly ever lost. This latter fact may render it difficult to judge when a sufficient degree of anæsthesia has been reached, but a good idea can be obtained by paying careful attention to all details, such as the condition of the respiration. The action of the atropine is noticeable owing to the fact that there is great diminution in the amount of the bronchial and salivary secretions, so that it is not necessary continually to clear out the mucus from the pharynx during the administration, and there is less tendency to post-operative bronchitis and pneumonia. Indeed, I do not recollect a single case of either of these complications arising in patients treated in this way. A number of surgeons have stated that it is impossible to obtain thorough relaxation of the abdominal muscles when scopolamine-morphine-atropine has been employed, but

I think this is the exception rather than the rule. It is in some cases due to the small amount of anæsthetic employed, but very occasionally cases do arise in which it appears impossible to overcome this rigidity. I have been impressed by the fact that this rigidity is very rarely a prominent feature in abdominal operations, gynæcological or otherwise, where the entrance into the peritoneal cavity is effected below the level of the umbilicus, and most of the cases in which it does occur are those in which operations are performed in the epigastric region, in which situation the recti show greater development.

After the operation the patient, almost without exception, sleeps uninterruptedly for three hours or more, and this may be considered the most advantageous fact in this connexion, as it does away to a great extent with post-operative shock, and also with the post-anæsthetic vomiting, which is so often such a distressing feature. Felix Rood, in the *British Medical Journal* of September 23, 1911, in a report on 400 cases, states that in 255 there was no vomiting, 120 vomited once or twice, and 25 did so several times. At St. Thomas's, however, the diminution in number of cases of post-anæsthetic vomiting has been even more marked than this, and the Sister of one of the female surgical wards reports that out of fifty consecutive operation cases so treated not one case of post-anæsthetic vomiting occurred. This treatment has been found so satisfactory in the gynæcological ward at St. Thomas's that all operation cases have the preliminary injection as a matter of routine. The small proportion of cases in this ward that have post-anæsthetic vomiting may be gauged from the fact that of the last 127 cases operated upon there only six have vomited; of these six, four vomited only once, and then only slightly, two were considerably affected for twenty-four hours after operation, but one of these latter was an acute case, and the preliminary injection was administered only ten minutes before the commencement of the operation. It should be mentioned that in this series the anæsthetics were administered by different individuals ranging from the senior visiting anæsthetist to the most junior house officers.

A point that is frequently raised by critics of the scopolamine-morphine-atropine injection is that it greatly increases flatulence and constipation following operation, but the atropine tends to counteract the effect of the morphine, and my personal experience is that the patients do not have, as a general rule, any more trouble in this direction than those who have not had the preliminary injection. The dryness of the mouth, caused by the atropine, is sometimes complained of by the patients, but generally proves to be a minor trouble, and can be rectified by frequently washing out the mouth with water.

In conclusion, I would say that roughly 600 surgical cases pass through my hands each year, all of whom I have the personal care of, both before and after operation. Of this number, 40 per cent. have a preliminary injection of scopolamine-morphine-atropine, and it is my honest conviction that these latter have a much better time in every way than those who are not so treated.

DISCUSSION.

The PRESIDENT (Dr. J. Blumfield) said he was particularly interested in Dr. Morcom's contribution, because he had had no experience of what Mr. Norbury had detailed—namely, using the mixture as an anæsthetic without inhalation. With regard to its use in conjunction with general anæsthetics, this had been before the Section before, and opinion was still divided about it among both anæsthetists and surgeons. The objections were based, he thought, on the rigidity met with in abdominal work. He had not met with cases in which the rigidity could not be banished, but he had found it took longer for relaxation to occur. After a considerable use of the mixture, his practice was now to give $\frac{1}{4}$ gr. omnopon, $\frac{1}{100}$ gr. scopolamine, and $\frac{1}{120}$ gr. atropine. It was a great help to the patient, but no help to either the surgeon or the anæsthetist. Sometimes there was a slowing of respiration during the operation owing to the scopolamine. He had several times been thanked by patients who had to have an anæsthetic a second time for giving them the scopolamine; they realized the absence of the discomforts formerly experienced. But he had met with delayed vomiting where scopolamine was used—i.e., for twelve hours following the operation the patient had been slowly recovering, and was apparently comfortable, but then had ordinary vomiting, such as often immediately followed ordinary inhalation anæsthesia. He did not use the mixture as a routine measure except for long operations in cases where the patient was of a robust type. He had known of cases where respiration had been dangerously slowed for hours after operation, but none such had occurred after the use of the mixture he now employed.

Dr. WALTER TATE said he had used the mixture before operations for about two years, in practically all the cases in his ward at St. Thomas's Hospital, and in most of his private operations. In a large number of the cases the patients were distinctly drowsy before the operation, and several had no recollection afterwards of the operation having been done. That in itself was an enormous advantage to the patient. With regard to vomiting, details of 127 cases had been carefully recorded, and in only five of these had vomiting occurred after the operation. The testimony of the Sister and nurses was of considerable value. Whereas formerly the patients had needed constant attention during the night following the operation, since the use of scopolamine and morphine the patients had slept the greater part of the night, and the work of the nursing staff had been rendered much lighter in consequence. It sometimes happened that rigidity of the abdominal muscles required a longer anaesthesia before it passed off, but he (Dr. Tate) had not found any serious inconvenience from this cause. The slight disadvantage which the surgeon might sometimes experience was more than compensated for by the great advantage to the patient. Dr. Tate was therefore strongly in favour of using this mixture.

Dr. LLEWELLYN POWELL said it was more difficult for the nurses, after the preliminary injection had been used, to judge the condition of the patient when put back to bed. One patient suffered from somewhat severe internal haemorrhage and did not show signs of the catastrophe until she was rather far gone.

Mrs. DICKINSON BERRY said the objection mentioned by Dr. Llewellyn Powell was also made in her wards; nurses said there was more anxiety after the preliminary injection had been employed because of the difficulty of judging the condition of the patient. In some cases, too, there seemed to be more vomiting after its use.

Dr. McCARDIE asked why atropine was added to the scopolamine and morphine, which sufficiently stopped secretion. He inquired whether the writers used the mixture for aged people or for children; he himself did not use it for either the very old or the very young, for whom he preferred atropine alone, or combined with strychnine—five parts of strychnine to one of atropine. Oozing of blood was much more marked after these alkaloids; that was often complained of by surgeons, particularly where a large surface was exposed, as in breast operations, and he had been asked not to administer it for such cases. And, because of the depressing effect of morphia on the respiration, it was not advisable to use it for operations in which deep chloroform anaesthesia was aimed at. Before ether, he could safely employ a fairly large dose of alkaloids, but usually $\frac{1}{6}$ gr. morphia and $\frac{1}{200}$ gr. scopolamine caused sufficient drowsiness in the patient; more than this might produce untoward effects, save in exceptionally powerful or alcoholic patients.

Dr. H. B. WILSON said that, at Great Ormond Street Children's Hospital, babies and others had an injection of atropine and morphia as a routine; but babies under 1 year did not have morphia. All the Sisters definitely approved of it, but some of the patients complained of great thirst. The following table showed the amounts given:—

A baby under 1 year	$\frac{1}{300}$ gr. atropine alone.
Between 1 and 4 years of age	$\frac{1}{[illegible]}$ gr. atropine. $\frac{1}{60}$,, morphia.
,, 4 ,, 6 ,, ...	$\frac{1}{200}$,, atropine. $\frac{1}{40}$,, morphia.
,, 6 ,, 8 ,, ...	$\frac{1}{150}$,, atropine. $\frac{1}{30}$,, morphia.
,, 8 ,, 12 ,, ...	$\frac{1}{100}$,, atropine. $\frac{1}{20}$,, morphia.

PROCEEDINGS

OF THE

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VOLUME THE SIXTH

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SESSION 1912-13

BALNEOLOGICAL & CLIMATOLOGICAL SECTION

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Balneological and Climatological Section.

October 25, 1912.

Dr. PERCY LEWIS, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Sepsis and Spa Treatment.

GENTLEMEN,—I would like in the first place to thank you for the honour you have done me in electing me President of this Section, and in the second place, would assure you that nothing will be left undone by me to prove myself worthy not only of the honour but also of my esteemed predecessors.

The subject-matter of my address to-night is the bacterial element of those diseases which usually come under treatment at spas and climatic resorts. For a long time past I have been struck with the frequency of cases that have not only failed to find a cure at the various spas, but have actually been made considerably worse than when they went there. Now since these cases have for the most part been under the care of very able physicians, it has occurred to me that something must be wanting in our ideas of the cause of these diseases; consequently this deficiency of ideas as to their causation finds expression in their treatment.

Although all recent writers have stated that there is a bacterial element in these complaints, yet I am under the impression that they do not attach sufficient importance to the matter. I have, therefore, recently acquired the habit, when studying these "failure cases," of considering how far their treatment has failed to be consistent with the principles of bacterio-therapy. Now, since having found that a rigid application of these rules has often commanded an easy and rapid success, I came to the conclusion that they would form a suitable subject for consideration on this occasion.

THE RÔLE OF BACTERIA IN NORMAL AND ABNORMAL DIGESTION.

It has for a long time been known that myriads of microbes normally inhabit the digestive tract, and that they form 30 per cent. by weight of the normal fæces. Their presence there is not only consistent with perfect health, but under normal conditions they actually assist the digestive process. Their presence is useful as far as their proteolytic powers supplement the digestive juices. It is indispensable, because animals fed aseptically are found to die or live defectively. Nevertheless, the intestinal flora is a source of danger to its host, because it is constantly generating poisons against which the organism has as constantly to protect itself. Under normal conditions these micro-organisms and their toxins are neutralized or eliminated, but under abnormal conditions, either the normal microbes or others may obtain the upper hand and destroy or hopelessly cripple the individual. The body protects itself against infective agents or their products by the action of its digestive juices, all of which possess antitoxic and bactericidal powers. But its chief protection is from part of the normal intestinal flora. The *Bacillus coli* and the *Bacillus lactis aërogenes*, by acting on the lactose of the food, form lactic and succinic acids. Thus is maintained an acid medium which inhibits the action of the organisms responsible for albuminous decomposition. Dryness of fæcal matter in the lower bowel diminishes the activity of the putrefactive microbes; while acute inflammatory or catarrhal conditions of the intestines give abundant semi-liquid mucous secretions which dilute the fæces and provide a fertile material for microbic growth. Hence, the fetid odour of these stools, the liquidity of which strongly favours absorption of putrid matter, so that auto-intoxication easily follows. The liver has great toxilytic powers, converting the poisons it receives from the digestive organs into bodies which are much less poisonous; in fact, it reduces by three-fourths the toxicity of the materials to be got rid of. But such toxins and living bacteria as get into the blood are also subject to further attacks from the secretions of the various ductless glands. These pour into the circulation oxidizing ferments which have a strong antitoxic action. Finally, such toxins as remain are eliminated by the kidneys, assisted to a small extent by the skin, and sometimes by the lungs.

Now, as the products of normal digestion are to some extent poisonous, it will readily be understood that the advent of putrefactive changes must increase the quantity of dangerous substances formed.

The nitrogenous bodies furnish the most toxic substances—particularly the ptomaines—and these bodies are the most potent factors in causing auto-intoxication. There is always a marked increase in the proportion of intestinal toxins whenever the digestion of albumins in the upper part of the digestive canal is incomplete, whether this be due to too much protein food, or to insufficiency of the digestive glands, or to exuberance of the intestinal flora. Whenever this increase of poisons takes place the antitoxic and eliminating organs work harder, and for a time maintain the equilibrium. And when one of the eliminating organs fails the others do more work, so that the signs of auto-intoxication remain inconsiderable.

Now the main channel of elimination of the excess will vary with each individual and each type of toxin—in some the skin, in others the kidneys, in others the intestine is most active. But it must be borne in mind that overwork will always mean wear and tear, and that sooner or later lesions will occur. Thus the liver will enlarge, the kidneys will become inflamed, and then symptoms of auto-intoxication will result. The enterotoxins, now only slightly modified, will be increased in the blood, and will set up irritation in all the organs, heart, blood-vessels, vascular glands, and nervous system. And the functions of these organs, in consequence of irritation, will break down, as will be evidenced by various morbid symptoms. With different individuals different organs will suffer. In the end the gradual accumulation of these poisons will, at variable intervals of time, bring about a crisis of elimination, as shown by feverish attacks, cutaneous outbreaks, or an intestinal discharge. All these morbid states leave the sufferer weakened, but improved in health for a variable length of time, until a re-accumulation of these toxic products determines the same symptoms. Even in a person with normal digestion auto-intoxication may occur from one or more of the defensive forces being weakened or rendered incapable of repelling attack. So auto-intoxication may result from hypo-destruction of enterotoxins or from hyper-production, or from both combined. Either of these processes may result from any cause which lessens the strength of the organism as a whole, and therefore diminishes its power of defence, such as influenza, pregnancy, anaemia, and alcohol or lead poisoning. To these we may add any cause which leads to intestinal stasis in any part of the large intestine, though this would apply in a lesser degree to the rectum.

Another cause, as we shall see presently, is the constant or intermittent advent into the digestive tract of muco-pus containing microbes not generally found in it or, if found normally, in only very small numbers. As this interferes with normal digestion, auto-intoxication results. A person may, however, swallow muco-pus for long periods without any apparent harm, until suddenly the local conditions are altered by indigestible food, or otherwise. Then the gastro-intestinal membrane may absorb it freely. The rôle of microbes is to break up dead matter into simpler chemical compounds. When tissues are lowered in vitality—that is, are brought a stage nearer to dead matter—then it is that the microbes, if they be present, find their "morbid opportunity," and attack them more or less successfully. Thus diseases, especially of the class under consideration, result.

Special Instances showing the Part played by Bacteria in those Diseases which are generally Treated at Spas.

After what I have stated, it is more than obvious that there is abundant evidence that the large quantities of bacteria which normally inhabit the digestive tract manufacture toxins with which in health the organism is able to deal. This point is an important one, for in recent years an increasing number of diseases have been discovered to be due in a very large measure to organisms possessing a low grade of tissue infectivity, which yet produce toxins which by gradual absorption cause changes in remote parts, the tissues containing the local nidus remaining unaffected. That this is the case is proved by the fact—and there are other evidences—that a suitable vaccine is able to cure or arrest a disease, though, of course, the failure of the vaccine is not a proof of the contrary condition. We cannot to-day consider this fact as regards all diseases, but we may inquire how far it applies to those complaints in which the treatment by baths, waters and climates is generally used—e.g., gout, rheumatism, arthritis, anæmia, dyspepsia, corpulency, &c.

Now, it is about eight years ago since Dr. Chalmers Watson[1] propounded before this Society the thesis that gout is in the main a microbic action. Since then his ideas have been adopted by many writers both in Great Britain and on the Continent. The question whether gout is caused by the action of the microbes in situ, or whether it is caused by their activity in the digestive tract, thus leading to the admission of toxins into the circulation, is still a matter of dispute. Our knowledge has so far progressed that it is generally recognized that most forms of gastric disorder are due to bacterial agency. The causative agents may be demonstrated by passing into the stomach a Dawson apparatus attached to an ordinary gastric tube. This metal box being fitted with a special contrivance can be opened in the stomach and closed again when filled. In this way the contents of the stomach can be examined without the contents of the mouth or œsophagus contaminating them. Thus various gas-forming organisms have been found in large numbers which could not be discovered in the passages leading to the stomach. And there are various other ways by which foreign bacteria can be ascertained to be present in the alimentary canal. Any causes tending to weaken or depress the resistance of the body, such as worry, an injudicious meal, &c., may give these micro-organisms an advantage and enable them to multiply abnormally.

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[1] *Journ. Baln. and Climat.*, 1904, viii, pp. 89-112.

Now, if the evidence of the bacillary cause in some cases seems inconclusive, what shall we say of rheumatoid arthritis? Here the evidence is strong and cumulative. For a long time past various observers have not only isolated a micro-organism which differs in different cases, but they have by means of a vaccine been able in case after case to arrest the complaint which had defied other energetic methods of treatment. During the last three years a series of cases of arthritis occurring in my practice have been arrested and the joints restored to usefulness by the use of vaccine made from the pus of the patients who were suffering from pyorrhœa. But it must not be assumed, as Mr. Goadby pointed out in his Hunterian Lecture last year, that because "pyorrhœa" is not patent mouth infection is excluded as the cause of the arthritis. Recently, at one of the London hospitals, a case of rheumatoid arthritis of the hip was operated on by a surgeon. He removed the projecting osteophytes in the hope of restoring the usefulness of the joint. His hope was not realized. Later the case came into the hands of a bacteriologist. There was, then, no pyorrhœa present, and so a dead tooth having been drawn, a vaccine was made from the ends of the fangs. After treatment with the vaccine this man, who had previously been an invalid, was restored to health to such a degree that he was soon hard at work, and is still working. By adopting the same procedure in my own practice, Mr. Goadby produced a very great improvement from the administration of the vaccine in a case of spondylitis deformans.

As bearing on the efficiency of vaccines made from extracted teeth or the alveolar process of the jaws, it is pertinent to relate the personal experiences of the bacteriologist before mentioned. This gentleman had suffered from three attacks of appendicitis. He therefore decided to have his appendix operated on. However, before submitting to the operation he determined on having the stump of a tooth, the remains of a previous effort at extraction, removed. This was done and the stump received into a sterile tube, and a certain Gram-positive bacillus was successfully isolated. It is interesting to relate that the same bacillus was found in the appendix, when afterwards examined. He recovered from the operation, but was subsequently attacked by colitis. This rapidly yielded to a vaccine made from the micro-organisms which had been twice previously detected. Some months later he suffered from an acute attack of nasopharyngeal catarrh, when, *mirabile dictu*, the discharges incident to the complaint, on examination, again showed the same micro-organism as had been found in the appendix and around the fangs of the tooth. A long course of the same vaccine seems at last to have freed him from this parasite. But what a demonstration of the potent ill-effects which may result from the retention of dead teeth!

In cases of arthritis the infection may come from many other sources than the mouth. For example, during the last year a number of cases have been recorded in which arthritis has been cured by vaccines made from the chronic uterine discharges of the patient. These discharges were often so inconsiderable in amount as not to have previously demanded special treatment. Sir Almroth Wright has told me of a case where secondary infection occurred in a patient who had for twenty years been the subject of chronic gonococcal rheumatism. The patient, suffering from a stricture, was seized with acute joint symptoms each time that a catheter was passed for its relief. There was a slightly purulent discharge from the urethra, which, on examination, was found to contain a coliform bacillus. After vaccine treatment for this micro-organism no joint symptoms followed the passage of the catheter.

I would mention the case of another patient, the subject of chronic general arthritis, who was sent to an English spa. Energetic general massage was applied as part of the treatment. This measure was followed by such acute inflammation in all the affected joints that continuation of the treatment was rendered impossible. Some months later the patient died of intestinal obstruction from a large malignant growth of the bowel. Microscopic examination of the outer part of the growth situated at a remote distance from the lumen of the intestine showed the growth to be teeming with streptococci. These micro-organisms, without doubt, were the cause of the arthritic symptoms. The effect of the massage had evidently been to generalize the infection.

Previously the great drawback to acceptance of the view of the bacillary origin of arthritis has been lack of demonstration that micro-organisms exist in the joint fluids, especially in the cases of arthritis of the rheumatoid form. Since, however, they can be shown occasionally in the peri-articular structures, the difficulties in the acceptance of of this view are removed. It is not only in cases of arthritis that a bacillary origin may be presumed from the success of the treatment with vaccine, but there is also strong presumption in cases of rheumatism, whether fibrositis or neuritis. I will relate a case which is not uncommon and which is typical of others. A certain doctor suffered from attacks of fibrositis, following the least unusual exertion. One of the first steps taken was to remove all his teeth for pyorrhœa. This was done without permanently benefiting his condition. He next underwent a course of treatment at Aix-les-Bains, but there was no improvement, and his condition remained the same as before. Then a bacteriologist examined his water, which was found to contain vast numbers of streptococci. After this discovery he then underwent a course of treatment with a suitable vaccine, and this gradually resulted in a cure. The urine in a bad neurasthenic case which came under my care was found to contain large numbers of staphylococci. Treatment by a vaccine made from these micro-organisms was followed by a marked improvement in the symptoms. I have been told of two ladies, aged about 50, who had been taking a spa course for neurasthenia, with depression. Both were made worse by the waters, and both were afterwards found to be suffering from pyorrhœa. In each case recovery followed very quickly on having a treatment by vaccines, both the pyorrhœa and the depression disappearing very soon. In another of my cases, one of endocarditis, following pyorrhœa, the same organism was twice found in the blood as had months previously been found in the buccal pyorrhœal discharges. This patient was treated with vaccines, and is getting on well—albeit with greatly damaged cardiac valves. Again, ophthalmic surgeons always look for sources of mouth infection in cases of iritis, sclerotitis, and cyclitis.

Cases of neuritis have in my own practice afforded samples of their bacterial origin. The following is typical of many another: A man, aged 70, had suffered for many months from neuritis in his arms, which caused insomnia and general depression of his health to such an extent that his friends, in alarm, decided that his constitution was "breaking up." All the treatments usually given at spas were resorted to without any improvement resulting. There was present a little pyorrhœa in his mouth, but there was also some beautiful bridgework. When these bridges had been removed there was brought to light a black foul mass of decomposing food and stumps. The stumps were then extracted and a vaccine was made from the mouth discharges. A few weeks of inoculative treatment caused a return of his health and vigour with complete cure of his neuritis.

Common, however, as mouth infection is as a source of disease, post-nasal catarrh is in all probability even more so. In my own practice I have had a number of cases of anæmia of the thin type, where cure was delayed until a vaccine made from the post-nasal discharges was administered. Some of these patients had suffered from ill-health, emaciation, and anæmia for years, and only enjoyed temporary benefit whilst under treatment, relapsing as soon as the remedies were discontinued. But the improvement in their general health under vaccine treatment was most marked, and appears to be permanent. Then again in three cases of chronic urticaria, the rash which had existed for over two years quite disappeared after a few weeks with inoculations made from their post-nasal discharges.

I have given you a few cases which have come under my own notice and only in a comparatively short time—cases which have been treated with such success by means of vaccines that the conclusions to be drawn from them must be of tremendous import. If we take into consideration the very large number which have been treated in a similar manner by members who are present here to-day, there is an array of facts so striking and so imposing as to command serious thought and attention.

Method of Action of the various Spa Treatments in view of the Bacterial Origin of the Diseases Treated.

Having arrived at this stage of my subject, the next matter for consideration is the manner in which the various spa methods act when we assume the bacterial origin of the diseases there treated. To keep in view the light shed on the bacterial genesis of these diseases is to find an easy explanation of some of the failures of spa treatment. A case has already been put before you—a case of chronic arthritis—which, by massage of a focus of infection, became acute, and such massage, I venture to assert, must often be the explanation of such failures. A patient under my care this summer had suffered for three years from chronic eczema varied by daily attacks of urticaria. The sufferer had been to Harrogate. There the treatment ordered was the daily ingestion of large quantities of sulphur water. Her condition was considerably aggravated by the course. Search was made for the source of the infection, the urticaria giving the suggestion of an intestinal toxin, when a considerable amount of fæcal accumulation was found to be present, and this in spite of daily action of the bowels. The treatment then ordered was Plombières douches, combined with the administration of charcoal and naphthol after meals. In this way the source of the toxins was removed and their formation inhibited, with the result that there was a very great improvement in and amelioration of the symptoms. It is obvious that failure to appreciate the blocked condition of the *prima via* may be a fruitful cause of disappointment when hopes are based on the treatment. It is apparent that any of the spa methods might have similar effects as the sulphur had in this case —the effect being to diffuse the toxins (but not necessarily the microbes generating them) throughout the body and so cause aggravation of the symptoms. To excite reaction or cause elimination in a patient suffering from toxæmia can be of no possible use unless the source of the toxin be removed either before or at the time of the treatment. And therefore the use of aperient waters, or in fact of any waters, as eliminating agents is greatly marred if at the same time the patient is still permitted to swallow daily large quantities of muco-pus from a post-nasal catarrh or a purulent antrum. Dr. Fortescue Fox has told us in his lectures on hydrology that spa treatment by no means infrequently converts a chronic stage into an acute stage of the malady, and he holds that this "intensive action" belongs to the process of cure and is one of the principles of treatment where chronic disease is concerned. But surely this intensive action admits of the more plausible explanation I have given.

If gout be regarded as primarily a metabolic irregularity, and it is probable that such irregularity does not occur independently of bacterial activity, there is much reason for looking to bacterial intoxication as a prime factor in determining the attacks. Just as, say, a case of phthisis is not always only an infection with the tubercle bacillus, but tends to become complicated by secondary infections due to the lowered vitality produced by the main disease, so many of the symptoms which we are in the habit of attributing to gout are really in the main these same secondary infections which find their nidus in the lowered local resistance produced by the altered metabolic processes. And we may yet find that Dr. Chalmers Watson is right in holding that the primary tissue alterations are bacterial, and that the causes of the lesions probably take place in the intestinal canal or its accessory glands. Be this as it may, I am assured by an eminent bacteriologist that many acute attacks of even old-fashioned gout in the great toe have been recently and with certainty traced to secondary infections, such infections coming from the mouth, as in pyorrhœa, chronic post-nasal or bronchial catarrhs, or from suppurating piles or even from fistula in ano. The acute attacks have quite ceased with the cure of the source of the secondary infections. In the light of this knowledge we find a reason why acute attacks of this disease now and then occur when a case of chronic gout undergoes treatment at a spa. The processes there applied, such as warm bathing and massage, not only send accumulated toxins into the circulation, but are apt to spread large doses of living bacteria from the affected parts into the system.

But it is not only when treatment at a spa does harm to a case, but also when it does good, that its actions may be viewed from the bacteriological standpoint. Dr. Oliver has said that "to the alkalinity of the blood must be ascribed some of the bactericidal power which this fluid possesses, since it has been found that this property varies and is proportional to its alkalinity. It is therefore a great defence. Increased acidity weakens vital resistance and predisposes to diseases." Many of the spa waters being alkaline will undoubtedly increase the blood alkalinity, and therefore they will exert an anti-bacterial action.

Spa treatment has always aimed at elimination, though opinions as to what was to be eliminated have at times varied considerably. At present Dr. Fox has told us in his lectures already quoted that the humoral theory still holds the field. If we take the bacterial view of these diseases, we have only to substitute the word toxæmic for humoral. It will therefore readily be seen how great an amount of room there is for improvement in a treatment which has in view, and only arrives at removing, the toxins in a disease, while leaving out of sight and not attacking the bacterial causation. But this treatment, albeit blind, has been more efficient than might have been supposed. Consider for a moment cases of arthritic joints where the infecting organisms are in the peri-articular structures. In these instances the treatment by local Aix douches and massage, by alternately dilating and contracting the vessels of the joint, tend to sweep out the streptococci as well as their local products into the circulation. In addition to this the local nerve stimulation in many cases enables the joint, at any rate for a time, to get the better of the invading organisms, and to recover a part of its usefulness.

In Bier's treatment, when applied to joints, much the same thing happens. The local resistance is in each case excited to more efficient action, by the artificial increase in the blood supply, bringing extra nourishment to the cells and an extra supply of leucocytes and alexins to aid the absorption of the diseased structures and their infecting agents.

An induction of general hyper-leucocytosis is one of the results of applying either wet or dry heat to the surface of the body, and is a usual result of the hydrotherapeutic measures used at the spas. It is unnecessary to point out that this, too, is an antibacterial procedure.

As regards the administration of waters at the spas, the method adopted which is founded on experience is generally bacteriologically correct. They are given in such a way as to assist the ordinary channels of elimination. Aperient waters, for instance, increase elimination by the bowel of the results of bacterial fermentations, and incidentally at the same time remove large numbers of bacteria. When given before breakfast, combined with a little gentle exercise, they are well calculated to effect the purpose in view. When the bacterial source is wholly intestinal, the good results of taking a course of waters are seen at their best. Thereby the digestive organs get rid of the cause as well as the results of the complaint, and the system is then enabled to absorb abnormal substances which have been deposited elsewhere in the body. Less good results are exhibited if the source of the disease, such as pyorrhœa, a post-nasal catarrh, or a uterine discharge, be overlooked. In these cases the relief obtained will only be of a temporary nature and an early return to the spa will be required, when the same futile process will have to be repeated.

With indifferent or diuretic waters the object is to stimulate elimination through the kidneys, and these waters, when given on an empty stomach, attain this desirable result. In the early morning, after a night of rest and warmth in bed, the body is short of water; the digestive tract being free of food, water is easily absorbed, and takes in its passage through the kidneys large quantities of toxins from the system with it. If given after breakfast, as is often done, the result is different. In this case the system, having replenished its water supply at the meal, is not in want of water. Being taken then, water is apt to remain in the bowels a long time before it becomes absorbed

In the bowels it is mixed with the food, constituting a warm, soup-like material, very favourable to intestinal putrefaction. In that case it aids, rather than hinders, the formation of toxins.

To stimulate excretion of the skin, hot baths are often given at the spas; and in cases of renal inadequacy, or for reduction of congestions, their employment fulfils the purpose for which they are given. It is necessary, however, to remember that, as Bouchard and others have proved, the skin at its best is a very inefficient eliminator as compared with the bowels and kidneys. By acting on the skin one is apt to lessen eliminative action by the other routes, so that the patient may be less better off than if the other routes had been stimulated instead. Far be it from me to decry the use of hot baths. My desire is to plead for their use with understanding of their correct rôle. As I have already said, hot baths produce hyper-leucocytosis. Dr. Fox has told us that "the whole effect of baths of every description is founded on *the power of reaction* possessed, sometimes in a very limited and partial degree, by the disordered tissues. Their power of response to applications of heat and cold, and the production of secondary consequences in the entire organism, are," he says, "the whole key to hydrotherapy."

Reference has already been made to the other chief therapeutic measure used at spas—viz., massage—and I have shown how its effect may be disastrous where given without regard to the bacterial factor. Applied with this point in view, massage may be, and often is, a very useful adjunct to spa treatment. Exercises, light baths, radiant heat, and all other accessory means of treatment are effective or not, according as they send microbes or their toxins into the blood in doses in which they can there be counteracted or eliminated, or in doses in which they cannot be so dealt with. From what I have said it can be seen that the spa course is a very powerful therapeutic measure. Used with accurate bacteriological knowledge it has a very wide range of usefulness. Without such knowledge its results are apt to be disastrous. In the light of this information one may be permitted to enter a protest against a modern tendency to shorten the course, not generally, it must be admitted, on the part of the spa physician, but demanded by the public. The result is that the treatment has to be more vigorous, and the dangers of generalization are thereby increased. Much more may with safety be accomplished by a longer mild course of treatment than by a short energetic one.

Before closing this part of the subject, attention may be directed for a moment to the last line of defence which our system contains for its protection against bacterial poisons—viz., the various ductless glands. Not much is really known about them, but the thyroid may be said to be in fashion just now. Some physicians think so highly of its importance that, like the blessed word "Mesopotamia," it is credited with working great wonders. Some doctors hardly ever write a prescription without adding thyroid extract to its other ingredients. Undoubtedly this gland has great antitoxic powers. Dr. Leonard Williams has used it with success in many cases of enuresis. But if enuresis be, as I have long held, only a polyuria or effort of the kidneys to eliminate the *Bacillus coli* which is present in excess in the colon, the simple treatment seems to be to remove it from the bowel, rather than to destroy it in the blood after it has got there. For years one has successfully treated these cases by disinfecting and clearing the bowel by calomel and by simplifying the diet. This treatment not only removes the cause, but alters the conditions which the bacillus finds favourable for its growth. One would not for a moment belittle the uses of preparations made from the thyroid and the other ductless glands, but would insist that the intestines, the kidneys and the skin, should be regarded as the strong charwomen of the body, the thyroid, &c., being looked upon more as the dainty housemaid with the feather broom.

SUGGESTIONS FOR MODIFICATION OF SPA METHODS IN VIEW OF THE BACTERIAL ORIGIN OF THE DISEASES THERE TREATED.

In the light of these new facts which I have been reviewing, one can hardly fail to come to the conclusion that all "spa diseases" will ultimately be found to possess a microbic origin. Granting this, the question then arises as to whether any modification is necessary in our treatment of those diseases which come under our notice at the various spas. Now, it is obvious that we are at present able by means of the recent discoveries in bacterio-therapy not only to remove the results of disease, but also through the agency of vaccine therapy very often to remove the cause at the same time. In many cases a return to health has been secured by means of a vaccine alone without any course of baths or waters; just as a recurring winter bronchial catarrh can be prevented by a suitable vaccine without necessitating the annual residence in a warmer climate. This happens because the system is often able of itself to remove morbific effects, when once the cause thereof has been taken away. This being so, it is obviously incumbent on us to make in all cases a thorough search for a possible cause of sepsis, for this is often found in the most unlikely places. In every case a thorough and special examination should be made of the mouth, fauces, nose, nasopharynx, nasal sinuses, the respiratory tract, the stomach, the intestines, the urinary tract, and the uterus. Then, as Dr. T. Horder has pointed out, due consideration must be given, not only to the nature and numbers of the micro-organisms when obtained, but also to the special evidence of affinity between them and the complaint under investigation. Merely to use any organism casually obtained and cultivated from a patient's gums, throat, or urine is to court disappointment in the results. If the search prove futile, the cause is almost certain to be present in the digestive apparatus, very possibly being due to simple stasis of the fæces in the upper portions of the colon. But be that as it may, it is useless to administer a vaccine unless the source of the poison be also removed. No good result can be obtained, for instance, by giving a vaccine for a pyorrhœa if dental pockets improperly drained be allowed to remain in existence, nor by inoculation for a pyelitis when the pelvis of the kidney still contains calculi infested with bacteria. But while vaccines occasionally constitute the only treatment needed, it is very seldom in practice that one can rely on them to operate alone. To use a vaccine in every case to the exclusion of other well-tried resources is to limit greatly one's sphere of usefulness. Important as vaccine therapy is, yet, after all, it is only one weapon in our armoury for fighting these diseases.

Thus far we have been concerned with the cause of disease. It next becomes necessary to eliminate the products of these micro-organisms—to wit, their toxins. In this attempt we shall endeavour to follow Nature and try to assist the efforts she makes to get rid of them normally from the body. It has already been pointed out that elimination, the ultimate aim of all spa methods, is for the most part carried out by the intestinal and renal routes. Bearing this in mind, our efforts should be to assist these channels, and here the aid of a spa should be sought. Our choice of a spa will be, to a certain extent, determined by which of these paths we think that elimination can, in a given case, be performed to the best advantage. If the intestinal channel be the one chosen, a spa having aperient waters will naturally be the one settled on. Should the kidney route seem the better, a spa possessing indifferent or diuretic waters will receive preference.

Then the mode of administration and the quantities of the water to be imbibed become matters of great importance. Bouchard has well said, "Subtraction of water is dangerous, but its excess is none the less so. It changes the conditions of osmosis; it causes swelling up of the cells and washes out their dialyzable material; it thus disturbs their chemical constitution, and weakens and perverts their functional activity." It may be due to excessive administration of water that many patients leave a spa feeling weakened and depressed, though better in their joints. Theoretically, at any rate, they should feel their vitality increased after a large elimination of toxins. One has to remember, too, that the wetter the contents of the colon the more the bacteria thereof flourish and multiply. The conclusion, then, to be drawn from this is, that all waters should be taken when the tissues have a water-want, as in the morning before breakfast. With regard to the aperient waters and the dose, care should be taken to administer at first, at any rate, only just a sufficient quantity to bring about an emptying of the large bowel. Indifferent or diuretic waters should only be given in such quantities as are rapidly absorbed. If this rule be borne in mind, there will be no tendency for increased multiplication of micro-organisms in the digestive apparatus, and the vitality of the system in the case of aperient waters will not be lowered owing to excessive action of the bowels. In fact, whatever is done during the whole course of treatment, whether it be the manner of giving the water, or the regulation of the diet, or the use of physical methods, attention should always be directed to maintaining as strict an intestinal asepsis as possible. In other words, in our endeavour to eliminate certain microbes and their toxins we must do nothing to encourage the growth of these or others, nor must we allow more to get into the blood at a given moment than the defences of the body are capable of dealing with. If waters be so used as to dilute the fæces instead of being absorbed, of what avail is it that the water contains potash or soda, bromine or iodine, sulphur or arsenic, or even that it be radio-active?

Now, Gentlemen, we have reviewed spa diseases from the bacteriological standpoint. What have we gained? Surely we may claim to have acquired a wider and larger view of the causation and treatment of these diseases. Not only so, but we may claim to see the septic element standing out in clear relief; we see the causes of the failures of the past when we have in view the probable bacterial causation of complaints; and we are encouraged by the acquisition of this knowledge to build up high hopes for the successful treatment of these diseases in the future.

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The Medical Treatment of Gall-stone Disease.

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IN the treatment of cholelithiasis success depends first upon an accurate diagnosis; secondly, remote causes, which vary with the case, must be clearly recognized. As a preliminary, therefore, I propose to examine shortly the data relating to gall-stone formation, and to refer to the prominent symptoms and differential diagnosis of this affection.

The predisposing causes of cholelithiasis may be enumerated as follows: (1) Sedentary habits; (2) stagnation of bile in the gall-bladder; (3) over-eating, irregular meals, and alcoholism; (4) anxiety and worry; (5) indigestion and constipation; (6) tight lacing; (7) Glenard's disease; (8) cardiac disease; (9) emphysema; (10) granular kidney; (11) pregnancy. It is more common in women than in men.

Stagnation of bile undoubtedly predisposes to bacterial infection. The factors which favour the stasis of bile are little or no exercise, tight lacing, stooping over desks, excessive eating, too long intervals between meals, Glenard's disease, cardiac affections, &c. In the artificial production of gall-stones stasis of bile is a necessary concomitant, otherwise the bacilli would be carried away by the bile. It is only in a small percentage of typhoid cases that gall-stones subsequently develop, hence we must assume an additional causative factor besides infection. Anxiety and worry, by deranging the gastro-intestinal processes, lead to disturbance of the liver functions, more especially in those who eat more than is necessary for the maintenance of their nutrition. It will be mentioned later that persistent anxiety or worry is a retarding factor in the treatment of gall-stone disease. Over-eating tends to produce dyspepsia, thereby increasing the work of the liver, and leading to a diminution in the flow of bile. Pressure of fæcal matter in the hepatic flexure of the colon, or possibly the pressure of a distended colon by flatus, may produce stagnation of bile. During impaired digestion poisonous products are absorbed from the alimentary canal which have an inhibitory effect upon the liver functions, and restrict the secretion of bile. In Glenard's disease, kinking of the cystic or common duct may retard or obstruct the bile-flow. Keith [5] states that gall-stones are almost invariably present in this condition. It is probable that, apart from mechanical effects, gall-stones do not give rise to any pronounced symptoms unless there is an accompanying cholecystitis. I have seen cases where the patients, after treatment, enjoyed perfect health for several months, and shortly after had to undergo an operation. The explanation is obvious: these patients relapsed into their former bad habits, and a recrudescence of the infection resulted.

It is generally admitted now that the exciting cause of gall-stone disease is microbic infection. Regarding the *modus operandi* of gall-stone formation there are several theories. Thudichum [9] explains the formation of gall-stones by assuming that the cause of cholesterin precipitation is to be looked for in the chemical decomposition of the bile, and that that may be due to the presence of bacteria. Naunyn [8] and his pupils maintain that the two chief constituents of gall-stones, "cholesterin and bilirubin calcium," are derived from the desquamated epithelium of the inflamed mucosa of the gall-bladder.

Whilst most of the attempts to produce gall-stones experimentally have been carried out by producing artificial retention of bile and infecting the gall-bladder, Gérard [4] imitated gall-stone formation *in vitro*. Sterilized solution of bile salts was saturated with cholesterin at 37° C. and mixed with 0·5 per cent. sodium chloride and 0·2 per cent. potassium phosphate; to the mixture a culture of *Bacterium coli* was added and the temperature kept at 38° C. After two or three days there was a crystalline deposit of cholesterin, this precipitate being due to a chemical change in the solution. Kramer [6] continued the experiments in another direction. He added typhoid and coli bacilli and staphylococci to a sterilized filtered mixture of human bile and nutrient broth. The first two gave an acid, the last an alkaline reaction. With the staphylococci there was no change in the medium, but with the other two organisms it became cloudy in a few days. He came to the conclusion that the stone-formers originate from the bile, and are precipitated by those bacteria which produce acid. Bacmeister [3] has reinvestigated the experiments of Kramer, and corroborates his results. Bacmeister and Aschoff [2] share the opinion that in the majority of cases stone formation precedes the inflammatory process, but they agree with Naunyn that infection of the bile passages is a necessary factor in the production of gall-stone symptoms. In opposition to other investigators, Lichtwitz [7] endeavours to explain gall-stone formation by purely physico-chemical processes.

Before alluding to the cardinal symptoms of cholelithiasis it seems to me desirable to draw attention to its early manifestations, because it is at this stage that medical treatment is most effective. The symptoms as a rule do not suggest involvement of the gall-bladder, consequently the affection very often escapes recognition, the gall-bladder not being palpated. The symptoms vary considerably. Flatulent dyspepsia is one of the commonest ; others are fullness, distension and discomfort in the stomach, acidity, heartburn, a slightly furred tongue, occasional pain in the epigastrium, coming on from thirty minutes to an hour after meals and induced by special kinds of food, lassitude, heaviness, drowsiness, disinclination for work, and occasionally a feeling of weight, or a dull, heavy sensation in the right hypochondrium. Often symptoms of intestinal indigestion predominate. Depression of a fleeting character is a frequent symptom, and may be the only one. The true nature of the case is revealed by examination of the liver. That organ may or may not be slightly enlarged, but there is invariably tenderness on pressure over the region of the gall-bladder. This tenderness, due to cholecystitis, either precedes the formation of biliary calculi or is associated with their presence. During the past seven years I have palpated the gall-bladder of every patient I have seen, and I am quite certain that mild attacks of cholecystitis are far more common than medical men suspect. I attach some importance to the method of palpation. The plan I adopt is this: The right hand is placed immediately beneath the ribs on the right side, and the patient told to breathe quietly for a minute or two. It will be found that the hand sinks deeper in the abdomen with each expiration, so that the presence of a tumour, or a very tender gall-bladder, can as a rule be easily detected. In the majority of the mild cases tenderness of the gall-bladder cannot be detected in this way. The patient is then asked to sit up on the couch, and to bend slightly forward. The examiner sits or stands (according to the height of the couch) behind the patient, and places his right hand under the right costal arch. With the abdominal muscles completely relaxed he can then palpate the liver quite easily. The statement of neurotic patients as to the presence and distribution of tenderness has to be accepted with caution; if there is any doubt on this point the gall-bladder is first approached from the left side, and then from the right. The tenderness in the early stage is circumscribed, and does not extend below the ribs. When it is detected in a line from the umbilicus to the costal margin the peritoneal investment of the gall-bladder has become involved, and the affection has passed beyond the initial stage.

I shall refer briefly to the cardinal symptoms of developed cholelithiasis. The pathognomonic sign is biliary colic, but it is absent from the clinical picture in about a fourth of the cases. The pain is intensely severe, and is due either to the movement of a calculus in the cystic or common duct, but more often to cholecystitis with violent contraction of the gall-bladder. There is contraction of the upper part of the right rectus when moderate pressure is applied; this also occurs in cases of duodenal ulcer. In severe cases of gall-stone disease there is rigidity of the muscle. Mayo Robson has drawn attention to the existence of a tender spot an inch above the umbilicus, and in a line between it and the right costal margin. He regards this spot as of great diagnostic importance, and says it is quite as constant as McBurney's point in appendicitis. In severe cases the spot is situated where he has indicated, but more commonly it is 2 in. above the umbilicus, and 2¼ in. to the right of the middle line. It has been already mentioned that tenderness on pressure in the gall-bladder area is a characteristic sign of early cholecystitis. In advanced cases of cholelithiasis this feature is much more pronounced. Spasm of the diaphragm is a most important sign, and rarely absent. The best method of eliciting this sign is to have the patient sitting on the couch bent forwards, to hook the fingers deeply beneath the right costal arch, and to ask the patient to take a deep inspiration. The diaphragm forces down the liver, and when the sensitive gall-bladder comes into contact with the examining fingers inspiration is suddenly cut short by a stabbing pain. Boas's sign—that is, tenderness on pressure in the right subscapular region—has considerable significance, as it is present in about half the cases of gall-stone disease. Slight yellowish tinge of the conjunctivæ is fairly frequent, but deep jaundice an uncommon symptom. When present the latter is due either to blocking of the common duct or, very rarely, to inspissated bile. When the gall-bladder is distinctly palpable there is enlargement of the viscus, or distension of the organ through blocking of the cystic duct, or it may be contracted. The outline of the gall-bladder can be mapped out more readily if the patient is placed in the genu-pectoral position.

The diagnosis in most cases is comparatively easy; in a few excessively difficult. The disease with which it is most likely to be confounded is duodenal ulcer. Occasionally it is impossible to decide with any degree of confidence whether the lesion is in the duodenum or the gall-bladder. In duodenal ulcer the distinguishing features are the periodic appearance and disappearance of the symptoms. During an attack the symptoms are regular in sequence. Pain comes on from two to four hours after meals, and continues up to the next meal, and, what is very characteristic, the pain generally rouses the patient about 2 o'clock in the morning. In gall-stone dyspepsia the symptoms are inconstant; they vary from day to day. In ulcer the pain does not radiate to the right shoulders, it passes round the loin. The tenderness in ulcer is mostly in the epigastric region, or about an inch to the right of the middle line; very occasionally it is situated in the gall-bladder area. When in that situation the ulcer is supposed to be in the second portion of the duodenum. The presence of melæna confirms the diagnosis.

The other affections which various authors state may be mistaken for gall-stone disease are acute dyspepsia, pyloric stenosis, neurosis, renal colic, intestinal colic, lead colic, appendicitis, floating kidney, pancreatic colic due to a calculus in Wirsung's duct, and angina pectoris. It seems to me that confusion as to these issues must be quite exceptional. Occasionally appendicitis and cholelithiasis co-exist, and it is not uncommon for one disease to follow the other.

In treating cases of gall-stone disease the first step is to improve the digestion. It is practically useless to give drugs such as urotropine to disinfect the biliary passages until there is a marked improvement in the gastro-intestinal disturbance. At the risk of hammering the point, I would reiterate my belief that the primary and essential factor in the treatment of this affection is the rectification of the digestive functions. By correcting dietetic errors, and stimulating the digestive powers, the nutrition and vitality of the individual are enhanced; the resistance of the tissues is increased, and as a corollary the affected organ shares in the general improvement.

Formerly, the treatment of cholelithiasis consisted in the administration of certain drugs with the hope of dissolving the biliary calculi *in situ*; olive oil was the favourite remedy. How the olive oil after ingestion got into the gall-bladder was and is a mystery.

We now know that the contents of the normal gall-bladder will dissolve any gall-stone that is introduced into it under aseptic conditions. The principles of treatment, therefore, are to improve the digestion by adapting the diet to the patient's digestive powers, to foster nutritional efficiency by suitable and, if possible, pleasurable forms of exercise, to disinfect the biliary passages, and to correct any injurious habits. The rational treatment of a case in the first instance is symptomatic. If there is flatulent distension of the abdomen I frequently begin by ordering three or four rectal douches of a saline solution at a temperature of 104° F. This removes any fæcal matter that may be lodged in the colon, and stimulates the musculature of the intestines. Of course, the bowels must be moved daily. I am in the habit of prescribing sulphur water before breakfast in doses sufficient to produce a watery evacuation. If salines do not agree, a pint of hot water in the morning may be substituted, with a laxative before dinner if necessary. Many patients state that Contrexéville water before meals relieves their gastric symptoms. There is no special diet: the essential point is to prevent or alleviate indigestion. The diet should be kept within the limits of the patient's digestive powers. In most cases it is advisable to restrict the carbohydrates and fats. Indigestible articles of food and alcoholic drinks should be forbidden. Regular meals, regular hours and regular exercise are routine measures specially applicable in this affection. Some authorities advise at least four meals a day in order to produce frequent contraction of the gall-bladder. In this regard the criterion of a judicious dietary is the disappearance of dyspeptic symptoms.

The liability to intestinal indigestion suggests that in gall-stone disease there is a reflex inhibition of the pancreatic functions. Acting on this assumption, I generally give one of the pancreatic preparations combined with sodium sulpho-carbolate, sodium bicarbonate and nux vomica half an hour before meals. Judging by clinical evidence, the introduction of the pancreatic enzymes seems to have a beneficial effect.

Mental tranquillity is an important aid to digestion. Patients improve more rapidly if they are relieved from business and domestic worries. Exercise, such as walking, shooting, golfing, or riding, tells largely in the treatment. It must be regulated; fatigue is harmful. Chill should be guarded against. By lowering the resistance of the tissues both fatigue and chill may lead to fresh activity of the invading micro-organisms.

If there is hyperchlorhydria, olive oil may be given, as it diminishes the amount of hydrochloric acid in the gastric juice. Hygienic measures should be enforced, and in some cases respiratory exercises and massage may be advisable.

Tenderness over the gall-bladder is most satisfactorily treated with mustard-bran packs. These packs can be applied for twenty minutes daily for five days, and thereafter on every subsequent day until the tenderness disappears or is markedly diminished. When the digestion becomes fairly normal I give cachets of cholalin and urotropine twice a day, and advise their continuance for at least three months. So far as clinical observation goes, urotropine is beneficial. The reason for combining it with a hepatic stimulant is that an increase in the quantity of bile will probably aid in the dissolution of the calculi. If urotropine produces irritation of the bladder, benzoate of sodium may be substituted, or borovertin. Patients with gall-stone disease of medium severity should be kept under observation for a considerable time, and it should be impressed upon them that they must carry out instructions faithfully, otherwise an operation will be necessary.

Regarding biliary colic, if the pain is severe, hypodermic injections of morphia and atropine should be given. If the pain is moderately severe, morphia and belladonna, either in the form of suppositories or by the mouth, should be used, and hot fomentations or packs applied to the right hypochondrium. For milder degrees of colic, aspirin, exalgin, and antipyrin may be tried.

The question is often asked, and answered in the negative by the surgeons, Is it possible to cure a case of gall-stone disease by medical measures? I have no hesitation in saying, Yes, if the case is treated in the early stage. Further, a fair number of more advanced cases obtain complete relief from their symptoms. Surgeons are apt to forget that gall-stones may and do recur after operation. Last year a lady had her gall-bladder explored. This year she has had repeated attacks of biliary colic. After cholecystotomy patients ought to be warned to take precautionary measures against recurrence, such as visiting yearly a suitable spa. Some day a potent vaccine may be discovered which will revolutionize our present methods.

Another question is when to operate. When the tenderness extends below the costal margin it is difficult at first to predict what the outcome of the case will be. One has to wait and see the results of treatment. I will cite an illustrative case. A patient suffering from indigestion and biliary colic consulted a distinguished surgeon six years ago, who expressed the opinion that nothing short of an operation could do him any good. The case was a severe one, and I advised the patient to undergo an operation. He, however, decided to try medical treatment. In three months' time he felt quite well. I have seen him each year since, and have been unable to find any evidence of disease. These agreeable surprises are not common.

Speaking broadly, if there is no diminution in the intensity of the objective signs after three weeks' treatment I advise an immediate operation. If the tenderness diminishes one is encouraged to persevere, but if progress is not marked during the next three weeks I consider that surgical interference is indicated. May I, in conclusion, express the opinion that the frequency of surgical operations for gall-stone disease is a distinct reproach to medicine?

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DISCUSSION.

Dr. EDGECOMBE (Harrogate) said he was in agreement with almost the whole of Dr. Bain's paper. He could have wished that more had been said on the non-infective origin of gall-stones. The impression he had gathered—possibly erroneously—was that the author of the paper believed an infection of the gall-bladder or bile passages to be a necessary antecedent to the formation of gall-stones. Recent research, however, had shown indubitably that infection was not an invariable concomitant of the formation of, at least, cholesterin calculi; though infection generally supervened and favoured the deposition of lime salts, leading to the formation of the harder varieties of stone. We were indebted largely to the surgeons for a fuller knowledge of the early symptoms of gall-stone disease. Careful inquiry into the previous history of cases which had come to operation had shown that there had almost always been a prolonged period of so-called "flatulent dyspepsia." So frequently was this the case that chronic "flatulent dyspepsia" associated with definite tenderness over the gall-bladder was practically conclusive evidence of gall-stone disease. It was in this early stage that medical treatment was of great service. He believed that if taken early and suitably treated these cases got perfectly well, and that recently formed soft stones were capable of solution in situ. Experimental research and clinical experience supported this view. To say, with some of the surgeons, that every case of gall-stones should be operated upon was, in his opinion, to go too far. It was a common experience of physicians to meet with patients who presented all the early symptoms which surgeons had taught them were invariably associated with gall-stones—with or without definite attacks of colic—and to find that under suitable medical treatment they got perfectly well and remained subsequently free from symptoms. These cases were seldom or never seen by the surgeon. When recovery had taken place it was, of course, impossible to prove with certainty (1) that actual gall-stones ever were really present; (2) that if present, they had been dissolved; and (3) that though recovery might apparently have taken place, stones might not still be present in the gall-bladder, giving rise to no symptoms. No one who had seen a considerable number of these cases could doubt that the first two conditions were not infrequently fulfilled; and, further, that gall-stones might remain quiescent for a life-long period without occasioning harm. To assert that these cases should be operated upon at once lest at some time in the future they should meet with the disasters so luridly depicted by the surgeons was, in his opinion, wholly wrong. But, on the other hand, medical treatment, when unavailing, should not be continued too long. If the symptoms continued in spite of it, and the patient was in a state of chronic distress, an operation should be recommended. To operate in the absence of symptoms with the idea of avoiding a problematical disaster in the future would seem to be a needless mutilation. Among the late results of gall-stone disease cancer of the gall-bladder was said to be unduly frequent. He was not aware of any reliable figures to prove the actual incidence of this condition, and would be glad if anyone could refer him to them.

Mr. CAIRNS FORSYTH regarded the treatment of gall-stones as entirely surgical. He did not deny that cure might occasionally take place by natural means, but the risks were great. Medical treatment might afford temporary relief by flushing the bile channels and reducing catarrh, but gall-stones could not be dissolved in the living body. They remained as foreign bodies, a danger to the possessor. No one, in these days, would dream of treating cases of renal or vesical calculus by medical means, and the same should apply to cholelithiasis. He did not agree with what Dr. Bain had said with regard to recurrence of gall-stones after operation. Many of the supposed cases were due to incomplete removal of the calculi at the time of operation. Mr. Forsyth pointed out that the complications due to gall-stones had not been mentioned in the discussion, although complications were frequent and often disastrous. One saw too many cases of obstructive jaundice nowadays. They should be submitted to operation before the obstructive stage. In the early stages of the disease the operative mortality was very small (0·5 per cent. in the Mayos' extensive series). To postpone operative interference until complications arose was a procedure which was unfair to both patient and surgeon.

Dr. H. D. MCCULLOCH desired to draw Dr. Bain's attention to the series of experiments and observations carried out by Mr. C. J. Bond, of Leicester. These were described in his Presidential Address in the Section of Surgery in 1905, entitled "On Ascending Currents in Mucous Canals and Gland Ducts, and their Influence on Infection."[1] This author's observations seemed to have an important bearing upon the ætiology of gall-stones. He suggested that the temporary stagnation of the bile flow, probably associated with cholelithiasis and present in certain pathological conditions of the liver and duodenum, may really operate by bringing about an entrance of micro-organisms from the intestine along the common duct, with possibly minute particles of foreign matter, which may serve as condensation nuclei. Having regard to the purely organic as distinguished from the inert mineral nature of renal and other calculi, it seemed to Dr. McCulloch that the resolvability of biliary calculi, when first diagnosed, should be more feasible by medicinal agents than the actual "stones." The salicylates were known to exercise a liquefying effect upon the more viscous bile secretion, a condition that was the precursor of inspissation of the bile and of such calculi. Further, he considered that in more advanced cases, short of surgical procedure, a subcutaneous intracystic injection of a non-irritating cholesterin solvent might with advantage be tried, after experimentation on animals. For this purpose this viscus, even in a semi-distended state, was more accessible than the urinary bladder. A satisfactory skiagram of the gall-bladder and its solid contents might also be obtained by an intracystic injection of one of the metallic colloid solutions in place of bismuth.

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[1] *Brit. Med. Journ.*, 1905, ii, pp. 232-8.

Balneological and Climatological Section.

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Dr. PERCY G. LEWIS, President of the Section, in the Chair.

A Discussion on Fibrositis.

Opened by LLEWELLYN J. LLEWELLYN, M.B.

As you are all aware, it was originally hoped that Sir William Gowers would have been present with us to-night. Unfortunately, a regrettable state of ill-health has deprived us of that added distinction which his presence and support would have conferred on our discussion. It would, I need hardly say, have been singularly appropriate that the subject, chronic rheumatism, should have been introduced by this eminent physician; for it was he who first gave to this morbid condition, if not a local habitation, at any rate the name "fibrositis," the term now generally in use to denote this troublesome affection. Albeit the conception that chronic rheumatism, pathologically speaking, is represented by an inflammatory change in the fibrous tissues, is perhaps one of the oldest in the history of morbid anatomy. Foreshadowed in the first instance by Bichat in his celebrated work on "Anatomy," it fell to the lot of a distinguished Scottish physician, Dr. Craigie, to lay down the dictum that whatever the clinical manifestation of chronic rheumatism, it was in all instances referable to an inflammatory change in the fibrous tissues. But without in any way detracting from the pioneer work achieved by Craigie, Scudamore, Fuller, and others, all will readily admit the pre-eminent part played by Professor Stockman in proving beyond cavil the nature of the anatomical lesions typical of chronic rheumatism. Armed with this pathological fact, we are in a position to unify all the varied clinical manifestations of the affection. For whether it affect the fibrous sheath of a muscle, the fibrous investments of a joint, or the similar coverings of a nerve-trunk, each and all are but variations of one and the same morbid process. Thus apprehended, we see that the differences which obtain between the various clinical syndromes, lumbago, sciatica and so forth, are due, not to any variations in the nature of the morbid process, but are determined solely by the structure predominantly affected, whether muscle, nerve, or joint. Therefore, having regard to the protean aspects presented by the disease, it would be idle for me to attempt an adequate description of all the clinical varieties. Consequently, I shall confine my attention to one group, muscular rheumatism, or muscular fibrositis, as I prefer to call it.

Firstly, in regard to the vexed question of the nomenclature, I should suggest that the term "myalgia" be abandoned, inasmuch as it predicates the absence of any anatomical lesion, and in addition assumes that the pain in muscular fibrositis is spontaneous, whereas it is always evoked by motion. Again, the term "myositis" is misleading, for the parenchyma of the muscle is not affected, save secondarily, the morbid process being primarily located in the interstitial fibrous tissues of the muscle. The term "myofibrositis," or "muscular fibrositis," seems most appropriate, inasmuch as it indicates clearly the site of the lesion, while it commits us to no definite view as to its pathogeny.

With regard to the ætiology of muscular fibrositis, I would only lay stress upon the great frequency with which its subjects are the victims of ancestral or acquired gout. Indeed, this ætiological factor, in my experience, figures more definitely than any other in the genesis of fibrositis of all kinds. Out of a series of 1,250 cases, no less than 28 per cent. showed definite stigmata in the shape of tophi, or a classical arthritic outbreak in the great toe. In this respect they contrasted very strongly with the records of previous attacks of acute articular rheumatism, as forthcoming in the same series, their incidence only amounting to 8 per cent. This incidence of gout would have been appreciably higher had we included all that motley group of disorders comprised under the gouty diathesis. Other factors assigned a prominent ætiological rôle by many are influenza and sore throat, but in the majority of instances we have to rest on an assumption of auto-toxæmia of varied origin as the responsible *fons et origo mali*. Of exciting causes, the chief are atmospheric changes, gastro-intestinal derangements, and prolonged muscular exertion. Nor should we overlook in this connexion the marked liability to the affection that characterizes certain callings.

With regard to muscular exertion, it is a curious fact that, while inordinate exercise is prejudicial, yet, paradoxical as it may seem, rest of the affected muscle is equally detrimental. On the other hand, moderate exercise is beneficial, a peculiarity in reaction which we have largely utilized in the therapeutics of the disorder, as shown by the various systematic exercises very generally adopted for its amelioration. The predisposing effect of certain occupations is strikingly exhibited by the fact that out of a series of 1,000 hospital male examples no fewer than 600 were miners, labourers, gardeners, or coachmen. In this connexion, did time permit, many interesting facts could be adduced regarding the marked influence that the special character of the occupation has in determining the localization of the morbid process.

Passing now to discuss the clinical features, time will preclude my doing anything more than referring to the alterations which ensue in the functioning and general reactions of muscles, when attacked by acute or chronic fibrositis. But firstly a few words may be profitably said with regard to the mode of onset of the acute varieties. This, in its classical form, is dramatically sudden, and usually occurs during some slight muscular effort. This abruptness, we believe, is more apparent than real, for the muscular effort impeached is often so trivial that it can hardly be regarded as the cause of the malady, but rather as the occasion of its appearance. In other words, we believe that, despite the startling suddenness of the pain, the responsible morbid process has been slowly, though silently, maturing. This supposition is favoured by the fact that inquiry in many instances elicits the statement that for some days previously the patient had been conscious of stiffness or lack of accustomed suppleness in his affected muscles. That an absolutely healthy muscle is ever suddenly attacked by acute muscular fibrositis is, we believe, highly improbable, the pain being simply an abrupt manifestation of a morbid process which has been developing insidiously in a latent form.

Of constitutional symptoms we need only lay stress, firstly, on the fact that a rise in temperature is far more common than is supposed, even in the milder forms of lumbago, torticollis, and so forth; and secondly, that even in these localized varieties endocardial changes are by no means common.

From the subjective side, the salient and often the sole symptom of acute muscular fibrositis is pain, often of excruciating intensity. Its distressing character lies in the fact that it follows not only volitional contraction but also passive movement, not only of the affected muscles themselves but in addition of those functionally allied to them. Nor does this exhaust the sources of induced pain in the affected muscles, for they are, in addition, exquisitely sensitive to the slightest variation in tension following contraction of their antagonists.

On the objective side, a muscle attacked by acute fibrositis undergoes three main changes:—

(1) An increase in volume as betrayed by swelling.

(2) Increased local reaction as shown by undue warmth of the affected area, presumably due to increased heat production.

(3) An alteration in the tone of the muscle.

With regard to the first two conditions, swelling and heat, these have long been recognized, but we do not think that sufficient stress has been laid on the most important factor, namely, an exaggeration of the tone of the affected muscle. A few words are demanded on this, which we consider to be the fundamental change that occurs in muscles as the result of acute or chronic muscular fibrositis. All skeletal muscles, as we know, are in a state of steady slight contraction, constituting what is known as their normal tone. This tonicity may be augmented by physiological conditions, as during physical effort, but this, of course, is a physiological hypertonus, in other words, an increase of a normal state, due to an increase in the intensity of normal stimuli. Now, from time immemorial physicians have noted that rheumatic muscles as a result of the morbid process become excessively irritable, and in consequence readily pass into a state of peculiar spasmodic contraction. The degree of their contraction, though variable, always exceeds the limits of normal variation; in other words, rheumatic muscles, as a result of the morbid process, are constantly maintained in a condition of excessive contraction or hypertonus. That this is so is easily ascertainable by palpation of any superficially situated muscle the seat of an acute attack and comparing it in this respect with the corresponding unaffected muscle on the opposite side. Let us take, for example, the clavicular bundle of the trapezius, very commonly at fault in rheumatic torticollis. On palpating it transversely to the general trend of its muscular fibres the muscle will be found unduly tense. This unusual tenseness is immediately increased if any pressure be made on the affected muscle owing to induced secondary contraction. The healthy muscle on the opposite side communicates an entirely different sensation, merely a feeling of softness, and, moreover, no contraction ensues in the muscle when pressed upon, as in the hypertonic muscle on the other side.

Now, in regard to the course of the disease, the important point to grasp is, that the objective changes outlast the subjective symptoms. Thus pain, the first symptom to appear, is also the first to disappear, this being closely followed by the decline of local heat and swelling.

But the marked hypertonus of the muscle present during the acute stage, while it diminishes with the decline of the subjective symptoms does not wholly disappear for some considerable period after their cessation, as may be ascertained by palpation. Clinically, this persistent hypertonus finds expression in the fact that long after the pain has passed away the patient is conscious of a feeling of stiffness. Moreover, the abnormal swiftness and certainty with which it reacts to the exciting causes, exposure to damp, draughts, and so forth, plainly indicate that the condition has merely passed into a latent stage.

Passing now to the chronic form, on the subjective side the intense pain of the acute variety is transmuted into a more or less persistent aching and stiffness, which from time to time lights up during periods of exacerbation. Indeed, the clinical aspect presented by the chronic form is that of a series of acute attacks linked together by more or less aching and stiffness. The hypertonus, so marked a feature of the acute form, is present in the chronic, though maintained at a lower level save during relapses. Its intensity is an index to the degree of irritability of the affected muscle. We have already pictured its slow decline during the acute attack and its persistence during the latent period, and we are inclined to find in it the clue to the chronicity of the affection, forming as it does a link between successive exacerbations. But we have another factor in the chronic forms which makes for the maintenance of this tonicity, the development of nodules in the belly of muscle or more commonly in the neighbourhood of its tendinous attachments. Of these two varieties of fibrous overgrowths, those situated in the tendons, to our minds, are the most important. Tendinous structures, as we know, are richly supplied with peripheral sense-organs, the organs of Ruffini and Golgi, which are keenly sensitive to the slightest variations in tension. This has an intimate bearing on the clinical features of the disease. Infinitely more hyperæsthetic than muscle nodules, the slightest touch immediately determines contraction and augmented tension in the affected muscle. Lying, as they do, close to the bone, these nodules are therefore extremely difficult of detection, for if perchance one be palpated it is immediately masked by the contraction of the overlying fibres, which instantly assume a felt-like rigidity. Herein lies their importance, in that they, by their very presence, perpetuate the irritability of the muscle and the resultant hypertonus. For a muscle the seat of such nodules is in an allied condition to one disabled by trauma, in that it displays a marked tendency to pass into a state of continued spasm. This is easily understood when we have regard to the fact that the pathological end-products are the same in both cases—sclerosis, or an overgrowth of scarlike fibrous tissue. Once established, both muscle and tendon nodules constitute a flaw in the continuity of the muscle, a constant menace to its functional integrity.

These fibrous overgrowths are, we think, the objective expression of the rheumatic predisposition. That vague and nebulous entity, the rheumatic diathesis, as it were, crystallizes itself and becomes incarnate in the shape of these fibrous hyperplasias, which confer upon any individual unfortunate enough to develop them a potential capacity to suffer from rheumatic troubles under the most trivial provocation.

We have before stated the belief that the fundamental change in the muscle is the increase of tone that develops as the result of the morbid process. In regard to this point, Sherrington's brilliant researches have, we think, furnished us with a clue to the manner in which this hypertonic state is probably induced, and a brief digression on this point will, we think, be not out of place. All familiar with his work on the physiology of muscles will recall to mind that his researches prove that the maintenance of tone in skeletal muscles is entirely dependent on the integrity of the reflex arc. Now all the muscular actions in their primitive form may be regarded as examples of reflex action, which, as Sherrington has shown, are associated and regulated by afferent impressions, of which two main groups may be recognized—the proprioceptive and the exteroceptive. Under the former are comprised those secondary afferent impulses arising in the deep tissues—viz., muscles, joints, and ligaments—which, as we have seen, are furnished with special sensorial endings, capable of excitation by any mechanical changes of tension or pressure set up by movement. Now, in health, these afferent impulses are continually passing from the muscles to act upon the related spinal centres, but the bulk of such impressions do not pass the threshold of consciousness, and consequently pass unperceived. Should these impulses for any reason become intensified, we are instantly made aware of their existence. Thus, perchance, if any group of muscles, say the lumbar, becomes attacked by acute fibrositis, we are painfully alive to the slightest variation in their contraction or tension. Moreover, not only is their response exaggerated and painful, but unduly prolonged, persisting long after the exciting cause has ceased to act.

The excessive irritability of rheumatic muscles is, we think, capable of the following explanation: The basal pathological change is an inflammatory process in the interstitial fibrous tissues of muscles, of toxic or infective origin. In this tissue lie embedded the afferent sense-organs, the muscle-spindles, and the organs of Ruffini and Golgi. Presumably, either through actual invasion or mere contiguity, these sensorial end-organs become hypersensitive, and the impulses transmitted by them correspondingly intensified. Now Sherrington has demonstrated that the normal tone of skeletal muscles is dependent on the integrity of the proprioceptive system, of which these afferent sense-organs are essential components. These sensorial endings in rheumatic muscles being the seat of an irritative lesion, the impulses transmitted by them are exaggerated, and therefore an exaltation of muscle tone, or hypertonus, ensues. We see, therefore, that the augmented excitability of these end-organs accounts not only for the pain but also for the spasmodic contraction of rheumatic muscles. To this condition of hypertonus is to be ascribed not only the distress aroused by volitional effort, but also that following both direct and indirect passive movement of the affected muscles. The same pain accompanies the performance of any complex movement in which they are synergically concerned.

A few words, however, are necessary to explain the reason why painful sensations are aroused in the diseased muscles when their antagonists undergo contraction. As we are all aware the contraction or shortening of one set of muscles involves the relaxation or elongation of its opponents. Now all skeletal muscles being in a state of tone varying their extension, it is obvious that in the absence of any special arrangement, every contracting muscle, by stretching its antagonist, would *ipso facto* raise its tone and thus automatically engender resistance to its own effective action. In short, under such conditions every muscular contraction would be foiled of its endeavour by the resistance of its opponents, and thus the subject would be "muscle-bound." But, as Sherrington's brilliant researches have shown, the necessary inhibition of the tonus of a voluntary muscle is automatically ensured by the excitation of its antagonist. It appears that normally the elongation of a muscle which follows the shortening of its opponent stimulates mechanically the sensorial end-organs of the muscle undergoing stretching or extension, and thus abolishes its tone. Now the phenomena exhibited in the more acute form of muscular rheumatism seem to indicate that this automatic inhibition of tone is in some way interfered with. Thus all must have noted that in acute lumbago the subject, in the attempt to flex his trunk forward, is brought up sharp, held in a vice as it were, and is for the nonce "muscle-bound."

Now in the normal state, when flexion occurs through contraction of the ventral spinal muscles their opponents, the dorsal extensors, undergo simultaneous elongation, this taking place easily and painlessly owing to the automatic abolition of their tone. Presumably in acute lumbago the flaw in the mechanism would appear to be that, owing to the diseased condition of the lumbar extensor muscles, the usual inhibition of their tonus does not ensue, hence the sudden painful check. We would suggest in explanation that the muscle-spindles, situated as they are in the inflamed fibrous tissues of the dorsal spinal muscles, become exquisitely sensitive. Their response to excitation, therefore, is correspondingly excessive and painful, consequently the tone of the lumbar muscles instead of being inhibited is increased. Now we know that muscle tonus may be abolished by destructive lesions of the proprioceptive or secondary afferent nerves, as is shown by the inco-ordination of movements which follow peripheral neuritis, when it affects the sensory nerves of muscles. This being so, it seems reasonable to expect that irritated lesions involving or influencing these same structures would augment muscular tone.

While the foregoing remarks may serve to explain the disorders of movement that result from the rheumatic process, we have now to account for the development of those objective changes characteristic of the affection. In the acute forms of the malady swelling and heat production are the chief phenomena, while in the chronic these are replaced by the formation of nodules. Now, it cannot be doubted that these are the outcome primarily of circulatory disturbances, which ensue in any muscle the seat of hypertonic contraction. To appreciate this sequence we must recall to mind the fact that the circulation through a muscle varies, according as to whether it undergoes the normal rhythmic contraction and relaxation, or is in a state of tonic spasm. Thus, in the former instance, every muscular contraction, while it has little effect on the muscular arteries, drives blood out of the veins which, during the period of relaxation, rapidly refill from the dilated arteries. As a result the flow of lymph derived from the capillaries is greatly augmented. On the other hand, in a muscle the seat of tonic spasm, while blood pours in through the dilated arteries, the muscle veins throughout the same period are being steadily compressed. Thus it comes to pass that much less blood flows from a tonically contracted muscle than one undergoing rhythmic contraction.

In the light of these facts we arrive at the following conclusions: Rheumatic muscles, as we have seen, both in the acute and chronic stage, are in a state of tonic contraction, or hypertonus. In the acute variety the hypertonus is of high grade, but of comparatively brief duration. But even so the circulation through the muscle is impeded, and becoming surcharged with blood, an increase in volume or swelling of the muscle takes place. This is, however, temporary, and subsides with the lessening of the hypertonus. The local reaction, as shown by increased heat, is attributable to this same tonic contraction of the affected muscle, for increased tension augments all the processes of muscle, including chemical changes and heat production. Where the pathological state is prolonged, as in chronic forms, the hypertonus, though slighter in degree, is correspondingly persistent. This being so, the rhythmic working of the muscle pump is permanently disabled, and a state of chronic venous stasis ensues in the affected structures. When such a state of venous stagnation occurs in any organ or tissue, sooner or later proliferative changes take place in the involved structures—the so-called congestive induration—with which we are all familiar.

In conclusion, while it may be doubted whether these mechanical results of hypertonus are adequate of themselves to produce the condition of fibrositis, they may reasonably be regarded as strong contributory factors. Such a conception does not necessarily conflict with the infective or toxic theory of the origin of fibrositis. For such disorders of circulation, by depreciating the nutrition of muscle and lowering its resistance, must pave the way for the effective action of microbic or toxic agencies.

RHEUMATIC AFFECTIONS OF THE MYOCARDIUM.

By JAMES MACKENZIE, M.D.

I HAVE been struck with the occurrence of heart complaints of such obscurity that it was impossible to be certain of their nature. Among them were cases where the condition was associated with attacks of muscular rheumatism, and it was reasonable to infer that the heart muscle was affected in a manner similar to that affecting the skeletal muscles. Hitherto the evidences of the nature of heart affections have been too slight to enable one to recognize its nature. The symptoms have been for the most part subjective, such as distressing action of the heart on exertion, or attacks of palpitation coming on at uncertain intervals. Usually the heart-rate is increased, while at other times the pulse becomes abnormally slow, regular, or with intermissions.

followed by such forcible beats as to cause much discomfort and mental distress. Breathlessness on exertion was frequently present, and, more rarely, pain, amounting to such severity as to be recognized as angina pectoris. Such symptoms as these might be occasioned by a variety of conditions, but that we can with good reason attribute them to an invasion of the myocardium by the rheumatic poison, whatsoever its nature may be, seems reasonable from a study of the nature of the heart's contraction, and particularly of the character of the heart's irregular action. In a number of cases such analyses reveal that there is a delay between the auricular and ventricular systoles. In some cases the intermissions have been due to the fact that the stimulus for contraction has occasionally failed to reach the ventricle. This failure may be so frequent that the ventricle only responds to every second beat, so that the ventricular rate is only half the auricular rate, while in one case the ventricle responded occasionally to every third auricular beat, and the pulse-rate fell under thirty beats per minute. In fact, in these cases all grades of partial heart-block may be detected.

As we know the exact situation where a lesion can produce this block—namely, in the small bundle connecting the auricle and ventricle—we can thus localize the seat of at least a portion of the complaint in the muscle wall of the heart, in the immediate neighbourhood of the auriculo-ventricular bundle, before it divides into its two branches to the right and left ventricles. We can also safely infer that the effect of the invasion of the heart by the disease-process is not limited to an impairment of the functions of the auriculo-ventricular bundle, but by its presence in other parts of the heart caused such irritability that it leads to the attacks of palpitation of much severity.

It is because we have proofs of a definite focal lesion affecting the heart muscle that I make this communication, and because it supports the view that the muscular pains, in what is called rheumatism, are caused by focal lesions in the skeletal muscles.

DISCUSSION.

Dr. Stockman: I have been asked, as my share in this discussion, to direct my remarks specially to the pathology of fibrositis, and I shall endeavour as far as possible to do so. The essential lesion is a chronic inflammatory hyperplasia of white fibrous tissue in patches, and as fibrous tissue is spread throughout the whole body the lesions may also be widely spread or may affect only a single limited area. The parts commonly involved are the fibrous origins and insertions of muscles, the fasciæ, aponeuroses, and intermuscular septa, the ligamentous structures of the joints, the periosteum, the panniculus adiposus, and the nerve-sheaths. When these are widely affected the individual suffers severely from "chronic rheumatism," whereas if the muscles, say of the loins, are alone affected his sufferings are restricted to repeated attacks of lumbago. When these small growths of new tissue are excised and examined microscopically they are seen to consist of inflammatory fibrous tissue, but the details of their structure vary somewhat according to their age, and to a less extent according to their situation. When, for instance, they occur in the heel, where the fibrous tissue is relatively coarse and where the nodule or patch is subjected to much and long-continued pressure, they are dense, compact, and firm, whereas in the perimysium the structure is finer and much more open, and in ligaments or fasciæ one gets a density intermediate between these. When the lesion is quite recent, it consists of proliferating fibrous tissue, rather œdematous and showing many fibroblasts, while there is a striking absence of leucocytes. The new tissue is well supplied with blood-vessels, and a very significant feature about these is the presence of marked peri- and endo-arteritis, while the walls of the veins are also thickened. The whole appearance suggests very strongly that it is the product of reaction to an irritant conveyed by the blood-vessels. Whether this irritant is a toxin brought from a distance, or whether it is originally due to a small local colony of microbes I am unable to say definitely, but in spite of numerous examinations I have never been able to find organisms or to get a culture. When the indurations are of older standing the fibroblasts are much fewer in number and the new fibrous tissue lies in an amorphous sero-fibrinous matrix. As they become older still the individual fibres are more defined and more closely packed, and in such parts as the heel present a very dense solid structure. In all, the thickening of the walls of the blood-vessels is very obvious, and in some this has occurred to an extreme degree. When these new formations are recent they may, and often do, resolve spontaneously, but, if they become very fibrous and if there is repeated causal irritation, they tend to spread and to increase in size. Fibrous adhesions present the same structure, and in cases of sciatica I have found (on operation) the nerve-sheath thickened on its surface, and these thickenings also show the above appearances under the microscope. The cicatricial tissue which often follows a sprain, or the rupture of a muscle, or a dislocation, has also the same structure.

The indurations or nodules can be readily felt through the skin and are found to vary much in size and form. They may be defined and circumscribed like a split-pea or half an almond. Sometimes they are like a strand running along a fibrous septum or fascia, or they may be widely spread out over an aponeurosis and through the neighbouring subcutaneous tissue, giving a hard, inelastic feeling to the skin as if the part were hide-bound. When the panniculus adiposus is affected (chronic subcutaneous fibrosis) they form small, round, tender fibro-fatty masses. The indurated areas are permeated by nerve-twigs in a condition of interstitial neuritis, or they may involve larger branches, and hence they are painful when pressed upon.

As regards their ætiology, they undoubtedly appear as the sequelæ of certain infections such as acute rheumatism, influenza, sore throat, rheumatoid arthritis, general gonococcal infection, and mucous colitis. Trauma causes them locally, and probably lead poisoning may be a cause. But many cases come on very gradually and insidiously, and must be due to causes with which we are as yet unacquainted.

The morbid anatomy and histology of fibrositis is, therefore, not a very complex matter, consisting mainly in the growth of pathological fibrous tissue in patches, and implicating the blood-vessels and nerve-twigs which supply it. I would like, however, in a few words, to attempt to correlate the pathological findings with the clinical symptoms of chronic rheumatism. It is well recognized that certain irritants such as muscular exertion, damp, cold, barometric changes, cutting winds, indigestion, and various toxins very readily cause these indurations to swell up, when they give rise to aching, stiffness, and pain. The aching may last for days or weeks at a time and the stiffness is also very persistent. Sudden tension of a muscle by exerting pressure on an inflamed swollen nodule may cause instantaneous and disabling pain, as is so often seen in lumbago. These phenomena of stiffness and aching occur to a much less degree in normal fibrous tissue, most commonly after unwonted muscular exertion, but there they pass off very rapidly as a rule, and in persons who are "in training" they do not occur at all. Further, in people who are "rheumatic," slight stiffness and aching may come and go several times during a day. The warmth of bed, exposure to a fire, or a short sleep, may cause this stiffness and aching, which wear off as soon as the person moves about. All these things point, I think, to vasomotor disturbance in the indurations as the cause of the symptoms. In comparing normal fibrous tissue with the pathological fibrous tissue of chronic rheumatism, the striking difference is the presence in the latter of marked peri- and endo-arteritis and of interstitial neuritis, and in this, I believe, lies the explanation of the symptoms. The diseased vessels and nerves are probably abnormally sensitive to the irritants I have already mentioned, and under the action of these irritants exudation of lymph and serum readily occurs and is only slowly and imperfectly absorbed. This gives rise to local œdema, swelling, and tension, with pressure on the inflamed nerve-twigs, causing pain, aching, and a feeling of weariness. Owing to the changes in the vessels reabsorption of the exudation is relatively slow and imperfect, and hence the persistence of the symptoms and the stiffness. The mechanical aid which movement and massage give to absorption of the exudate would also explain the beneficial effect of these remedies.

In conclusion, I would like to say a few words about the subcutaneous rheumatic nodules which sometimes occur in acute rheumatism, and which were described in this country in 1881 by Barlow and Warner.[1] As is well known, these appear often in crops which frequently all disappear in a few days. So characteristic is this that in France, where they have been exhaustively studied, the term "ephemeral" is applied to them. Many of them, however, are not very ephemeral, as they sometimes persist for weeks or longer. Their histological characters are those of rapidly proliferating fibrous tissue with numerous vessels showing peri- and endo-arteritis and many capillaries. The great difference between them and the lesions of chronic fibrositis is the occurrence in them of large necrotic areas where the newly formed tissue is already breaking down. This allows of their rapid absorption and explains their "ephemeral" character. The longer they persist the more fibrous they become, and they may remain permanently in a fibrous condition.

Mr. KENNETH W. GOADBY: All the divisions of fibrositis have a common pathological basis—namely, the inflammation of fibrous tissue, mainly that acting as a supporting and insulating medium for muscles, nerves, and peri-articular structures of joints. Fibrositis is sometimes used to indicate fibromyitis alone, but the term should be applied in its more general sense.

According to Osler, fibromyitis follows rheumatism, is associated with painful and often swollen muscles which become swollen and flabby, the symptoms indicating an interstitial inflammation of the connective tissue surrounding the muscles, followed in severe cases by secondary degeneration in the muscle-fibres themselves. Many of such cases are associated with pain along the nerve-sheath, where the fibrous thickening of the sheath may be often felt; a condition often referred to by the patient as neuritis, although pathologically a neuro-fibrosis. Should the inflammation remain persistent, or the causative agent continue in operation, the tendon-sheath may be affected, the muscular tissue or subcutaneous tissue becomes swollen and œdematous, pitting on pressure. The œdema may persist, fibroid changes taking place, resulting in permanently thickened structures. The lymph channels and lymph spaces are no doubt involved in this exudate, and whether we adopt the views of Starling and Cohnstein, the mechanical method of lymph production, or the secretory theory of Asher, Hamburger, and Lazarus Barlow, we must admit the action of some toxic agent which modifies the normal process of lymph production and removal.

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[1] *Trans. Internat. Med. Congr.* (7th Session), Lond., 1881, iv, pp. 116-128.

In those instances where a toxic substance is known definitely to exist in the tissues, as for instance, in the vicinity of acute inflammation due to infection of the staphylococcus or streptococcus, tissue œdema occurs, not only due to the mechanical blocking of the vessels, but to the action of the toxins themselves, whilst similar and probably linking phenomena between the early stages of fibrositis and acute infective processes are the œdemas met with in serum rashes (anaphylaxis) and the blebs of urticaria.

From the inception of bacteriology the processes related to bacterial infection have always been those acute affections in which the demonstration of the causative agents is direct and easy, and it is only of recent years that attention has been directed to the action of bacteria and their poisons in more chronic forms of disease where the demonstration of the infecting agent is rarely easy, and often impossible to find at the site of the chief pathological change. Considerable evidence exists to show that in the normal organism bacteria are constantly finding their way into the tissues; thus Ruffer found that leucocytes were present on the intestinal mucosa and had engulfed bacteria. Bizzozero and Ribbert found that bacteria could be demonstrated in normal mesenteric glands. Nicholls and Ford proved that bacteria could be recovered from healthy organs. Wrosczek found that by feeding animals with non-pathogenic chromogenic organisms these might be recovered from the internal organs, although there was no lesion of the alimentary tract, and both Adami and Schnitzler attribute to this type of sub-infection those cases of cryptogenic infection occasionally met with. There is little doubt that bacteria normally gain entrance to the blood-stream, that normally these organisms become destroyed, but that they may, and undoubtedly do, settle down in various internal organs, as a rule without causing any pathological lesion. Without labouring the point further we may consider that sub-infection is a regular, almost normal, process of the human organism. On the other hand, the physiological explanation of certain transudates as due to alterations of osmotic pressure or salt concentration is no doubt accountable for certain forms of tissue œdema. But even in such instances the altered metabolic change predisposes to localized sub-infection. Whilst recognizing such a fact, I would submit for your consideration that a considerable number of cases of fibrositis and its allied conditions may be traced to the action of bacteria or their toxins, and that although the evidence I submit does not contain the one clinching fact—namely, the cultivation of organisms from the actual site—yet the inferential and circumstantial evidence is too strong to neglect. I have for a long time been struck by the fact that when treating post-nasal catarrhs, inflammation of the mouth of various types, and other allied diseases, by means of vaccines, that certain associated symptoms classed under the general term "rheumatism," but without articular involvement, have reacted in an extraordinary manner, with the improvement of the suppurative lesion.

I have collected a certain number of these cases in tabular form, classing them according to the severity of the symptoms exhibited. In all the cases dealt with, a complete bacteriological examination was undertaken of the actual focus of infection, and of the urine, and in a few instances of the fæces as well. The bacteria from such sources were carefully isolated, and submitted to various tests to determine, if possible, the infecting organism in the given instance. Use was made of :—

(1) The presence of organisms engloboed by the phagocytes on the examination of direct smears.

(2) The phagocytic and opsonic behaviour of the patients' serum towards the isolated organisms.

(3) The alteration in the clinical symptoms upon the use of vaccines prepared from the killed cultures of such organisms.

(4) In a certain number of instances examination of the behaviour of such organisms upon inoculation into animals.

All the organisms isolated were not so tested, but typical examples of the streptococci, streptobacillus, and the *Bacillus necrodentalis* were used. Those organisms which showed a low opsonic index, or which gave agglutination of the patient's serum and a low opsonic or phagocytic index, and were found in considerable numbers in the pus or other material examined, were the first ones used. On testing these organisms on the tissues of animals (rabbits), they were found to exhibit a very low degree of virulence, practically no suppuration being produced locally when up to two whole agar cultures (twenty-four hours) were used for inoculation. The inoculations were made in the vicinity of joints or tendons, or in the subcutaneous tissue in the vicinity of muscles. Although no suppuration followed these injections, swelling and fibroid changes occurred in the surrounding tissues, in many cases leaving permanent fibroid thickening. When the inoculations were in the vicinity of the joints, very pronounced fibroid peri-articular increase occurred; in several instances swelling of a tendon in other situations than that inoculated occurred.

An attempt was made to recover the organisms from the sites of inoculation from the animals. If the swelling was very persistent, the organism could be recovered at the end of three weeks; but, as a rule, in ten days' time aspirated fluid or direct curettings of the infected area proved sterile on culture. Notwithstanding this, progressive changes were seen to have taken place. These negative results confirm the results I have obtained in making cultures from œdematous and fibro-œdematous swellings in man; in ten cases the cultures all proved sterile.

It must be remembered that in attempting cultivations from a swollen tendon or areolar tissue in man, the chances of meeting with the organism are remote; very few organisms are likely to be present, and the small area infected by the culture gives very little chance of finding the actual organisms. But that organisms do exist in these situations is strongly suggested by the two following cases:—

Case I.—A peri-articular arthritis, with considerable swelling of hands and feet, some alteration in both elbow-joints, and with constant pain referred to various situations, tenderness, but not pain, elicited by digital examination of many nerve-trunks; slightly oscillating temperature running up to 99·5° F. Examination of the patient failed to discover any obvious source of infection, the urine proved sterile, and examination of the mouth and the post-nasal spaces was negative, so far as pathological lesions were concerned. There was, however, some evidence in the mouth that old inflammation had occurred, and cultivations made from both nose and throat and mouth showed the presence of a certain number of streptococci, of two varieties: (*a*) The common mouth streptococcus; (*b*) an organism nearly related to the *Streptococcus fæcalis*. Vaccines were prepared from the streptococcus, but no alteration of the temperature occurred even with a dose of 300 million, and no change in the clinical symptoms was observable. Two other organisms isolated from the mouth were also used, and with a like effect. Massage was therefore given to the swollen areas, hands, and feet, and Bier's bandage applied, and the following morning a catheter specimen of urine taken which showed the presence of organisms. At the end of four days the urine was sterile again. The same process was then repeated, and the same organism again obtained from the urine. A vaccine was therefore prepared of the bacillus thus obtained, and the first injection gave a definite temperature reaction with general exacerbation of pain over the body. The vaccine was continued, and the temperature gradually assumed normal limits; the swelling and œdema of the hands very largely disappeared. The organism obtained from the urine was a diplobacillus resembling in many characters the *Bacillus necrodentalis*.

Case II.—In this case a similar line of treatment was adopted, and in this instance a streptococcus was obtained, and similar results followed the inoculation of this organism.

In the following tables I have epitomized for the sake of brevity a series of cases in which a complete bacteriological examination has been made, and in which the symptoms of fibrositis of muscle, nerve, and tendon have seemed to be definitely related to the organisms specified, in that the administration of the corresponding antigen has resulted first in an exacerbation of the symptoms, temperature reaction, ultimately followed by amelioration, and in many cases by complete disappearance of the symptoms.

For the sake of brevity, and to avoid dealing with the clinical facts of the cases, I have divided the cases in which the organisms were obtained into three classes:—

(*A*) Those with simple rheumatism or rheumatic symptoms and without involvement of articular structures.

(*B*) Those exhibiting similar symptoms to Class A, but with the addition of inflammation and fibrous metamorphic changes in nerves and peri-articular structures.

(*C*) Those exhibiting symptoms of the previous two classes, but with definite changes in the joints, as evidenced by digital examinations and skiagraphs.

The urine for examination was obtained by means of a sterile catheter in every instance. Only five examinations of fæces were made, but in these cases only was there any clinical indication for such bacteriological investigation. In eleven of the urine examinations only was a positive result obtained; the remaining twelve were negative.

In the tabulation of the organisms found, the ordinary short chain streptococcus of the mouth, the sarcinæ, and the class of anaerobe found in the mouth and nose are not tabulated. A streptococcus may be obtained in all normal as well as abnormal mouths. On the other hand, three main types of streptococcus have been observed:—

(1) A streptococcus growing out into very long chains, many hundreds of cocci going to the chain. This organism is often found in the urine, and was observed in the urine in three of the cases, and in all three it was present in the mouth or nose.

(2) A streptococcus with much larger cocci than the previous one, tending to rapid involution, with transverse divisions in the individual cocci. It frequently occurs in agar cultures and also even on fluid media in a conglomerated mass, and is the organism usually known as *Streptococcus conglomeratus*.

(3) A streptococcus closely resembling the staphylococci in its microscopical characters: it has a fine growth, not unlike a feebly growing staphylococcus. On fluid media, however, it grows out into chains, but on agar is in staphylococcal form. I call this organism *Streptococcus medius*.

An attempt was made to differentiate these streptococci by means of the carbohydrate tests suggested by Gordon, Andrewes and Horder, &c., but the fermentations were so irregular that it was difficult to class the organisms by this means. As a general rule, the long chain streptococcus and the *Streptococcus conglomeratus* do not ferment manitol, whereas the *Streptococcus medius* does. This organism may be regarded as the *Streptococcus fæcalis*. The *Micrococcus catarrhalis* was present in a certain number of cases, but in only one case, as will be seen in Table III, was any increase in the rheumatic symptoms produced by its use.

In obtaining the bacteria from the mouth two methods have been adopted:—

(*a*) Using the expressed pus from the gum margin in cases of alveolar suppuration, after first cleansing the parts with dilute lysol, and gently expressing a bead of pus on to a small platinum loop.

(*b*) The removal of one or other of the affected teeth, syringing out the socket with sterilized distilled water, and removing a portion of the alveolar bone with a sharp spoon. (Even when this method is adopted, the *Micrococcus catarrhalis* may be found, probably having penetrated into the tissues in conjunction with the other bacteria, as films made from the curettings frequently show Gram-negative cocci, a fact which I pointed out in my Erasmus Wilson Lecture at the Royal College of Surgeons some time since.)

(*c*) In obtaining cultures from the nasal passages a sterile spring speculum was used for the anterior nares. For the posterior nares a bent platinum loop was used, passed rapidly behind the soft palate and withdrawn without touching the sides of the cavity: before making the agar smear the wire is bent straight with a pair of sterile forceps.

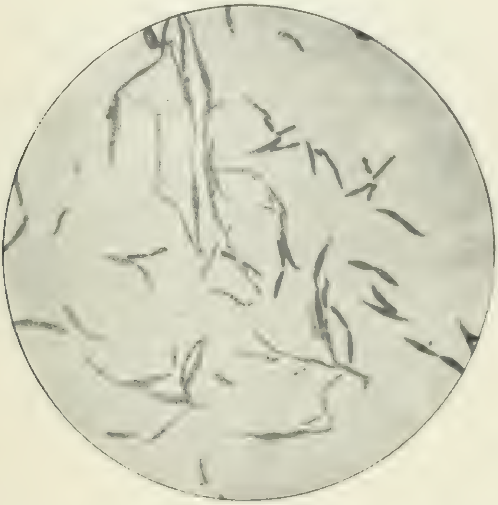

Fig. 1.

Thread-forming bacillus with Babes-Ernst granules isolated from mouth and urine. (× 1,000.)

Two other organisms were found with considerable frequency. The *Streptobacillus malæ*, an organism in many ways resembling the streptococci, but distinguishable from them by the curious oat-shaped form in which its usual development takes place, and the *Diplobacillus necrodentalis*, which was also found in the depths of the alveolar process as well as in the pus from the posterior nares. The *Staphylococcus viscosus*, an organism somewhat resembling the *Micrococcus catarrhalis*, but forming a firm, hard gelatinous colony, the whole of which comes away with a platinum needle, is to be regarded as an ordinary saprophyte of the mouth.

In examining the post-nasal spaces the long chain streptococcus is frequently found, together with various mouth organisms, mainly the streptobacillus, with the *Bacillus necrodentalis*. The *Micrococcus catarrhalis* may, of course, be found at times, but this organism did not appear in the three cases in which the post-nasal space was the only site of inflammation. In one case the *Bacillus coli communis* was present, and in one the *Bacillus septus*.

In Class B, of ten cases of urinary examination, six showed the presence of bacteria; and in Class C, of seven cases examined, four showed the presence of bacteria. It will be seen on reference to Classes B and C that both streptococci and the *Streptobacillus mala* were found in the urine, and could be distinguished one from the other. The *Streptococcus conglomeratus* was not found, the two organisms found being the long chain streptococcus and the *Streptococcus medius*. The streptobacillus was found in Classes B and C, but did not occur in Class A. No other mouth organisms were isolated from the urine, though I have met with both the *Micrococcus catarrhalis*, Friedländer's bacillus, as well as *Micrococcus gingivæ* and *Saccharomyces buccalis* in other cases where the only site of localized affection has been either the post-nares or the mouth.

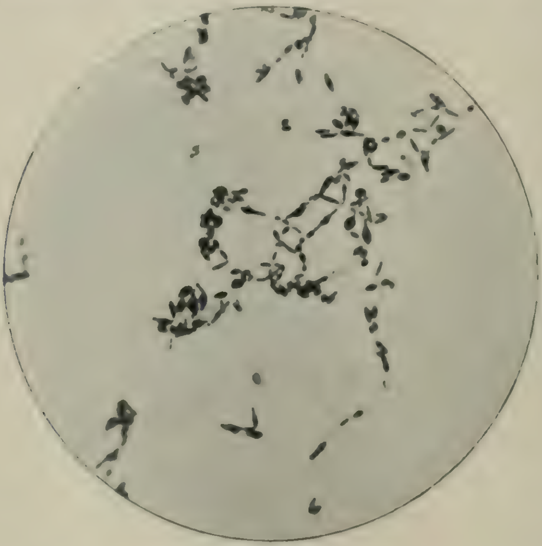

FIG. 2.

Streptobacillus malæ, twenty four hours' agar cultivation, stained Gram. (× 1,000.)

The long chain streptococcus is exceedingly difficult to eliminate from the urine. At the present time I have under observation three cases in which this organism persists in the urine, notwithstanding a nine months' course of vaccine and treatment at Harrogate and Marienbad, and the clinical symptoms, though less, are still in evidence. Other cases show recovery coincidentally with the disappearance of the organism from the urine: one examination is not sufficient to base an opinion upon, as is also the case with chronic infection elsewhere.

The relationship of mouth and nose infection, and the excretion of the organism by the urine, and the presence in most of the cases of gastro-intestinal disturbances of fermentative type, generally unassociated with pain, emphasize the close relationship, well known to exist, between "rheumatism," using the word in its widest sense, and intestinal inadequacy; not only this, but many forms of "gout" are brought under suspicion, such as gastro-intestinal crises, &c.

In concluding this contribution to "fibrositis" I would submit to your consideration:—

(1) That the bacteriological evidence I have adduced points to the probable exciting cause in a large proportion of cases.

(2) That a proper bacteriological investigation of nose, mouth, urine and fæces greatly assists in the proper understanding of the disease complex.

(3) That the presence of chronic infection of the upper part of the alimentary canal, even though it be apparently small in amount, is a potential source of danger.

TABLE I.

Class A.—Myofibrositis—muscular rheumatism, sciatica, lumbago, joints unaffected 3 cases. Bacteriological examination; Gums, 14; alveolar process, 6; post-nasal space, 3; rine, 20; fæces, 3.

Organism	Post-nasal space (3)	Alveolar process (6)	Gums (14)	Urine (20)	Fæces (3)
Streptococcus (*a*) *conglomeratus* (*b*) *longus* (*c*) *medius* ...	2	5	8	9	3
Bacillus malæ (streptobacillus)	2	5	10	8	0
Diplobacillus necrodentalis	2	3	7	0	0
Micrococcus catarrhalis	0	4	5	0	0
Staphylococcus viscosus	0	2	4	0	0
,, *aureus* . .	0	0	1	0	0
,, *albus*	0	1	3	4	2
Micrococcus gingivæ . . .	0	1	4	0	0
Saccharomyces buccalis ...	0	0	4	0	0
Diplococcus pneumoniæ	0	1	2	0	0
Bacillus coli communis ...	1	0	0	3	3
,, *septus*	1	0	0	0	0

TABLE II.

Class B. Myofibrositis, tenofibrositis, neurofibrositis—peri-articular structures of joints involved in many instances; no cartilaginous changes in joints; no osteophytes.

Organism	Gums (10)	Urine (10)	Fæces (2)
Streptococcus (*a*) ***conglomeratus*** (*b*) *longus* (*c*) *medius*	9	5	2
Bacillus malæ **(streptobacillus)**	8	4	0
Diplobacillus necrodentalis	6	0	0
Micrococcus catarrhalis	5	0	0
Staphylococcus viscosus	6	0	0
,, ***aureus***	1	0	0
,, *albus*	2	3	0
Micrococcus gingivæ	3	0	0
Saccharomyces buccalis	3	0	0
Diplococcus pneumoniæ	1	0	0
Bacillus coli communis	2	1	0
Thread-forming bacillus, **B.E. granules** ...	2	2	0

TABLE III.

Class C. Neurofibrosis especially—peri-articular arthritis with enlargement and alteration of joints; much pain or irregular twitchings of affected muscles—7 cases.

Organism	Alveolar process (5)	Gums (2)	Urine (7)	Fæces (2)
Streptococcus (*a*) ***conglomeratus*** (*b*) *longus* (*c*) *medius* ...	2	2	2	1
Bacillus malæ **(streptobacillus)** ...	4	2	2	1
Diplobacillus necrodentalis	3	0	0	0
Micrococcus catarrhalis	3	1	0	0
Staphylococcus viscosus	1	1	0	0
,, ***aureus***	0	—	0	0
,, ***albus***	2	0	2	0
Micrococcus gingivæ .. .	1	0	0	0
Saccharomyces buccalis	0	0	0	0
Bacillus coli communis	0	1	0	2
Diplococcus pneumoniæ	1	1	0	0

TABLE IV.—ORGANISMS FOUND BY TRIAL TO GIVE REACTION WITH CASES.

Species	CLASS A			CLASS B			CLASS C		
	Mouth and nose	Urine	Fæces	Mouth and nose	Urine	Fæces	Mouth and nose	Urine	Fæces
Streptococcus longus ..	0	3	0	1	0	0	1	0	0
,, *conglomeratus*	0	0	0	1	0	0	1	0	0
,, *medius* (S.)	0	1	1	0	2	0	0	1	0
Streptobacillus malæ ..	4	3	0	2	1	0	2	1	0
Diplobacillus necrodentalis ...	7	0	0	0	0	0	1	0	0
Micrococcus catarrhalis	1	—	—	—		—	—	—	—
Bacillus septus ..	1	—	—	—	—	—	—	—	—
,, *coli* .	1	—	—	—		—	—	—	—
Diplococcus pneumoniæ ..	1	—	—	—	—	—	—	—	—

Balneological and Climatological Section.

March 6, 1913.

Dr. Percy G. Lewis, President of the Section, in the Chair.

A Discussion on Fibrositis.[1]

DAILY HABITS IN CIVILIZED LIFE AS FACTORS IN THE CAUSATION OF FIBROSITIS: WITH SUGGESTIONS FOR ITS PREVENTION AND CURE.

By R. Ackerley, M.B.

We are at present only groping about for the cause or causes, admittedly obscure, of the overgrowth and inflammation of fibrous tissue. The belief that it is due to the direct or indirect action of bacteria, especially those of the alimentary canal, is prevalent. A clinical experience of some considerable extent leads me to the belief that any toxin, whether chemical, bacterial, or metabolic, *if retained in the body*, is sufficient to cause (1) fibrosis, and (2) fibrositis: the first being a gradual and painless process, the latter being due to any strain or injury to fibrotic tissue or to the irritation of that tissue by any extra or sudden formation of toxin, or to a similar extra or sudden interference with the excretion of toxin. The fibrotic changes which Metchnikoff indicates as those of old age would on this hypothesis be caused not simply by toxins absorbed from the bowel, but by any toxins or waste matter not perfectly eliminated. Treatment aiming (1) to prevent day by day formation of unnecessary toxin of any kind, and (2) to ensure complete daily excretion of any toxin formed, has such excellent permanent results even in severe conditions of fibrositis as to be, as far as it can be, confirmatory of this theory.

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[1] Adjourned from January 30.

In works of medicine where ætiology is discussed it is common to find that, except in acute infective diseases, secondary and not primary factors are enumerated as causes—e.g., in the case of fibrositis, exposure to cold, strain, fatigue, damp, indulgence in a hearty meal, can, at most, be only precipitating causes. Sore throat is, too, only a secondary cause, itself being a sign of some sepsis. In chronic disease, if we exclude bacterial infection and trauma, we have left as common primary factors heredity, environment, and daily habits, and of these the first has received too much attention, and the last hardly any at all.

There is almost a consensus of opinion that inflammation of healthy fibrous tissue does not occur, except possibly in acute infective conditions. I should be reluctant to accept the suggestion that the pains which almost invariably accompany some of the acute infections are due to transitory fibrositis, though I think that the pains are due to irritation by toxins of nerve structures running through and terminating in fibrous tissue, and that toxins, in circulation, are the cause of much so-called rheumatic pain. But apart from this, under ordinary circumstances the immediately antecedent conditions which induce attacks of fibrositis so frequently in the middle-aged or unhealthy do not have the same result on the healthy tissues of the adolescent or healthy and vigorous adult. As a rule, fibrous tissue to be susceptible to inflammation must either be anatomically normal fibrous tissue in a morbid condition, or be itself a new growth due to some morbid influence. And Professor Stockman has shown us that new fibrous tissue is frequently, even if not always, found microscopically in specimens taken from people suffering from fibrositis. How does this fibrous tissue come into existence? First of all I am going to suggest that any irritant, physical, mechanical, or chemical, applied to the body, or to any part of it, for a considerable length of time, provided it is not violent enough to cause necrosis, leads to an overgrowth of fibrous tissue. Chemical irritants absorbed from the alimentary canal will cause it, as we know from the pathological results of chronic arsenical poisoning or chronic lead poisoning. But practically in the cases under our consideration the poisons must generally be endogenous. These may be either of microbial origin, or may be simply one or more of what I might call the normal waste products of the body which have not been properly eliminated. In either case it would be quite correct to designate them toxins.

In earliest infancy—and I should like to take the breast-fed suckling as the type of most perfect health, agreeing fully with Dr. Leonard

Hill's statement that "the body of a newborn babe is a glorious and perfect machine, the heritage of millions of years of evolution"—it is probable that every form of waste product is thoroughly eliminated day by day. And later on, in the child's body, with its greater sensitiveness to the presence of noxious influences, it is probable that accumulations of toxins to any extent do not occur, as they are thrown off in what might be described as some form of metabolic storm. And in adolescence and early adult life I am sure, from clinical experience, that the so-called bilious attack is one way in which the body, from time to time, gets rid of toxic material which has been accumulating, but which has, probably, not been deposited in special tissues. It is interesting to note how very frequently one finds that those who in middle age suffer from fibrositis or multiple arthritis have been sufferers from some kind of migraine in earlier life. But after the suckling age, besides the toxins which come from the normal cell metabolism of perfectly healthy tissue, there is a growing tendency to form additional toxin. Among the influences tending to cause this we may take (1) improper food, (2) excess of food, (3) faulty food habits, such as imperfect mastication and imperfect insalivation, and (4) the addition of noxious mineral substances, especially common salt. We have also unhealthy surroundings, including defective aeration and defective sunlight, and, lastly, infective foci, especially those of the nose and mouth. The acute specific infections of childhood no doubt contribute their share, so that, even in adolescence and early adult life, there are many factors leading to the formation of toxic matter over and above the inevitable physiological products. This necessarily throws a very large amount of work on the excretory organs, for however toxins may be got rid of, they must be excreted from the body in some way if perfect healthiness is to be maintained. And if there is any interference with the excretory organs, or if the materials with which those excretory organs work are supplied insufficiently, more or less retention of toxins must take place; and as the sensitiveness of the body gradually becomes less as years go on there will be less tendency for these toxins to be thrown off periodically by metabolic storms.

Before going further I should like to submit to you some data obtained from the last 200 well-to-do patients seen by me during 1912, so that we may see to what extent in these people there may have been either excessive formation of toxin, or defective elimination, or both. Out of the 200 there were 115 cases of fibrositis. Of the 115, 26 were men and 89 women. Their ages, taken in decades, were: 5 between

20 and 30; 9 between 30 and 40; 33 between 40 and 50; 36 between 50 and 60; 25 between 60 and 70, and 7 over 70. All of these had had two or more attacks of so-called rheumatism or neuritis, either widely distributed, or more especially described as sciatica, lumbago, neuritis of neck, arm and shoulder. Of these, 44 had an arthritis or peri-arthritis of one or more joints. The history of rheumatism varied from one year to thirty-two years.

Now to go to the factors which caused increase of toxins. Beginning with the digestive system, there was, and had been, evidence of digestive derangement in nearly all cases, that is to say, there was troublesome flatulence in 85 cases, constipation requiring a daily aperient in 59, and fairly frequent looseness of bowels in 12 cases. Out of the 26 men, 12 had markedly dilated stomachs. Dilatation of stomach in women, too, is fairly frequent, but as I examined all my men, and not all my women, for this condition, it is of no use giving the figures for women. As a sign of toxin formation in the alimentary canal I regard flatulence as most important, as it implies abnormal fermentation. Further evidence of putrefactive changes is afforded by the presence of indican. In 15 out of the last 60 cases where a routine examination was made for it this was found in excess. On searching for causes for these intestinal disorders, I found that in 66 cases there was definite oral sepsis at the time of my examination, and in 13 others it had existed a few months previously, and in only 9 cases was I absolutely satisfied that there was, and apparently had been, no trace whatever of any sepsis. Defective teeth, unrelieved by artificial means, were found in 24 cases. Fifty-six had one or more dentures, and of these 17 kept the plates in the mouth all night—a practice described by a former president of the Odontological Section of this Society as "altogether bad."

As regards this question of oral sepsis, in a paper I read three years ago[1] recording the condition of the mouth in 1,000 consecutive cases of chronic disease, I noted oral sepsis in 22 per cent. At that time, in my own words, "the information I have got as regards the teeth and habits of mastication is arrived at in the ordinary examination of the patient; not the minute examination made by the specialist, but, if anything, the minimum that ought to satisfy the conscience of any medical man, however busy he may be, when consulted regarding any general condition of health." But for the last two years I have made much more thorough examinations, using a mirror and going over all the gums carefully with swabs of wool, and the result is that a large number of patients are found to be suffering from definite gingivitis or pyorrhœa which would have escaped detection in a less careful examination. It must be remarked in passing that, though the danger of oral sepsis has been recognized by a small percentage of dentists and doctors, by both doctor and dentist as a rule a slight or moderate degree is treated as of slight importance. It is precisely when it is slight that the most strenuous efforts should be made to effect a cure—the small fire being so much easier to extinguish than the large one, though potentially quite as dangerous.

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[1] *Proceedings*, 1909-10, iii (Odont. Sect.), pp. 98, 103.

But quite apart from septic gums and defective teeth, the food habits of the vast majority of these 115 were altogether bad. Sixty-four admitted to bolting their food; 109 allowed soft foods such as porridge and milk puddings to go down into the stomach without any attempt to insalivate them—using the mouth like an opening into a letter-box—and 110 drank during their meals. It will be said that these are common habits. Yes, common in civilized life, and intestinal putrefaction and its results are common too. Fibrositis is common, and some form of malaise in people aged over 40 is regarded as almost natural. But however common these habits are, they are none the less bad.

As regards mastication, we are all agreed—theoretically—as to its importance; but the need of insalivation is neither appreciated nor insisted on. Moist foods, as we know from Pavlov, excite no natural flow of saliva. Yet softened starchy foods are taken almost daily by a large number of people, no attempt being made to mix them with saliva. And I may once more reiterate my conviction, founded on experiment and experience, that even milk to be properly digested requires admixture with saliva, and should be sucked, not merely sipped. The fact that no animal in creation but man, and the few domestic animals dependent on him, can obtain milk except by sucking ought of itself to suggest to us that the habit of drinking or even sipping milk, and of bolting it mixed with some form of starch, is physiologically wrong. The common habit of drinking with meals, too, leads to a diminished flow of saliva, and the bulk of the liquid added to that of the solid food tends to overstretch the stomach. And a stomach overstretched day after day for years tends to become and becomes a dilated organ, and food is consequently retained in it too long and becomes subject to fermentative or putrefactive changes. Time will not allow me to go into detail as regards other faulty food habits, but one may mention too frequent meals, the appeal to the palate by seasonings, &c., instead of relying on healthy appetite, as habits which lead to civilized man taking far more food than he needs, and therefore having from this source, too, further excess of waste matter which will need excretion. Excess of alcohol and excess of sugar (because sugar being especially a muscle food is not required by people leading sedentary lives) are recognized as harmful factors.

Without reference to other septic foci than those of the mouth and nose, it has been shown, I think, that there is abundant evidence of habits or conditions leading to excessive toxin formation after early infancy. Now let us consider how far there is also defective excretion. Taking the excretory organs in order: As regards the bowels it is not improbable that they do exercise a true excretory function—i.e., that from the bowel there is an excretion not merely of the residue of foodstuffs, or of materials formed from the foodstuffs, but some catabolic products from the body generally.[1] Where there is constipation, not only is there much more absorption of toxin from the bowel, but also metabolic toxins which should normally be excreted by it are, too, obviously retained. And chronic constipation is, as we have seen, present in fifty-six of the cases under review.

As regards the lungs, it is difficult to prove that any toxin is absorbed by them, or that there is defective elimination through them. Dr. Leonard Hill does not believe that, except in cases where the vitiation is very great, the evil effects of living in closed, or nearly closed, rooms comes from the chemical impurity of the air. That is to say, that excretion of waste products from the lungs is not interfered with in a slightly impure atmosphere. Clinical experience convinces me that he is wrong, and that indoors, as most houses and buildings are ventilated, we either absorb toxins or fail to get rid of some toxic material. I am in agreement myself with Dr. Alex Hill's statement,[2] "that there is no degree of vitiation of the air we breathe which can be pronounced innocuous." Laboratory experiments are not convincing on this point, as they are not carried on for years on end, whereas we human beings, most of us, day after day, for the whole of our lives, continue to live in a vitiated atmosphere. Luciani (vol. ii, p. 384) speaks of "the undeniable fact that the oxidizing process is the most important in the animal body." Now it is quite certain that defective respiration must and does lead to defective oxidation, and the habit of defective respiration is common. The chest movements are, in the average man, imperfect. I feel sure that we have taken a wrong standard as regards fullness and frequency of respiratory movements, and have regarded the average as the physiological normal. Apart from other evidence of defective oxidation, out of the 115 cases no fewer than 40 had more or less bluish lips. There is one other factor leading to defective excretion through the lungs, and that is the growing habit of living in a warm, moist atmosphere. The absolute humidity of these warm rooms is very often considerable, and this interferes very considerably with the work of the lungs and skin. Evidence of habits (apart from want of exercise) which would lead to defective excretion through the lungs is afforded by the following data of the 115 patients: 28 expressed their dislike to open windows day or night under any condition; 56 did not have windows open at night; 33 had a window open at the top at night, but covered with blinds or curtains; and only three had windows widely open at night, allowing free currents of air to play about their rooms.

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[1] See Luciani's "Physiology," translated by F. Welby, ii, pp. 342-75.

[2] "The Body at Work," 1908, p. 192.

As regards the skin, one of the largest organs of the human body, it is hardly an exaggeration to say that among the western civilized nations there seems to be a general opinion that it is of no use; at any rate it is hardly used and is very much abused. For an organ to perform its duties properly it must be not only healthy but be in constant activity under physiological conditions. Though the main duties of the skin are protective, its excretory functions are considerable.[1] How can it perform those duties properly if it is kept, as the civilized skin is, in a state of continual congestion. By relying day after day, and week after week, on external coverings, or warmed air, to give us the feeling of warmth of skin which we have agreed to call comfort, the power of the skin to respond to stimuli of heat and cold has almost been destroyed; by covering it with impervious clothing its respiratory and perspiratory functions have been rendered imperfect and abnormal.

But as regards habits affecting the skin—other than that of being in warm, unventilated rooms—the following facts obtained from my 115 patients may be of interest: 60 of them had hot baths regularly, using hot water only; 14 had warm baths—I have often found that "warm" meant something over 100° F.; 14 had a tepid bath; 6 an occasional hot bath; 2 cold baths; 3 a hot bath followed by cold sponging; and 16 of these well-to-do people—i.e., about 12 per cent.—took no bath at all. I shall allude later to the evil results in fibrositis of the hot bath. But I may say at once that none of these people, except those who took the hot, followed by cold, bath, were really encouraging the physiological activities of the skin.

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[1] *See* Luciani's "Physiology," ii, chap. 9; and Leonard Hill, "Recent Advances in Physiology," p. 263.

Another habit leading to interference with the true function of the skin is that of wearing impervious clothing, such as skins, furs, &c., and also the use of the eiderdown or counterpane at night.[1] Forty-eight out of the 115 declared that in at all cold weather they could not do without an eiderdown. The person who sleeps under an eiderdown has his skin in a more or less parboiled or coddled condition, and he lies in a bath of his own cutaneous excretions, as it is impossible for the moisture that is exhaled through the skin to pass away through the dozen or so holes in the eiderdown.

We now come to what, for many reasons, one must regard as the main organs of elimination—the kidneys. Whatever the toxins may be that pass through them, no one doubts that the toxins are largely excreted by the kidneys.[2] But there is no doubt that, apart from special toxins, it is through the kidneys that waste matter, especially any that is imperfectly oxidized or imperfectly oxidizable, leaves the body, and the retention of those substances in the tissues, or in any part of them, must necessarily act as an irritant.

Now, if we consider the urinary excretion of the suckling, or even of the very young child, we find that although there is every reason to believe there is no retention of waste products—that is, that the body is every day excreting its daily waste completely—the proportion of solid matter to water is remarkably low. In Case's "Practice of Pædiatrics," 1906, pp. 770-71, I find a table compiled from the studies of Holt, Churchill, Morse, and other observers, which shows that in the first three years of life the *average* specific gravity of urine is between 1006 and 1012. As in the case of the frequency of respiration, so the average specific gravity of urine has come to be regarded as the normal or physiological one. But I venture to suggest that urine of a specific gravity from 1016 to 1020, which is accepted as normal for the adult, is urine containing too much solid matter If this is the case, is it not obvious that the less soluble substances would tend to remain behind in the body when the quantity of water that can be spared by the body for urinary excretion is deficient? However this may be generally, in the 115 cases under consideration there was evidence of deficiency of water in the urine in 63 cases. Apart from my own analyses, specific gravity and so on, my justification for this statement was that these 63 told me that their urine frequently deposited urates. This is very largely due to insufficient water drinking. Thirty-one persons stated that they never took plain water, hot or cold, whether with or apart from meals, and only 27 took water, generally hot water, apart from meals, and with only 4 was the quantity greater than a pint. And in 47 the quantity of fluid of all kinds, including alcoholic drinks, tea and coffee, was manifestly considerably below the body's needs.

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[1] *See* Leonard Williams, "Tubercle and Underwear," *Clin. Journ.*, 1908-9, xxxiii, p. 190.

[2] As to special toxins, *see* Luciani, ii, p. 415.

Clinical experience, again, convinces me that when leading a physiological life the specific gravity of the urine passed by an adult should not be much, if at all, in excess of that of the suckling, and with such urine there would be little danger of retention of any of the waste products which should pass through the kidneys. This brings me to mention common salt again. The evidence of imperfect renal excretion in these and in many other cases being so clear, does it not seem irrational for us to take into the body from 15 to 20 grm. of sodium chloride daily, which has to be excreted normally by the kidneys? It is not only that this uses up water which would be otherwise available for dissolving and washing out endogenous waste products, but it renders less soluble any organic salts with a sodium base.[1] In my cases, quite apart from any question of harmfulness of a moderate amount of added sodium chloride, 43 took more than an average amount. The effect of other extraneous mineral substances in causing fibrosis is at least suggestive that sodium chloride may have the same effect, and I believe it has.

I think I have adduced a fair amount of evidence tending to show that after the suckling age retention of waste products and toxins is exceedingly likely to occur, and personally I am convinced that it does occur and is exceedingly common. We talk continually of defective elimination—the treatment at a spa is said to be eliminative—eliminative of what? Of toxic matter, not from septic foci only but from the body generally. We have no term to apply to this condition of imperfect cleansing of the tissues of the body, and I venture to suggest that we need such a word. A classical friend has suggested "dyscatharsia," pointing out that catharsis is used both in Greek and English for a cleansing of the body generally, and not by the bowel alone. In connexion with this dyscatharsia, and especially with fibrositis, I want to bring before your notice one almost constant symptom which has not been mentioned in any book or paper I have read, but which I find is always to be found if one looks for it—viz., pigmentation of skin. It is not confined to fibrositis, but in every single case of fibrositis that I have seen there has been a general sallowness of skin: the more severe and chronic the symptoms are, the more marked is the pigmentation, and in the worst cases it is not merely general, but there are places here and there of deeper pigmentation. The splashes and splotches have not infrequently been recorded in connexion with rheumatoid arthritis, von Recklinghausen's disease, morbid uterine conditions, cancer, pregnancy, &c. What has apparently escaped recognition is, that those splotches occur in a skin already generally pigmented. The effect of chronic irritants externally in causing pigmentation of skin is well known. I suggest that this pigmentation of skin from inside is entirely due to the presence and irritation of retained toxins. Its detection depends on careful examination of unexposed parts of the body not subject to friction, and I generally examine the flexor surface of the upper part of the forearm. In slight cases it is revealed only by the result of treatment, but in a very large number of cases it is at once apparent to the practised eye, and if the patient is a woman, and is asked about the colour of her skin, she will almost invariably reply that she, or her friends, have noticed that she is becoming sallow. The fact that with suitable treatment the pigmentation of skin disappears almost *pari passu* with the disappearance of the symptoms of fibrositis—even the deep splotches fading away after greatly extended treatment—goes to show that pigmentation and fibrositis are due to a common cause. Assuming, for the sake of argument, that this dyscatharsia does exist, how will the body dispose of toxic waste products "which diffuse with difficulty"? Will it not deal with these exactly as a river deals with débris which it cannot dissolve and cannot keep in suspension? The river deposits its débris wherever there is any retardation of the rate of its flow, but especially its finer débris is deposited where the stream widens out, or wherever it may lazily and not actively overflow its banks. Similarly in the body, accumulation of unexcreted waste material would be deposited where the blood-stream runs most slowly and in tissues that are not adequately used.

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[1] *See* Hopkins and Hope, *Journ. of Physiol.*, xxiii, p. 284.

If we consider the localities in which fibrositis is most frequently found, we shall find that it is precisely where the blood-stream broadens out at the periphery in and beneath the skin and in tissues that are least used and least moved, and in tissues damaged by injury or strain, where power of resistance has necessarily been lowered. The localities where we get fibrositis most commonly are on the back, neck, shoulders, back of leg, heel, upper arm, about hip and gluteal region. The vast majority of us do not once in a month get our arms fully extended above our heads, laterally or posteriorly; we rarely, if ever, fully flex or extend the muscles of our neck or back. Full extension or flexion at the ankle is almost unknown, and generally if we really stretch any muscles they are very few in number, and not the ones generally subject to fibrositis. The comparative rarity, except in cases of injury or strain, or in acute infective conditions when several joints are affected, of fibrositis about the elbow-joint should make us think. The elbow-joint, more than any joint in the body, gets daily and frequently almost complete physiological movement, so that in the structures round it there is plenty of movement, and, in consequence, a good blood supply. But the hip, the shoulder, the neck, the ankle, and the smaller joints of the foot do not get moved in this way. But quite apart from deposits in tendons and fibrous structures, there is another part of the body where tissue exists which in civilized life is not used—I refer to the skin and the subcutaneous tissue.

The habit of using warm rooms, hot baths, eiderdowns, and impervious clothing, leads inevitably to a passive congestion of skin and the tissue beneath it. Take the hot bath: To bring the blood to the surface of the body artificially day after day and year after year and use no artificial means to empty the structures thus rendered hyperæmic, necessarily leads to a chronic congestion; the blood-stream is retarded just where it is widest and is naturally flowing least rapidly, and, as in a river, rubbish gets deposited. In my own cases I found that nearly all had more or less superficial tenderness which could be elicited on pressure; in 35 cases this tenderness was excessive. A large majority of these people were more or less obese; and I am in agreement with Professor Stockman in regarding at any rate much that is described as Dercum's disease as a fibrositis in the obese. I may say that 58 of the 115 cases, or over 50 per cent., were more or less obese. The painful fat is, again, specially to be found over the unused muscles of the body, the back of the neck, between the shoulders, the back of the upper arm, on the breasts in women, and the abdomen and buttocks.

Other evidence of extensive fibrotic changes beneath the skin is the tendency to bruise easily on slight pressure, or without any ascertainable cause. This was present in 48 out of the 115 cases. It is fairly obvious that this is due to fibrotic degeneration of the walls of the vessels.

To conclude our examination of daily habits, imperfect exercise was a feature in almost all my 115 cases. As exercise is an absolute essential both for healthy metabolism and excretion, the effects of this are almost incalculable. General exercises were taken by one daily, and by two occasionally. Walking was the only exercise of 50, and of these 10 admitted that their walk was a short one; 38 admitted that they took no exercise whatever; and these were not the patients of advanced age, but rather markedly patients between 30 and 50.

If this contention that the overgrowth and inflammation of fibrous tissue is due to retained toxins is correct, and that the formation and retention of toxins is due to faulty habits, what light does it throw on the rational treatment of fibrositis? First, it is obvious that the true treatment is preventive. Secondly that, if fibrositis does exist the proper treatment is first to eliminate toxins which are retained, and which are largely contained in the fibrous tissues, and then to guard carefully against a reaccumulation by attending to daily habits.

As regards the suggestions which follow, I should like to say that for complete cure and not mere temporary relief we must attend to *all* and not only to one or two causal factors. It is the whole underlying condition, and not some of its results, that requires radical treatment. First and foremost, attention must be paid to any septic foci, especially of the mouth and nose. To attempt elimination of toxins, and at the same time to have a factory of toxins busily at work, is obviously absurd. As regards oral sepsis, in all probability an edentulous man is in a better condition than a man with a mouth containing thirty-two undecayed teeth and half a dozen cesspools. It must not be thought that even the least, obvious, sepsis can be left alone with impunity. But this is not enough. With some modification and amplification I endorse the suggestions made by Professor Stockman, and also by Dr. Maxwell Telling in an excellent paper entitled "Nodular Fibromyositis, an Everyday Affection," which appeared in the *Lancet* of January 21, 1911. Massage comes first and foremost; the deposits of toxin *must* be, first of all, as it were, broken up and got once more into the blood-stream, so that they can be eliminated; movement and an active blood supply must take the place of an immobility and congestion. The fact that local swelling occurs, and not infrequently swelling of neighbouring glands, as a result of massage and movement is an indication of the actual and continued presence of toxins, the lymph being thrown at them when they are disturbed from the tissue in which they have been deposited, in an attempt to dissolve or otherwise move them on. The fact that pain results from massage too often suggests to both doctor and patient that massage must be wrong; but an explanation of the cause of the pain, that it is due to the swelling caused by the disturbed waste matter, with consequent pressure on sensitive nerves, but that, like dust in a corner of a room, it cannot be removed except by disturbing it and bringing it into evidence for the time being, will nearly always reconcile a patient to continue the treatment. But in severe or chronic cases a warning should be given at the beginning that though some relief may be experienced fairly soon, weeks or many months of continuous treatment may be necessary for a complete cure. The massage must be thorough and be given by a skilled masseur. It must be general as well as local. It is of no use in spring cleaning to get the dust merely out of the corners and allow it to settle elsewhere—it must be eliminated from the whole room. But I am in full agreement with Professor Stockman and Dr. Telling that, in the words of the latter, "the tender and infiltrated areas should be specially marked out for treatment." Constitutional disturbance not infrequently follows massage—sense of fatigue, headache, general malaise, and, especially in the young, some degree of fever. If the fever is at all considerable, it probably means that more toxin is being disturbed and thrown into circulation than the eliminative powers of the body can deal with, and in that case the severity or duration of the massage should be temporarily reduced. The skin must be got into a healthy condition and toned up by means of the alternation of hot and cold douches, avoidance of warm, moist rooms, and impervious clothing. Beginning gradually with the massage, we should have first passive and then active exercise involving *all* parts of the body, but especially of parts not as a rule moved properly in daily life. It is not enough by means of massage to get rid of deposits of toxic matter—we must not let them reaccumulate, so the exercises must not be for a week or a month, but so far as possible must be such as to obtain the full physiological use of the muscles involved for the rest of life. The skin, besides being brought into a condition of activity, should be well rubbed every day to prevent re-deposits there. Breathing exercises, to ensure a full expansion of the chest, are absolutely necessary.

As regards diet, I find myself to some extent disagreeing with Professor Stockman, as I regard it as most important. I exclude all food preserved by means of mineral substances—i.e., all salted foods—both on account of the salt and the absence of whatever the anti-scorbutic element may be. All food should be plainly cooked; entrées, hashes, stews, curries, and rich sauces and gravies should be avoided

Apart from that, all fresh, properly kept, meat, fish, poultry, eggs, vegetables and fruit, plainly cooked, are, and should be, given, avoiding only such things as salmon, mackerel or herring, or any other article that may be obnoxious to the particular individual. Rhubarb, too, I think, is generally to be avoided. But some uncooked vegetable or fruit, including in the latter dried fruits or nuts, should be given daily. Then, too, the vegetable foods, including bread, should be served with all their constituents. Long before Dr. Leonard Hill[1] made his experiments of feeding, and killing, rats on white bread I became convinced that wholemeal bread and vegetables cooked so as to retain all their salts were of enormous value in *all* conditions of health. The information we have got of the probable or certain causes of beriberi points to the folly of the human being regularly depriving himself in his vegetable foods of important constituents—of living on half instead of on whole foods. Good food habits are essential. Thorough mastication and thorough insalivation must be learned and practised, and the latter will never be arrived at by people who drink during their meals. There should be copious water drinking on an empty stomach, especially during massage treatment—i.e., first thing in the morning, last thing at night, and half to three-quarters of an hour before the mid-day and evening meals; the water being cold and plain tap-water, if a bacterially safe water is available. The presence or absence of some lime is of no importance whatever. People who say they cannot take cold water should be got to either suck it through a tube or have a small quantity of hot water added so as to bring it up to summer heat. Meals should be of few courses, and with proper intervals. Three meals a day, with at least a five hours' interval, are sufficient. As Dr. Hill says, "the organs concerned in the assimilation of food respond most readily and efficiently to the natural stimulus, food, if the meal has been preceded by a fast."[2] The ideas that improved nutrition is obtained by much food and many meals, as also that increase of weight is a sign of improved health, are prevalent, but wholly erroneous.

To be in fresh moving air by night and day is also essential—and here, as to moving air, I am in full agreement with Dr. Leonard Hill. Windows, wide open top and bottom with no blinds over them, ensure this better than any mechanical appliances.

Clothing, day and night, should be light and porous—for underwear

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[1] Leonard Hill, "Muscular Exercise and Open Air," *Brit. Med. Journ.*, 1912, ii, p. 601.

[2] "Recent Advances in Physiology and Bio-chemistry," by Leonard Hill, p. 507.

I prefer linen to cotton, woven so as to have a largish open mesh. Woollen underwear is most undesirable. Neither eiderdown nor counterpane should be on the bed at night.

As regards drugs, the fewer that are given the better. Some form of aperient is, in many cases, advisable for a time. In intestinal toxæmic conditions small doses of hyd. subchlor. are useful; and where there is not a dilated stomach liquid paraffin is sometimes beneficial. Intestinal antiseptics are often useful, and there is no doubt that small quantities of mineral acids and pepsin, especially where there is a dilated stomach, are very useful.[1] Analgesic drugs, of which aspirin is a type, are better left alone; they do not assist in elimination. Their use encourages the idea that pain is the worst feature of the disease, so that valuable time is lost in its early stages, and the relief they give to pain is at the best temporary. I have had patients who have been taking as much as a drachm of aspirin a day, under the doctor's eye, steadily going downhill for months, simply because relief of pain was the one thing aimed at. Giving up common salt does more good than taking potash salts.

As regards high frequency and other applications of electric current, including cataphoresis, I think they are useful to the extent that they improve the circulation of any parts affected, and not otherwise; and as to cataphoresis, it appears to me that our problem is not to get some fresh poison into the body, but to remove what is already there.

Active hyperæmia by hot, dry applications—especially of hot air—are useful if applied locally, and assisted by massage. But the general hot-air baths, where excessive heat is used, as in the Dowsing bath, in the long run, in cases of any severity, are as likely to do harm as good.

It will be seen from what has been said that, except so far as good massage requires training and experience, the essentials of treatment are, in my opinion, independent of time or place, and do not depend upon the fashionable medical craze of the day, whether it be Dowsing or Greville baths, Plombières treatment, cataphoresis, or the use of radium water.

Professor Stockman recommends spa treatment, and I fully agree, not only because at no other place can so much be done in a short time, but because when the patient is away from home and free from daily cares and duties, one can use the time to get him out of habits that are bad and initiate him into habits that are better. My own view being, in the words of an American editor of one of Dr. von Noorden's treatises, that "in sanatoria and in watering places too much attention is generally bestowed upon the immediate result, too little upon the ultimate effect; the most important element of the treatment being the education of the patient not for a few weeks but for the rest of his life."[1]

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[1] *See* a paper by Dr. Woodwark and Dr. Mackenzie Wallis, "The Relation of the Gastric Secretion to Rheumatoid Arthritis," *Lancet*, 1912, ii, p. 942.

Dr. A. P. LUFF said that with regard to Dr. Llewellyn's paper, he would only join issue with him on one point—viz., his remark that, in his experience, gout was a common ætiological factor in the development of fibrositis. He (Dr. Luff) saw many cases of gout, and many of fibrositis, and his experience was that gout was not a common factor in developing fibrositis. He felt in agreement with every word which Professor Stockman said, and it was to him that so much was owing in regard to the postulation of the pathology of fibrositis. He took it that most would agree that it was a hyperplasia of the white fibrous tissue, and that it was associated with both proliferation and exudation in relation with the connective tissue elements. An interesting point was as to what brought about that hyperplasia, exudation and proliferation. Dr. Schmidt had recently propounded a theory that fibrositis was a neuralgia of the muscle sensory nerves, a neuralgia dependent on a lesion of the posterior spinal roots. Dr. Schmidt had a case of fibrositis in a patient who died from some other complaint, and in which he made a careful examination of the spinal cord, but he could not discover any lesion of the posterior spinal roots. Moreover, anyone reading his paper must be impressed by the fact that Schmidt was constantly confusing fibrositis with different diseases, such as acute and subacute rheumatism. Therefore he thought Schmidt's theory must be abandoned.

With regard to Mr. Goadby's paper, this was very interesting to listen to, but in the light of his (the speaker's) experience it was very disappointing. What connexion could Mr. Goadby show between the microbic infection and the development of the fibrositis? As far as he gleaned, the paper amounted to this: that in a few cases of fibrositis in which there was thickening of the fibrous tissue around the joint but no actual arthritis of the joint itself, he found that, after the employment of massage and the Bier method of treatment, he could discover certain organisms in the urine which had not been found there before. Mr. Goadby described those organisms as of low pathogenicity, but he did not show that they were capable of producing fibrositis, and he (the speaker) did not think there could be a direct connexion between them and the fibrositis. He believed a microbic cause of fibrositis was very rare. He only knew of one case in which it had been fairly proved, and that was in a case described by Ware. This was in connexion with some fibrositis of the shoulder muscles around the shoulder-joint which was, at the time, the seat of gonococcal arthritis, and in the muscles which were affected with typical fibrositis he was able to show the presence of the gonococcal organism.

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[1] "Clinical Treatises on Pathology of Metabolism," by von Noorden, No. 9 (preface by Dr. Crofton), New York, 1910.

He would add just a few points to the discussion. All were aware that there were many forms of fibrositis; but he would draw attention to three to which he did not think sufficient attention had been devoted. One was in connexion with that common affection of a stiff and painful shoulder-joint, where there was imperfect use of the joint, any rotation or abduction causing much pain, and in which there was definite creaking, too frequently termed grating. He regarded these as cases of sub-acromial bursitis. The seat of this fibrositis was in the bursa which was sometimes called the sub-deltoid, but he preferred the name sub-acromial. He believed these cases had nothing to do with the joint of the shoulder, though in some of them the condition simulated an actual arthritis of the shoulder-joint. X-rays, however, showed the joint to be perfect. Under suitable treatment these cases cleared up, which they would not do if they were due to arthritis. The second form he wished to speak of was fibrositis of the abdominal muscles, which was not always recognized. He did not think that condition was rare. Nodular thickenings in those muscles could cause definite paroxysmal pain, which might lead to the erroneous opinion that the internal organs were at fault. Failure to recognize this condition was probably responsible for some of the many fruitless laparotomies for supposed appendicitis, renal colic, and so on. The pain could almost exactly simulate that associated with appendicitis, gastric ulcer and renal colic. The other form he wished to speak of was that of the occipital region, affecting the pericranial aponeuroses. An increasingly common form of headache in this region was due to a fibrositis affecting the muscles attached to the skull; it applied especially to the retro-colic muscles, and followed sitting in a draught. Motoring was responsible for a great increase of this affection, and particularly where a wind-screen was used in front, with the back of the car open, so that a powerful back draught was produced. He advised the banishment of the screen from the front, or the opening of the upper part of it. The condition was also found in people who sat by an open window in a fast train. The muscles particularly affected in such cases were those attached to the side of the skull.

With regard to the importance of spa treatment in fibrositis, there was no dispute whatever; therefore he would not discuss it. But he would say a word as to drugs and local treatment. Drugs which were beneficial for acute or subacute rheumatism were useless for fibrositis, hence they should only be given with the idea of relieving pain, not with any hope of curing the condition. His experience was that there was no drug so useful for fibrositis as iodide of potassium, and he always gave it in large doses, unless large doses could not be borne. It had a very direct action in removing serous exudation, and in reducing the hyperplasia of fibrous tissue. One of the best local applications he knew for fibrositis was the application of radiant heat followed by ionization. The heat he especially advised was from a leucodescent lamp of 500-candle power, to be followed by iodine ionization. He knew it had been said that iodine ions were caustic, and that chlorine ions were better, but he had not met with such causticity.

Mr. PAGAN LOWE said that he was especially impressed by Dr. Llewellyn's opening remarks with regard to the tonicity of muscles in fibrositis, in which he followed Sherrington's classification of two main groups—proprioceptive and exteroceptive. The importance of Professor Stockman's views lay, he thought, in the statement that he had never been able to find organisms in fibrositis nor had he been able to get cultures; and that brought him to the point of the few remarks with which he had to trouble the Section. He thought the profession should entirely give up the name of "chronic rheumatism," which was so generally applied to many forms of fibrositis. By "rheumatism" he understood an acute or subacute febrile disease, accompanied by joint pains and sweating, running a more or less definite course, and subject to relapses. But he did not think that there could be chronic rheumatism any more than chronic measles. Fibrositis might follow rheumatism in the same way that it might follow other infections, such as gonorrhœa or mucous colitis.

He thought it would be interesting to see the result of a series of experiments, carried out on the lines indicated by Professor Stockman, in patients who had been rendered radio-active. The difficulties associated with bacterial investigations in chronic disease were, he knew, extreme. Nevertheless, comparative examinations, before and after the patient had been rendered radio-active, should be attempted with regard to the opsonic and phagocytic changes of serum.

He joined issue with Dr. Luff in regard to his belief in the non-gouty origin of fibrositis. It was very difficult to prove whether fibrositis had any connexion with gout or not, but certainly it had very little connexion with rheumatism; in only about 8 per cent. of cases of fibrositis was there a history of acute rheumatism, but there was about 28 per cent. giving a history of gout. He agreed that iodide of potassium was the only drug of much value in fibrositis, but that was not a reason for regarding fibrositis as gouty any more than actinomycosis was gouty because it yielded to the same drug; still, he had an impression, from seeing many cases and going into their histories, that the people suffering from fibrositis had a gouty ancestry, even if they had not themselves had a definite attack of gout.

Dr. LENNOX WAINWRIGHT said that the greatest trouble connected with the subject was the remoteness of the cause of the fibrositis and the difficulty of assigning a cause. He was sure that many cases which were lowered by bacterial infection from oral sepsis and other sources were more subject to fibrositis than were others. The continuation of a cramped posture had much to do with stasis in the muscles, causing hyperplasia and, later, fibrositis. With regard to the heart condition, which Dr. James Mackenzie so forcibly put forward to account for many of the obscure murmurs which appeared and disappeared, he called attention to the mitral stenosis with progressive valvular disease which was always so troublesome, and which might have some association with fibrositis. In these cases, even where there was no history of rheumatism, there was much benefit received from the iodides. The question of blood-pressure and the state of the vessels had always to be considered, and he desired to utter a warning against the indiscriminate use of fibrolysin and other substances of a similar kind. He had found that in several cases where it was injected fibrolysin raised the blood-pressure. Where the fibrositis was associated with pyorrhœa and other mouth infections vaccines were very useful, though they were likely to be of but little use unless the focus were removed. He also had found leucodescent light and cataphoresis of iodides or salicylates immediately following a most valuable method of treatment. He had given iodopin in many cases which did not bear iodine well.

Dr. LEONARD WILLIAMS said it was a great relief to him, personally, to be able to join in a discussion in which one was not immediately overborne by a microbe. There had been something like an orgy of microbic theories thrust upon those belonging to that Section from time to time, and in every conceivable connexion; at the last meeting they had not escaped an invasion of that kind. From the remarks made in that Section, one might almost think that the microbe theory of disease had only just been discovered, that like a new toy or King Charles's head, it was produced on every occasion. Those who were so busy talking about the microbe seemed altogether to forget that although the seed might be sown, however potent it might be, unless the soil were in a receptive condition it would not germinate. That, he supposed, was the reason of Dr. Ackerley's incursion into the region of general principles of health drawn from the heavens above, the earth beneath, and the waters under the earth. He understood Dr. Ackerley to mean that the soil on which fibrositis grew was of a particular kind. He (Dr. Williams) had always believed that it was a gouty soil. But he did not know, and he did not think anybody else knew either, what a gouty soil was. He had himself suffered from fibrositis, and he had reason to consider that he had something in the nature of a gouty soil. There were two matters in connexion with attacks of fibrositis which he had not heard or seen noticed in the discussion, but which he thought required explanation. One was that the pains of fibrositis were so much worse at night. He had awakened at 6 o'clock in the morning with the feeling that he would be unable, on account of the pain, to dress; but matters would gradually but very materially improve as he got about. The other point was that the attack of fibrositis might come on with the suddenness of a blow from an unseen hand. One such attack which he had was experienced during a cold bath, and he could scarcely get out of the bath. Nothing which he had heard in the discussion explained those two facts. Another interesting question was as to why the attacks should be more frequent in a damp climate. He had known people resident in the South-west of England sufferers from fibrositis, whose removal to the drier climate eastward of the former place caused a cessation of the tendency to the attacks. The only other thing he had to say about the previous discussion was that he did not agree with Professor Stockman, and consequently with Dr. Luff, that fibrositis had anything to do with Dercum's disease. The pains in the latter affection were due to a different cause.

Dr. BUCKLEY said that after the remarks of one or two of the previous speakers he felt somewhat diffident about expressing his views; but he was a whole-hearted believer in the bacterial theory of fibrositis. He thought no one could have listened to Professor Stockman's paper without realizing that in fibrositis one met with the typical phases of non-suppurative inflammation. There was some leucocytosis, but a far more definite migration of fibroblasts, and from those fibroblasts the fibrous nodules developed. He did not think there would be a migration of those cells in the affected area, with the subsequent formation of fibrous tissue, without some definite causative factor. He was himself a motorist, and he frequently suffered from the type of fibrositis which Dr. Luff described; and he felt sure it was because at such times he had also some type of infection of the body, such as Mr. Goadby mentioned. He had been surprised at the number of cases of fibrositis in which he had found a coliform bacillus in the urine, which was obtained and examined with the greatest precautions against contamination. He thought it would be found that fibrositis was due, in a number of cases, to an organism belong to the group of colon bacilli. That view was much strengthened by Metchnikoff's observations on arterio-sclerosis. Metchnikoff had preached the doctrine that arterio-sclerosis was due to toxins from the large bowel. He (the speaker) believed that arterio-sclerosis and fibrositis were much the same thing—namely, proliferative inflammation of white fibrous tissue. Professor Stockman had pointed out how common was peri-arteritis and endarteritis of small arteries in fibrositis, changes very suggestive of arterio-sclerosis.

Dr. FORTESCUE FOX desired to dwell somewhat on the question of diagnosis, as it was important. With most of the writers on these subjects, he could not help feeling the necessity of maintaining the distinction between gout and rheumatism, and he was with Dr. Luff in continuing to draw that distinction. Every physician of experience must recognize that gout was subject to various disguises. He must also recognize the *rheumatic* subject and the *rheumatic* family. It was well known that if certain individuals were exposed to certain exciting causes of disease, such as damp cold, east winds, sudden barometric changes, traumatism, they responded in an uncomfortable manner, and developed symptoms which had long been described as rheumatic. He would also put in a plea for the retention of the word "rheumatic" as applied to those chronic ailments. They belonged to a definite type of constitution and were due to definite exciting causes. They were distinct from acute rheumatism, and represented a group of symptoms which practical people recognized. One important point of pathology struck him as having emerged from this discussion. Two of the papers laid emphasis on the fact that in fibrositis under all its forms there was a circulatory disturbance, and Professor Stockman said it was a vasomotor disturbance. He hoped members would get into their minds that this circulatory disturbance, which was really nervous, was at the root of the matter, because such a conception would assist in the right treatment being adopted. If cold and the other conditions he had named could bring on fibrositis, could not heat alleviate it? Did they not, in fact, find that when rheumatic persons resided in a climate favourable to them these symptoms were not developed? In observations sent from India good observers had noted that there were wide tracts in which there appeared to be no rheumatism. It must be recognized that in our own country a very different state of things obtained. Here, then, was a climatic disease analogous to catarrh, with attacks induced by physical agencies; and he argued that heat was a prime consideration in the relief of it. In spite of what Dr. Ackerley had said, he considered that baths held a very important position in the treatment of chronic rheumatism. Hot or warm baths equalized and "sedated" the circulation in and under the skin, and were perhaps more likely than any other agency to control the various localized disturbances. Dr. Luff's recommendation of iodide of potassium for rheumatism—for it was a very old remedy—reminded him of another which was not yet exploded—namely, sulphur. There was a tradition at sulphur spas all over the world that there was no ailment so much benefited by sulphur waters and baths as chronic rheumatism. A similar use of sulphur had survived in the well-known form of the "Chelsea pensioner."

Dr. F. Parkes Weber said he was sure all members knew how often the taking of salicylates, and especially acetyl-salicylic acid (aspirin), temporarily relieved the pains of fibrositis, notably the nocturnal pains.

He suggested that these drugs had a direct soothing effect upon the organs of Golgi and other nerve-endings in the tendons and fasciæ. That might explain the remarkable anodyne effect which they exercised in various forms of fibrositis. In confirmation of that theory he mentioned that if people suffered from similar pains, not due to fibrositis, but to actual injuries which affected thick fibrous tissue near the periosteum and the periosteum itself, these drugs sometimes relieved the pains resulting from the traumatism like they did those resulting from fibrositis. He had tried this in his own person for severe pains due to an injury to the lower right ribs. No bony crepitus could be felt, but an X-ray examination (skiagram) some time afterwards showed that two ribs were fractured, and that callus was already forming. As no crepitus had been obtained, the periosteum had probably not been completely torn, but doubtless the fracture had been sufficient intensely to irritate the organs of Golgi situated near the muscular attachments to the periosteum.

He was a great believer in the modern teleological explanation of pathological problems; by that he meant the Darwinian teleological view that there was some use in most of the phenomena seen in disease; that the result of a long process of natural evolution was not only the survival of the fittest, but also the survival of the means of survival—namely, the most suitable ways for the body to spontaneously and automatically react towards injuries and injurious agents of all kinds. In regard to fibrositis, however, an obvious "vicious circle" occurred, which had to be "broken." Why was the automatic teleological action of the body at fault? Fibrositis very often attacked persons who underworked and overfed their fibro-muscular system and lived indoors in stuffy rooms. When such persons suffered from the pains of fibrositis on movement, they naturally became still less inclined to take muscular exercise. To some extent, then, as far as one could judge, there was a really faulty reaction of the body towards fibrositis from the teleological point of view. The reason of this was to be found, he believed, in the fact that fibrositis was largely a pathological condition of civilized life, that is to say, it was a disease of late development from the evolutionary standpoint, though (since it included everything which was formerly classed under the term "muscular rheumatism" and a great deal of what was previously included under the term "chronic rheumatism") it was one of the oldest morbid conditions to be treated by baths and thermal springs and mineral waters, that is to say, by balneotherapeutic methods. The body had apparently not yet adapted its mechanism of automatic reactions so as successfully to oppose fibrositis. The sufferers from fibrositis in civilized life were not killed off, but probably passed on their fibrositic proclivities to their descendants. In wild animals and in primitive races of mankind he supposed that fibrositis was very rare, unless under artificial conditions (such as an enforced sedentary life). The human body seemed still, if one might so express oneself, to deal with fibrositis as if it mistook the fibrositic pains for the race's much older acquaintances, namely, the effects of traumatism—i.e., traumata to bones and periosteum—the proper healing of which required as an essential condition more or less local immobilization for a time. Hence the origin in mankind of the "vicious circle" just alluded to. In wild animals there was no chance of any such "vicious circle" arising. An injured wild animal was not likely to rest itself unnecessarily, because, more than an uninjured animal, it was obliged to guard itself from falling a prey to other animals.

Dr. Ackerley had alluded to cutaneous pigmentation amongst the sufferers from fibrositis. But he (Dr. Weber) had found that superficial pigment-nævi (using the term nævus to signify any congenital or early developmental "mark" on the skin) were very common amongst all persons, and he suggested that patches of brown or dirty-looking pigmented skin on the trunk, which were really the most superficial form of pigment-nævi—i.e., "simple" pigment-nævi—might easily, when met with amongst the subjects of fibrositis, be mistakenly regarded as in some way specially connected with the disease in question.

In regard to the term "fibrositis," he (Dr. Weber) acknowledged that, though a mongrel of Latin with a Greek termination, it was a convenient term to use, and conveyed a definite pathological idea. Yet, in using it, one robbed the original possessor—namely, "rheumatism"—of the greater part of his kingdom; for, as already stated, one now gave to fibrositis every morbid condition formerly known as "muscular rheumatism," and a great deal of so-called "chronic rheumatism" as well, all of which had probably belonged to the domain of "rheumatism" ever since the early days of balneotherapy at thermal springs. In regard to new medical terms and the abolition of old terms, the question was sometimes one of a choice of evils, and one might imagine the older and more popular, if less exact, term retorting to his younger, more exact, but less popular, brother, "They would not kill *me* to make *you* king."

Dr. Edgecombe, speaking of the relationship between gout and fibrositis, asked whether others had observed the clinical fact which had become impressed upon his mind—namely, the difference one found, on examination of the urine, in cases of fibrositis as compared with the urine of gouty people. In the former, acidity of urine was almost universally below normal, whereas in gout the acidity was above the normal. The urine commonly met with in fibrositis was of low-grade acidity, with large quantities of phosphates, presumably owing to deficient acidity in the gastric juice; and the administration of hydrochloric acid not only improved the digestion, but materially helped to dissipate the fibrositic symptoms.

Mr. Goadby, in reply, said that there was no need for any extensive answer. He was sorry he had not further convinced Dr. Luff. Yet he was not surprised, because he admitted that the evidence he brought forward was not direct. Much of it must of necessity be rather circumstantial, because he had not had success in making cultures from tendon or fibrous tissue. But Dr. Luff had apparently overlooked one point: he (the speaker) stated that he had succeeded in producing fibrous changes in the vicinity of tendons and intra-muscular tissue with the low type of pathogenic organism with which he was dealing. But when he was using streptococci and other organisms, the lesions were definite suppurative ones. The point about the class of low virulent organisms was that they produced not local suppuration when injected into animals, but the gradual growth of fibrous tissue without formation of pus, of the type seen in the photographs exhibited by Professor Stockman. He had not any sections of these fibrous patches at present, but he was investigating the lesions. The macroscopic changes simulated nodes. It was a point of some interest which should not be overlooked, and although the organisms might not be found locally, it was not improbable that their toxins were associated with local lymph changes. He knew that Dr. Leonard Williams had expressed his attitude towards bacteriology generally, and he agreed that there was much to be said about the misuse of bacteriological knowledge, but it was well not altogether to overlook it, and in a discussion of this kind, when one was dealing with phenomena quite possibly due to toxins or to a change produced by organisms, any facts bearing on the general bacteriological question were perhaps not out of place. At all events, in this discussion bacteriology deserved at least a subsidiary place. With regard to Dr. Buckley's question, he (Mr. Goadby) had found a certain type of organism, not in the urine but in the fæces of cases of chronic constipation associated with fibrositis, which gave the characteristics mentioned by Dr. Buckley; that was, it grew badly on bile salt broth; sometimes it did not stain by Gram's method. They were unlike true coli organisms in their growth, and he believed they were capable of forming certain curious protein degradation products. He had seen no adequate facts adduced during the discussion against the view that the fibrositis might be a toxic, or even directly bacterial, disease, not so much reason as there seemed to be in favour of his own bacteriological evidence that it might be so.

Dr. LLEWELLYN, in reply, said he based his remarks on the relationship of gout to fibrositis on the fact that in 1,200 cases 28 per cent. had stigmata of gout—tophi—or had previously had an attack in the great toe. The series included examples of neuritis, neuralgia, as well as arthritic and muscular types of fibrositis. He noticed Dr. Luff did not agree, but it was difficult to reconcile his present statement with one occurring in his book on gout, in which he alluded to brachial neuralgia, brachial neuritis, and sciatica, and laid stress on the important part which gout played in their ætiology. He (the speaker) took it that such cases of neuritis or neuralgia were due to fibrositis of the nerve-sheaths. Therefore if Dr. Luff found gout had a good deal to do with sciatica and brachial neuralgia, and neuritis, he failed to see how its influence could be excluded in lumbago, or other types of muscular fibrositis. The fibrous tissues were the site of the pathological lesion in both cases, and only topographically differentiated. Moreover, a large percentage of cases of sciatica were preceded by lumbago, and in the same way many cases of brachial neuralgia or neuritis were preceded by deltoid fibrositis. In both instances there was simply an extension of the morbid process from the interstitial fibrous tissues of the muscles to the contiguous nerve-sheaths. Granting, therefore, that gout was a potent ætiological factor in sciatica and brachialgia, the conclusion seems inevitable that the same cause was at work in the closely related muscular types of fibrositis. Attacks of lumbago frequently were replaced by or alternated with arthritic gout, and recently two cases of pleurodynia had come under his notice in which with the subsidence of the attack acute gout supervened in the great toe.

Regarding urinary hyperacidity in fibrositis, he would direct the attention of Dr. Edgecombe to some researches on the acidity of the urine in all forms of fibrositis recently published in the *Johns Hopkins*

Hospital Bulletin, and in which hyperacidity of the urine was an outstanding feature, its decline under a de-alkalizing regime being followed by a rapid amelioration of symptoms. He cordially supported Dr. Luff as to the importance of fibrositis of the abdominal wall muscles; he had known such cases confused with appendicitis and dyspepsia, and in an article by Adler that authority states that many innocent appendices had been removed in cases where it was afterwards found that the symptoms were due to nodules or infiltrations in the internal or external oblique abdominal muscles. He was glad that Dr. Luff had called attention to this very important form of fibrositis as a fertile source of many diagnostic errors. He could also confirm what Dr. Luff said as to sub-acromial bursitis: he had in a case recently under his care advised the removal of the bursa which was found to be extremely thickened. He could not hope in the short time which remained to deal satisfactorily with all the points which had been raised in this very fruitful debate.

Dr. ACKERLEY replied that there was a very essential diagnostic sign of fibrositis of an abdominal muscle. When the abdominal pain depended on disease of one of the organs pressure elicited less pain when the abdominal muscles were contracted, but when there was fibrositis pressure on the *contracted* muscle gave greater pain. In his full paper it would be found he had dealt with the questions of damp and cold. In answer to Dr. Fortescue Fox, he (the speaker) had had experience of one relatively dry country, Egypt, and he knew that rheumatism did exist there, though not to the same extent as in England. In Egypt the people chiefly affected were the women, whose life was more an indoor one. A moist atmosphere, whether warm or cold, aggravated the pain of fibrositis, and his suggestion was, that under those conditions neither lungs nor skin were adequately excreting.

Dr. BAIN said it was unfortunate that the adjective "rheumatic" should have been applied to this condition, because in many instances it occurred in gouty subjects. From both the pathological and clinical points of view the term "fibrositis" was appropriately indicated, therefore this term should be adhered to and all other designations discarded. Fibrositis was a common affection. It was more frequent in those aged over 40. Gastro-intestinal disturbance was a predisposing factor and chill an exciting cause. When the inflammatory exudation occurred in the panniculus adiposus the patches were much more painful on manipulation than was the case when they were situated more deeply, the explanation being that the nerve-endings were more numerous in the former region. The treatment of the affection might be briefly summarized thus: Treat the patient on rational lines, paying particular attention to the alimentary tract, and order massage. If the patches were in the panniculus adiposus massage must be carefully and skilfully performed.

Balneological and Climatological Section.

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Dr. F. A. de T. Mouillot, Vice-President of the Section, in the Chair.

The Significance, Treatment, and Prognosis of High Blood-pressure.

By Wilfrid Edgecombe, M.D.

Five years ago I had the honour of reading before you a paper entitled "Blood-pressure in Spa Practice" [6]; a plea for the routine observation of blood-pressure in the chronic cases with which, mainly, we have to deal. Since that time the method has come into more general use and the blood-pressure manometer is now included in the outfit of every spa physician. Much literature has appeared on the subject; many papers have been written by eminent men; and it is with some diffidence, therefore, that I venture again over well-trodden ground. I have no new facts to bring forward; my aim is briefly to review the significance, treatment and prognosis of high blood-pressure in the light of the added experience gained during the last five years.

Reliability of the Method.

At the outset it is worth while to touch briefly on the controversy as to the reliability of the method in common use—the mercurial manometer with broad armlet. Is the reading afforded by the manometer a true measure of the arterial pressure, either systolic or diastolic? How far do clinical observations lend support, on the one hand, to the views of Russell [11] that the instrumental reading represents the true lateral pressure within the vessels plus the resistance offered by the vessel wall, the latter factor being so increased by arterial hypertonus as to account largely for the high readings obtained; or, on the other hand, to the views of Hill [8] and his co-workers, that the readings afford a true measure of the systolic blood-pressure, the resistance offered by the vessel wall being too inconsiderable to affect the result appreciably. Clinically, in this regard, four types of cases are met with:—

(1) *The atheromatous type*, met with chiefly in old people, in whom the arteries are involuting, visibly and palpably tortuous, with lengths or patches here and there of calcareous thickening, separated, it may be, by lengths of soft-walled vessel. In these the pressure may be normal or low, but is not infrequently high, though nothing like to the same extent as in the diffuse form of arterio-sclerosis. Occasionally, different readings may be obtained from the right and left brachials respectively, the one being more calcareous than the other, and failing to be completely compressed by the armlet. In a series of senile cases in a workhouse infirmary an examination of the blood-pressure in twenty-five men, of ages ranging from 65 to 89, the average age being 75, all of whom had arteries sclerotic in various degrees, showed a difference in the systolic pressure on the two sides in eleven cases—the maximum difference being 25 mm. Hg. Similarly, in a series of sixteen women, of ages ranging from 61 to 89, averaging 74 years, there was a difference in systolic pressure on the two sides in five cases, the maximum difference being 30 mm. Hg. This source of error may be to some extent guarded against by taking readings from both brachials, and if necessary from both forearms, from which a fairly accurate estimate may be formed of the true blood-pressure. If the whole of the brachials and radials are hard and pipe-like I doubt if a true measure of the blood-pressure is ever derived from the armlet manometer.

(2) *The diffuse arterio-sclerotic type*, with thickened vessels, firm but not necessarily hard, uniform and not patchy. The manometer almost always gives a high reading, probably in most cases approximately correct. Corroborative evidence of long-continued high pressure is found in the heart and elsewhere. In those cases, occasionally met with, having a systolic blood-pressure apparently enormously high, incomplete obliteration of the vessel taking place with an armlet pressure of 300 mm., I am inclined to doubt the correctness of the method and to attribute the high reading to imperfect closure of the vessel owing to thickened walls. Co-existence of the nodular and diffuse forms of arterio-sclerosis would furnish an explanation. It seems

unlikely that such a pressure, if truly registered, could be tolerated for long without disaster.

(3) *The hyperpietic type*, in which the blood-pressure is moderately high, the vessels full and tense, but not firm or hard, and the heart as yet undamaged. In these the rise recorded by the instrument may be exaggerated owing to hypertonus of the vessel wall, relaxation of which leads to a speedy fall. No evidence of high pressure is obtainable from the heart and vessels, the mischief not having lasted sufficiently long to have caused gross changes; but evidence that the pressure is really above normal is to be found in subjective symptoms. If the hypertonus endures sclerotic changes set in and the high pressure becomes permanent.

(4) *The "neurotic" or "functional" type*, generally seen in women, but by no means uncommonly in men. The vessels are soft, apparently normal to the touch, and to the finger is conveyed the impression of a normal or low blood-pressure. On taking the manometer reading the systolic pressure is found to be above normal, sometimes to a considerable extent. The diastolic pressure, however, is not raised in the same proportion. The heart shows no displacement of the apex beat, nor evidence of hypertrophy by percussion, but the sounds are unduly forcible and the pulse-rate almost invariably raised. Undue apprehension at the moment of examination accounts in some cases for the disturbance; and when familiarity is engendered by repeated subsequent examinations the blood-pressure may be found normal. The rise in systolic pressure is merely temporary and is due to increased cardiac activity. But there are other cases of the nervous type, having no signs of arterio-sclerosis nor cardiac hypertrophy, in whom the blood-pressure, both systolic and diastolic, is constantly high, apart from transitory nervous excitation. Does the manometer give in these cases a true reading or an exaggerated one, due to spasm of the vessel wall? I believe the reading to be true and the explanation of the high blood-pressure to be that the pressure is set, as it were, at a higher level and is the expression of a more rapid rate of tissue life per unit of time. The condition is capable of differentiation from hyperpiesis and may not unjustly be termed "functional" high pressure as opposed to organic.

On the whole, then, with the exceptions stated, the method must be regarded as reliable and as furnishing a useful clinical guide. It is difficult to reject entirely the conception of arterial hypertonus giving rise to exaggerated readings, but inasmuch as the pressure is probably always above normal in these cases, though not perhaps to the extent shown by the instrument, and inasmuch as continued hypertonus may ultimately lead to sclerosis, the need for treatment arises and therefore the error is not of much account.

It is a matter for regret that no entirely satisfactory means has yet been devised for measuring the diastolic pressure. Determination of the point of maximum excursion of the indicator is subject to a wide personal equation. A graphic record by Erlanger's or by Gibson's instrument is impracticable in ordinary clinical work. Further, there is the uncertainty as to the exact interpretation to be placed on the pressure taken at the point of maximum oscillation. The importance of the measurement is manifest, for though the systolic pressure may be high, the stress on the heart and vessels is much less if the diastolic pressure be relatively low.

In the cases referred to subsequently the figures given for the diastolic pressure are approximate, and represent the pressure taken at the point of maximum oscillation of the indicator.

THE SIGNIFICANCE OF HIGH BLOOD-PRESSURE.

I am disposed to think that, during the last few years, as one result of the attention drawn to the subject, the symptom of high blood-pressure has been regarded in rather too serious a light, not only by the profession generally, but also in the mind of the laity, to whom imperfect, hazy knowledge has filtered, leading to the usual untoward consequences. Many a patient has been unduly alarmed, many have been unnecessarily treated, and many are by no means the better for our treatment of this condition. I do not wish to seem pessimistic, but to sound a note of caution as to excessive and unnecessary treatment. During the last five years my attitude has become one of greater optimism towards the subjects of high blood-pressure, and of greater pessimism as to the wisdom or utility of energetic treatment in a large proportion of them. Given a patient with a high blood-pressure, the following questions arise:—

(1) Is the pressure above the physiological limits? Woley [13] gives the results of a series of measurements made in 1,000 healthy individuals, of ages ranging from 15 to 65. The observations were made by five physicians, checking each other, and two different instruments were used. The following table summarizes their findings:—

	Mm. Hg.
Average pressure in males of all ages	127·5
,, ,, females ,,	120·0
,, ,, all persons, ages 15 to 30	122·0
Average high pressure	141·0
,, low ,,	103·0
,, ,, all persons, ages 30 to 40	127·0
Average high pressure	143·0
,, low ,,	107·0
,, ,, all persons, ages 40 to 50	130·0
Average high pressure	146·0
,, low ,,	113·0
,, ,, all persons, ages 50 to 60	132·0
Average high pressure	149·0
,, low ,,	115·0

Note : Average high pressure means the average of the highest 15 per cent.
,, low ,, ,, ,, ,, lowest 15 ,,

Sir Lauder Brunton [3] gives the systolic pressure as 100 to 120 from ages 15 to 21, and 120 to 150 from ages 21 to 65 ; figures which approximate closely to those above. It may be taken, then, in general terms, that up to the age of 40 an enduring pressure (systolic) of 150 mm. Hg., and up to the age of 60 or over an enduring pressure of 160, may be regarded as pathological. Differences in size, weight, and physique must, of course, be taken into account ; a man of 20 st. usually having, other things being equal, a higher normal pressure than one of 11 or 12 st. Also it must be borne in mind that even high pressures may show considerable diurnal variation.

(2) Is the high pressure temporary or permanent ? In a case showing no obvious signs of sclerosis repeated observation may be necessary, and the results of treatment gauged, before it is possible to determine whether the high pressure is due to transitory causes or is the expression of a permanent condition.

(3) If permanent, is it "functional" or organic ? The determination of this point rarely gives rise to difficulty ; most cases are easily relegated into one or other group. Occasionally the problem may be hard to solve. Tactile examination of the vessels, the condition of the heart and kidneys, together with evidence of the neurotic temperament derived from general observation, and inquiry into the personal history, will usually suffice for a distinction to be made.

(4) If "functional," does it require treatment at all ? In the large majority of instances I should say no. Here are brief notes of two cases, both in women, of functional high pressure that I have watched for some years. In neither has the high pressure given rise to any symptoms, nor do they seem the worse for it in any way.

Case I.—Mrs. P., aged 70: first seen in 1902 (then aged 59); highly strung, nervous woman; suffers from mild arthritis; vessels, heart and kidneys sound. Blood-pressure record as follows:—

Year	Blood-pressure Diastolic	Blood-pressure Systolic	Pulse-rate
1902	—	180	96
1904	—	160	90
1905	—	150	96
1906	120	150	96
1907	140	170	94
1909	130	160	88
1910	130	160	96
1911	110	155	84
1912	120	170	84

Case II.—Miss C., aged 55; highly strung, neurotic woman; first seen in 1908; vessels soft and uniform; heart slightly enlarged; no bruit; urine normal; no symptoms referable to the high blood-pressure.

Year	Blood-pressure Diastolic	Blood-pressure Systolic	Pulse-rate
1908	140	190	70
1909	140	200	72
1910	145	210	72
1911	140	200	70
1912	140	200	72

In neither case have any symptoms developed in consequence of the high blood-pressure. In the first, the systolic pressure is not unduly high, in the second it is uncomfortably so; but in both it will be noted that the diastolic pressure is relatively low, to which fact, no doubt, they owe their immunity from trouble. Formerly when cases of this type came under observation I made an attempt to reduce the blood-pressure by treatment—diet, baths, drugs, &c.—with the result that when the pressure did fall they were by no means so well as when it was at their normal high level. Interference in these cases was unnecessary, though happily not prolonged sufficiently to become harmful. When left alone they keep in good health—apart from the minor ailments which led them to consult me. They are typical of many, from whom I have learnt that masterly inactivity is sometimes wiser than fussy interference. Here is an example in a highly strung, nervous man who was sent to me last year as a case of arterio-sclerosis with high blood-pressure requiring energetic treatment.

Case III.—Mr. O., aged 50; good health all his life; no illnesses; has led an unblemished life; always been highly nervous and excitable; suffers from insomnia. His blood-pressure had been taken some months previously and had been found high, and the fact had been incautiously communicated to him. He had been treated by strict diet and a vigorous course of iodides and nitrites. His complaint was that he felt much worse while taking the medicines than without them. On examination his blood-pressure was 120 180, pulse-rate 104; heart not enlarged; a faint systolic bruit at apex and aortic areas: sounds unusually loud; vessels soft and uniform. Urine: Specific gravity 1.020, no albumin. The diastolic pressure, it will be noted, was low relatively to the systolic. There was no evidence of arterio-sclerosis, and the case was a typical one of "neurotic" high blood-pressure. He was reassured, his medicines were stopped, and his diet restored, which, with a course of bromide and valerian, speedily enabled him to regain his normal state of health.

Do these cases of what I have ventured to call "neurotic" or "functional" high blood-pressure live to the average term of life, or is it their tendency to be short-lived? Inasmuch as they live at a more rapid rate physiologically than the phlegmatic, low-pressured individual, they tend to wear out the quicker, but I am by no means convinced that their long-continued high pressure is of necessity a prominent factor in their premature decay. The interesting point is that they do not, as a rule, show signs of cardio-vascular degeneration, nor are they prone to die from vascular catastrophes. This may be explained by the fact that their diastolic pressure is low relatively to the systolic, and their vessels, therefore, are not in reality called upon to bear an excessive continuous stress; the stress is merely intermittent. Herein is emphasized the importance of not relying on the systolic pressure alone but of estimating the diastolic as well, and of considering the relation between the two. In a case, then, of high systolic pressure, with relatively low diastolic, and with the signs of cardio-vascular degeneration absent, no treatment *(ad hoc)* is as a rule required, and the prognosis is good.

(5) If organic, how far is treatment desirable, and if desirable, how far is it of any avail? In the first place, let us consider what we are called upon to treat. We make certain assumptions which are more or less capable of proof.

(*a*) That the cause of the high tension is the presence in the circulating blood of toxins of chemical or bacterial origin.

Proof of this is afforded by the action of adrenalin and of pituitary extract; the effects of the poison of specific infection, such as syphilis, and the infective fevers, notably typhoid; the work of Metchnikoff on the bacterial toxins formed in the alimentary canal; the more recent researches of Bain [2] and others, showing that pressor bases found in the urine and derived from the putrefaction of proteid in the bowel are instrumental in causing a rise of blood-pressure. Since Josué demonstrated that arterio-sclerosis could be produced in animals by the hypodermic injection of adrenalin, the suggestion has been made by Sajous [12] that toxæmia of various kinds may give rise to arterio-sclerosis not by the direct action of the particular toxin concerned, but indirectly through stimulation of the adrenal glands to excessive secretion. Cannon, Aub, and Binger [4] showed that nicotine in small doses caused, in cats, an augmentation of adrenal secretion. Gilbert, Petit, Wybaux, and others have shown that the infective fevers produce adrenal over-activity, sufficient to cause marked hypertrophy of the glands. Twenty years ago Abelous and Langlois showed that one of the functions of the suprarenal glands was to destroy the waste products of muscular work, and the suggestion is not unreasonable that prolonged over-activity of the glands thus induced may be the essential cause of the resultant arterio-sclerosis. From his experimental work G. von Anrep [1] concludes that every rise of blood-pressure brought about by the agency of the nervous system involves the co-operation of the chemical mechanism represented by the adrenal bodies.

In a recent address on high blood-pressure [9] by Sir William Osler it is stated that the arterial changes of the acute infections rarely take the form of a widespread sclerosis. With respect to typhoid, I have quite lately met with at least four cases of advanced generalized arterio-sclerosis to which no cause could be assigned other than a severe attack of typhoid fever years previously.

Case IV.—Miss C., aged 49; typhoid fifteen years ago, pulse-rate 122; blood-pressure 180/250; heart much enlarged; no bruit; vessels firm, hard, cordlike, uniform. Urine: 1.018, no albumin.

Case V.—Mrs. W., aged 48; typhoid eighteen years ago; pulse 84; pressure 200/280; heart enlarged; systolic bruit at aortic. Urine: 1.015, no albumin; vessels cordlike.

Case VI.—Miss G., aged 50; severe attack of typhoid twenty years ago; pulse 96; pressure 160/220; vessels firm and cordlike, heart enlarged, systolic bruit at aortic; no albumin.

Case VII.—Mr. G., aged 36; severe attack of typhoid at the age of 7, no other cause; has led a clean, moderate life; pulse 78; pressure 110/150; heart enlarged; vessels too easily felt, moderately firm. Urine: 1.015, no albumin.

In the first three cases sclerosis is fully developed; in the last there can be no doubt that it is commencing. A persistent systolic pressure of 150 in a young man aged 36 is ominous of future trouble. In cases of arterio-sclerosis in which the cause is obscure inquiry into the previous history will frequently elicit typhoid fever as the most likely factor.

(*b*) That the long-continued high tension leads to hypertrophy of the vessels and ultimately to sclerosis.

Proof of this is afforded by the morbid anatomy of vessels examined after various degrees and durations of high pressure. Or the problem may be viewed in another light, according to the suggestive theory advanced by French [7], who considers the toxin theory of arterio-sclerosis insufficient. He points out that sclerosis of the visceral vessels is constant in cases of prolonged high tension, and supposes the sequence of events to be: sclerosis of the visceral vessels resulting from excess of food plus sedentary habits; diminished function of the viscera; high tension of the peripheral circulation established compensatorily to ensure an adequate blood supply to the internal organs; peripheral arterio-sclerosis following on long-continued hypertension.

(*c*) That we can control or limit the supply of toxins.

In so far as it applies to toxins generated in and absorbed from the alimentary canal, this assumption is in part true. By modifying the diet, diminishing fermentation, and promoting excretion, we can limit the supply of toxins. In so far as it applies to other forms of toxæmia, for example the infective diseases, we are powerless, in the present state of our knowledge, to prevent the consequences of infection; prevention of infection alone is possible. Similarly with regard to mental or physical stress; if this acts, as has been suggested, through stimulation of an excess of adrenal secretion, we have no means of limiting the amount produced except by lessening the work, at a time, probably, when irreparable mischief has already been done.

(*d*) That we can influence vessels already affected by the morbid process.

The proof of this is wanting except with respect to iodides in syphilitic arteritis. That iodides will cause the absorption of fibrous tissue in other forms of arterio-sclerosis the proof is unconvincing. The same applies to decalcifying agents, such as citric acid and the citrates; also to fibrolysin, which has been occasionally used in this condition. Even were it possible to effect the partial absorption of hypertrophied tissue in the vessel walls, it is, to say the least, doubtful how far such a result would be salutary when we consider that the vessels have to support an increased pressure that has become necessary for the efficient performance of organic function.

In the second place, we must consider what becomes of cases of high blood-pressure when left untreated. The longer I follow up these cases the more am I struck by the length of time they survive and continue in apparently good health. Two examples may be quoted:—

Case VIII.—Mr. B., aged 63; first seen in 1902 (then aged 51): an active business man, leading a strenuous life; has had good health all his life; moderate in his habits, except that he has smoked to excess. His blood-pressure record is as follows. During the whole period he had no special treatment beyond an occasional very brief spa course, and throughout he maintained his unusually strenuous habits.

Year	Blood-pressure		Pulse-rate	
	Diastolic	Systolic		
1902	125	170	84	Heart sound, not enlarged; vessels normal
1903	120	160	80	,, ,, ,, ,,
1904	100	135	80	,, ,, ,, ,,
1905	100	130	80	,, ,, ,, ,,
1906	125	170	84	Apex displaced outwards, systolic bruit at aortic area; vessels readily felt; urine 1.015, no albumin
1907	130	180	82	Heart further enlarged
1908	120	160	78	Physical signs unchanged
1909	135	180	90	Breathless; slight œdema of legs
1910	130	175	84	Systolic bruit more marked; vessels firm; œdema increased; urine 1.006, trace of sugar, no albumin
1911	130	180	75	Developed chronic glaucoma
1912	120	165	80	Physical signs unchanged except that the vessels are becoming harder
1913	140	170	78	Systolic bruit, apex and aortic; urine, 2 per cent. sugar

This patient continues to follow his business, though perhaps with somewhat diminished vigour. His pressure throughout has not been superlatively high and the morbid changes are but slowly advancing. He probably has several years of useful life yet before him yet.

Case IX.—Mr. O., aged 63; seen in 1901 (referred to in previous paper, Case VI); marked gouty history; a free drinker and heavy smoker; an athlete in youth and has taken vigorous exercise all his life. Blood-pressure record:—

Year	Blood-pressure		
	Diastolic	Systolic	
1901	145	185	Heart enlarged; aortic second sound accentuated; no bruit; vessels hard and thick
1902	110	130	Had several attacks of epistaxis; apex beat in nipple line; systolic aortic bruit developed
1904	125	165	Systolic bruit more marked
1906	130	175	Apex beat 1 in. outside nipple line; diastolic aortic bruit as well as systolic; had epistaxis, after which pressure fell to 100/145
1907	120	165	Diastolic aortic bruit more marked
1908	120	175	Apex beat behind sixth rib, 2 in. outside nipple line; systolic aortic bruit very loud
1909	135	180	Systolic bruit at apex in addition to aortic; repeated epistaxis
1910	130	185	Œdema of legs; heart failing
1911	—	—	Heart failure gradually became more advanced, and he died early in 1912, at the age of 75

This patient was a subject unmindful of restraint, and had not Nature provided suitable treatment for him in the form of recurrent epistaxis it is doubtful if he would have lived so long, with such pronounced circulatory disabilities.

Again, a visit to the wards of any workhouse infirmary will convince one of the fact that these cases may live for many years without treatment, in spite of an arterial condition that may be described in some instances as truly appalling. In the series of cases already quoted, of the twenty-five men examined rather more than half showed typical involutionary or atheromatous sclerosis, the remainder belonging more to the type of generalized or fibrous arterio-sclerosis. It is difficult to separate them clearly as the two groups overlap, many showing both conditions, as far as could be detected by tactile examination. Their average age was 75; average systolic pressure 176, maximum 250, minimum 130; average diastolic pressure 120, maximum 160, minimum 95. The difference between the average systolic and diastolic pressures was 56 mm. Hg. A systolic pressure of over 200 was met with in seven cases, all of generalized sclerosis. A systolic aortic bruit occurred in seven cases, having an average pressure of 215. The urine in all cases was of good specific gravity, and contained no albumin. No case showed signs of hemiplegia either of hæmorrhagic or thrombotic origin. Of sixteen women, the average age was 74; average systolic pressure 179, maximum 260, minimum 135; average diastolic pressure 124, maximum 160, minimum 100. The difference between the average systolic and diastolic pressures was 55 mm. Hg. Three cases had a systolic pressure over 200; five cases showed a systolic aortic bruit, their average pressure being 193. Hemiplegia occurred in three cases. It may be objected that there is no evidence how long the high pressure had endured; but from the state of the vessels the process must have been one of many years. Also, that these represent the survival of the fittest in a vascular sense. This is true, and the probable reason for their survival lies in the interesting fact which emerges from the figures; that, although their average systolic pressure is high (176 in men, 179 in women) the diastolic is relatively low (120 in men, 124 in women). In other words, that the vascular stress has not been extraordinarily great. The conclusion, then, to be drawn from the foregoing is that the presence of a high systolic pressure need not necessarily fill us with undue alarm as it is not incompatible with many years of useful life.

In the third place, how far can we by treatment prolong life or avert disaster in these cases? It is difficult to institute a comparison between treated and untreated cases, for we have no means of knowing how long the former would have lived if untreated. In the following examples of declared arterio-sclerosis it is not irrational, however, to assume that life has been prolonged by suitable treatment.

Case X.—Mr. S., aged 62, bank manager; first seen in 1906; complains of breathlessness, attacks of dyspnœa and fainting; pulse 88, intermittent; pressure 170/210; apex beat 1½ in. outside nipple line; systolic bruit apex; pulmonary and aortic second sounds accentuated; vessels hard and cordlike. Urine: 1.015, trace of albumin. His mode of life was altered, he was dieted, his work cut down and rest enjoined, and he had a course of baths and massage twice a year.

Year	Blood pressure		Pulse-rate	
	Diastolic	Systolic		
1907	140	170	80	Kept well through the year
1909	150	190	78	Systolic bruit at aortic; urine 1.018, trace of albumin; attacks of dyspnœa more frequent
1910	160	195	—	Pulsus alternans; rate 96
1911	—	—	—	Death from heart failure

In view of his condition when first seen it is surprising that he survived as long as five years.

Case XI.—Miss P., aged 58; seen ten years ago, when she complained of headache, numbness and tingling of the right hand, with some loss of power. She was found to have generalized arterio-sclerosis, the cause remaining undiscovered. The blood-pressure was 150/200: heart enlarged: no bruit. Urine: 1.015, no albumin. She was put on a careful regime as to diet, exercise and work, which she carried out faithfully. Her drug treatment consisted of a long course of Trunecek's serum, periodic courses of iodine and the iodides, a bi-weekly mercurial pill, and occasional doses of nitrite of sodium, which always gave relief to her symptoms. During the years that followed her blood-pressure varied from 180 to 230 systolic, and from 150 to 180 diastolic, being never below the smaller figures. When it approached the higher level her old symptoms of headache, paresis, and paræsthesia would return, invariably on the left side and accompanied sometimes by anginal pain. She went about as usual and performed her duties in a modified degree, always leading a quiet life. The heart gradually enlarged further, and two years ago a systolic aortic bruit developed and the vessels became thicker and firmer. The urine gave an average specific gravity of 1.012 with, latterly, a trace of albumin. The pulse-rate remained uniformly about 66. In August, 1912, she quite suddenly developed auricular fibrillation: the pulse went up to 120 and showed the characteristic irregularity; dropsy supervened and the urine became loaded with albumin. A long period of rest in bed, with digitalis, wrought a marked improvement. The dropsy disappeared and the pulse came down to 70-80, though still irregular, the auricular fibrillation continuing. Since then she has taken her optimum dose of digitalis every day and keeps fairly well, though unable to do much. The heart is struggling against defeat, and how long it will take to succumb it is impossible to say, but there can be little doubt here, as in the preceding case, that careful following out of the treatment prescribed has resulted in prolongation of life.

While the treatment of well-established cases of arterio-sclerosis is, in a sense, unsatisfactory, in that a cure is not to be looked for, that of hyperpiesis, if caught early enough, is very gratifying.

Case XII.—In my former paper (Case I) I related the case of a university professor, aged 40, who was a good example of hyperpiesis, due to overloading of the viscera and a sedentary life of hard mental work. I saw him first in 1902, when his pressure was 145/190, reduced by treatment to 130/170. He altered his mode of life and followed out the regime laid down for him. His pressure subsequently has read: 1903, 120/150; 1904, 115/140: 1905, 100/135: 1906, 100/130. I saw him again in 1910 after he had had a nervous breakdown from various causes, when his pressure read 90/105, and after a course of tonic treatment 100/120. In 1911 the pressure was 100/120. In 1912 he had a mild attack of articular gout, and his pressure was 105/130. Through all these years there has been no return of the high blood pressure. I could parallel this case with many others, but as they all follow closely the same lines their narration would be tedious.

An attempt may now be made to answer the question, how far is treatment desirable? and if desirable, how far is it of any avail? In the early cases of hyperpiesis treatment is imperative and usually attended by excellent results. In declared arterio-sclerosis general measures should always be recommended, designed to limit the production of toxins and to facilitate their excretion, with the view of delaying the inevitable progress of the disease; together with suitable modifications as to mental work, stress, exercise, and so forth. If drug treatment—with iodides, decalcifying agents, nitrites, and the like—be attempted it should be with caution; bearing in mind that a middle course must be steered between the Scylla of disaster from cerebral hæmorrhage on the one hand, and the Charybdis of cardiac defeat on the other. That unwise attempts to avert the former may, by overdrugging, precipitate the latter is no unreal danger. A fairly trustworthy guide may be found in the statements of the patient. If he feels the better for the reduction of pressure consequent upon drugs they may be continued; if he complains, as so many of them do, that he feels worse when taking the medicines it is well to discontinue them, bearing in mind that the gradually acquired high pressure has become essential for the efficient performance of the circulatory functions under the altered conditions.

What should the treatment be? From the foregoing it will be gathered that, in my judgment, far greater reliance should be placed on general measures than on drug treatment. Diet stands first; the intake of nitrogenous food should be lessened and the total bulk of food reduced. Alcohol should be limited or forbidden and abundance of plain water taken apart from meals. Next in importance stands elimination; the output of waste products must be promoted by all excretory channels, bowels, kidneys, and skin. Intestinal putrefaction, and the consequent absorption of toxins, must be prevented or lessened. Exercise, graduated to the needs of each case, must be enjoined and free action of the skin promoted. Mental work and stress should, as far as possible, be regulated. That mental stress and overwork are potent factors in inducing high blood-pressure there is abundant evidence to show. In my experience the mentally hard-worked, harassed, free-living town-dweller is much more prone to suffer from generalized arterio-sclerosis than the physically hard-worked, plainer living out-door worker, who develops, rather, the focal atheromatous form. In this connexion I may quote an instructive case under my observation.

Case XIII.—Miss B., aged 30, came under my care in 1905 suffering from neuritis. Her blood-pressure was 80 100; in 1906, 85 110. In that year her circumstances changed, and since then she has, from various causes, led a life of continuous mental stress and worry which has become more acute during the last two years. Her pressure record reads: 1907, 100 120; 1908, 110 135; 1909, 105 125; 1910, 105 125; 1911, 110 145; 1912, 120 150. The pressure is slowly mounting and her vessels are becoming unduly palpable. A permanent systolic pressure of 150 at the age of 38 is suggestive of commencing arterio-sclerosis.

Into the details of these general measures I do not propose to go; they will be familiar to you all. Venesection must be mentioned as a useful proceeding in many cases. About drugs I have not much to say. The iodides, used so largely in arterio-sclerosis, owe their effect largely, I believe, to their action in reducing the viscosity of the blood. Insufficient importance has been attached to increased viscosity as a factor in the production of high pressure and consequent arterio-sclerosis. Cheinisse [5] draws attention to this and points out that in arterio-sclerosis iodide reduces the coefficient of viscosity from 5 to 10 per cent. after a fortnight's exhibition. Whether iodides can effect the absorption of newly formed fibrous tissue in the vessels is, to say the least, highly problematical. Too prolonged or excessive use of them tends to induce loss of tonicity and to accelerate cardiac defeat. Of the questionable utility of fibrolysin and of citric acid and the citrates as decalcifying agents I have already spoken. The nitrites and the whole group of vaso-dilators should be used sparingly to combat symptoms. The cases that react to them are the early ones in which the sclerotic process has not progressed far. Advanced cases will hardly react at all. A reduction of pressure effected by dilators is often of temporary service; but to use them freely, to secure a continuous reduction, is to invite impairment of cardiac tonicity and premature heart failure.

In the later stages of cardiac defeat digitalis is invaluable. The work of Price [10] and others has effectually laid the bogey of the risk of giving digitalis in cases of high blood-pressure. It may be given freely without raising the tension. In cardio-sclerosis the reaction to digitalis is said to be less than in the rheumatic heart, but in my experience the reaction has been good, more especially in those cases where auricular fibrillation has set in. The reaction probably depends on the amount of healthy muscle tissue left. No hesitation need be felt in giving digitalis, in the optimum dose, for months or even years continuously.

THE SPA TREATMENT OF HIGH PRESSURE.

In early cases of hyperpiesis incomparably the best treatment is that to be obtained at a good elimenatory spa. Removal of toxic material from the bowel by aperient water or intestinal lavage; stimulation of the liver; copious diuresis and diaphoresis; acceleration of metabolism by massage and massage baths; reduction of pressure by warm saline and other baths; exercise, freedom from business worries—all the desiderata are fulfilled. A course repeated twice a year at first, then yearly, due attention being paid in the interval to preventive measures, will suffice for the cure of most cases.

In declared arterio-sclerosis periodic spa treatment is of great service; the aim in view being the prevention, if possible, of further advance rather than the cure of mischief already done. The injudicious use of hot baths, as of drugs, in these cases may induce loss of tonicity and premature heart failure. Of radium emanation in the treatment of arterio-sclerosis I have no experience. Diathermic treatment may be helpful in relieving spasmodic cramp and intermittent claudication. The high frequency current is, in my experience, disappointing as a depressor agent. In the so-called "functional" or neurotic high-pressures sub-thermal immersion baths, from 90° to 98° F., are useful to allay cardio-vascular excitability and to promote a reduction of pressure.

PROGNOSIS.

What are the points that guide us in forming a prognosis in any given case of high blood-pressure? In hyperpiesis, if reaction to treatment is fairly rapid and satisfactory, the heart and vessels are probably as yet undamaged, and the prognosis is good. Recovery is permanent if a rational mode of life be followed. In declared arterio-sclerosis the following points help us:—

Age.—Other things being equal, the younger the patient with a high blood-pressure, not reacting to treatment, the worse the prognosis.

Height of Pressure.—Within certain limits, say from 170 to 200 systolic, the height of the pressure does not help us much in forming an estimate. The contest lies between pressure on the one side and heart and vessels on the other. A high pressure with strong vessels may not be as harmful as a lower pressure with weaker vessels; and inasmuch as by the methods at our command we can form at best but a very imperfect conception of the real state of the vessels (and especially of that indefinable thing, their quality) we cannot get much help to prognosis in this direction. In the higher ranges of pressure—250 and upwards—the prognosis is, of course, bad; and the higher the diastolic in relation to the systolic pressure the worse the outlook.

The State of the Heart and Vessels.—Estimation of the quality of the vessels and the extent to which they are damaged is necessarily imperfect. Beyond the aorta and the iliac arteries, when accessible to palpation, we can form little idea of the extent of *visceral* sclerosis. Even when the peripheral vessels are apparently hopelessly damaged it is surprising how long a case may last. Two may be briefly cited:—

Case XIV.—Miss L., aged 75; seen in 1907; slight hemiplegic attack; recurrent unilateral headache with hemianopsia; sensory phenomena in hands.

Year	Blood-pressure		Pulse-rate	
	Diastolic	Systolic		
1907	155	210	96	Heart enlarged; no bruit; vessels very hard, thin, and cordlike
1908	165	220	—	Anginal pain; breathlessness
1910	170	220	88	Irregular from extrasystoles
1912	175	230	84	Irregular; anginal pain worse

During the five years, in spite of her advanced age, and high pressure, her downhill progress has been very slow.

Case XV.—Mrs. W., aged 50; seen in 1909; attacks of loss of consciousness with temporary hemiplegia.

Year	Blood-pressure		
	Diastolic	Systolic	
1909	180	250	Heart enlarged; systolic bruit at apex; vessels markedly sclerosed
1913	200	280	Hemiplegic attacks more frequent

She is now practically confined to bed and able to do very little. It is remarkable that with so high a pressure she should have survived so long as four years.

Too gloomy a prognosis as to the probable duration of life need not be given even when the heart has begun to show definite signs of defeat. Cases X and XI narrated above are examples in point.

Pulse-rate.—In a case of high blood-pressure the slower the pulse-rate, generally speaking, the better the prognosis. A persistent pulse-rate of 90 or over is of serious import.

The Urine.—The presence of a trace of albumin in a urine of good specific gravity is not of much moment. Far more important is the specific gravity itself; if that be constantly low the prognosis is worse. A sudden increase in the amount of albumin is a bad sign.

Taken singly, these signs may not afford much help in prognosis; when more than one are present, and especially if all, the outlook is very bad, as the following case illustrates:—

Case XVI.—Mr. C., aged 44, schoolmaster; previous history good; no illnesses; has led a very strenuous life mentally. Early in 1911 he had a slight hemiplegia with aphasia from which he made an almost complete recovery. His pressure then was 170/250; pulse 108; heart much enlarged; systolic bruit at apex and aortic; vessels very full and firm. Urine: 1.010, trace of albumin. He was carefully treated and every precaution taken, in spite of which his pressure rose further to 260 at the end of 1911. All the signs mentioned as of grave import were present, and in consequence a bad prognosis was given. Early in 1912 he had a severe cerebral hæmorrhage and died.

Lastly, in giving a prognosis it should be borne in mind that the subjects of arterio-sclerosis are ill-fitted to stand the strain of any acute intercurrent disease.

SUMMARY.

(1) The measurement of blood-pressure by the mercurial manometer is, on the whole, reliable, and furnishes a useful clinical guide.

(2) There is a condition of "functional" or "neurotic" high blood-pressure not due to arterio-sclerosis and distinguishable from hyperpiesis.

(3) In the majority of cases of "functional" high blood-pressure, direct treatment is unnecessary and may be harmful.

(4) "Functional" high blood-pressure does not readily lead to arterio-sclerosis.

(5) The significance of high blood-pressure as a symptom is less serious than is usually supposed.

(6) The treatment of early cases of hyperpiesis is imperative, and is usually satisfactory. Spa treatment gives the best results.

(7) The treatment of developed arterio-sclerosis is unsatisfactory, and should be mainly by general measures and periodic spa treatment rather than by drugs.

(8) Excess of treatment may be harmful as liable to accelerate cardiac defeat.

(9) The prognosis in cases of high blood-pressure is less gloomy than usually considered.

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The Treatment of Confirmed Cases of High Blood-pressure; the Undesirability of actively applying Therapeutic Means to reduce it.

By Alfred Mantle, M.D.

The instrumental estimation of blood-pressure is comparatively new in the investigation of disease, and without doubt it has been very helpful in the treatment of certain cases. But whilst the sphygmomanometer gives us a reading which forms a good working basis in treatment, its readings must not always be considered as representing the exact measurement of blood-pressure, for in certain circumstances, particularly when the subject is emotional or unduly anxious, the readings may be higher than they should be, and this we should allow for to some extent. It is rather more than a year ago, when I opened a discussion at the Leeds Medico-Chirurgical Society on some aspects of high blood-pressure requiring special consideration in treatment, that I particularly pointed out that in some cases increased pressure remained high whatever measures were taken to lower it, and that it was wrong treatment to press vaso-dilator remedies under such circumstances.

It has been one of the unfortunate by-products of our recently acquired ability to determine a patient's blood-pressure in millimetres of mercury that the sight of the column near the top of the tube has so often led to immediate endeavours to reduce the pressure at any cost (Janeway). This point is not sufficiently recognized by some practitioners who make use of the instrument, and I get cases of high blood-pressure sent to me for special treatment at Harrogate to reduce it, in which nitrites, large doses of iodide of potassium, and other depressing remedies have been used, with the story that the pressure will only partially yield to treatment. The fact is that, owing to the gradual increase of blood-pressure, each individual readjusts his cardiovascular physiology to compensate this, and the physiological limit is now not 140 or 150 mm. Hg., but may be 220 or 250, and to reduce the pressure below this new physiological limit is not only not indicated, but bad therapy. "The fact should be emphasized that supernormal arterial pressure is not itself a primary condition to be attacked or controlled as such. It is invariably a result of some pathological cause or causes, and it is, moreover, often a necessary evil, the result of conditions we cannot remove, and then it must be looked upon as a compensatory effort, as one of the natural defences of the body" (Oliver).

A gentleman, aged 65, was referred to me two years ago as having had a high blood-pressure for some years, and he had had considerable therapeutic attention paid to it. He had been a great athlete, and rowed in his university boat three years. When I saw him the pressure showed a reading of 180 mm. Hg., and he was dyspnoeic on exertion. His heart was enlarged, the apex beat 1½ in. outside the nipple line with very irregular action. There was no albuminuria, and the vessels were not palpably thickened. It was clear that the compensatory hypertrophy which had helped him so long was failing. I gave him no lowering treatment, but that which was directed to the raising of his pressure, advising moderate exercise with plenty of rest between. I gave him strophanthus and nux vomica to raise his pressure, and Nauheim baths and Schott exercises to open out his peripheral circulation which was contracted. After three weeks' treatment his blood-pressure was 15 mm. higher, and the heart more regular, and he returned home much better. He made another visit last year, having taken strophanthus as required, under the direction of his doctor in the interim, and his blood-pressure was in a still more satisfactory condition, for it was steady at 200 mm. Hg., and he was able to do his ordinary amount of walking exercise quite comfortably. This was plainly a case where it was wrong to deprive the patient of a support and force necessary to maintain his smaller circulation, by giving vaso-dilator remedies indefinitely.

For three years I have had a patient under observation, aged 69, who when I first saw him had a blood-pressure of 220 mm. Hg., and with the exception of occasional attacks of giddiness after prolonged exertion, he suffered no serious symptoms. The heart's apex was very little out of its normal position, and his kidneys apparently were good. He had had constipation for years, and the sphincter ani had been stretched to relieve it. I therefore gave him a little intestinal lavation, but it was not found necessary to do so after a few treatments. His pressure was down to 200 after this treatment and some massage douche baths, but I heard from his doctor that it was soon back again to 220. He had been given 20 gr. doses of iodide of potassium to reduce the pressure for a considerable time, but with no marked result except to mentally depress him. My advice to him has been to live a healthy life, avoiding alcohol, red meat, and salt, to take aperients for the constipation, and exercise, and leave the iodide and other depressants alone. I have recently heard from him that he is keeping well and is continuing his regime.

The following case is an extreme example of a class not uncommonly met with: High blood-pressure with some cardiac hypertrophy and symptoms for years referable to a certain amount of disturbance of the liver and bowels in the middle-aged, who show no signs of arteriosclerosis in the systemic periphery, and no clinical evidence of nephritis after examination for albumin and casts. Sir Clifford Allbutt has called attention to these cases, and he names them "hyperpiesis." It is a curable condition if caught early, but if not it frequently ends in a permanently high pressure and possibly in apoplexy or cardiac defeat in trying to overcome the supernormal pressure.

A medical man, aged 58, was sent three years ago for Harrogate treatment. He had been seen by two physicans in a northern city who thought the symptoms I am about to describe due to the development of some cerebral disease. The patient had taken a fair amount of alcohol, but none for the five years previous to his illness, but he had been a big eater of red meat. He worked hard in a country practice, and was subject to bad headaches, for which he frequently took calomel and antipyrin. He had, however, what he called a seizure, which he described as being constituted by great pain and throbbing in the head, and marked giddiness in the erect posture. I was asked to see him in consultation with Dr. Black some weeks after this attack. He was very emotional, and was rather like a candidate for cerebral softening. He complained of throbbing in the head, and was more or less giddy in the erect posture. On examination, the pulse was one of very high pressure to the touch, and the sphygmomanometer showed a systolic pressure of 220 mm. Hg. As is my custom, I looked for corroboration of this in the position of the heart's apex, which was a full inch outside the nipple line. There was no albumin, and the urine was of good specific gravity. We came to the conclusion that the symptoms were due to the high blood-pressure, and that the only possible chance for the patient was to give him perfect rest with a very strict diet. All meat was forbidden as well as salt. He continued to have nothing but milk and fish for the three or four months whilst under our care. He was well purged and the skin was kept active, but he was much too ill to have any balneological treatment. The blood-pressure gradually came down, and the apex beat came in, and after four months' rest he was able to walk fairly comfortably without much throbbing or pulsation. The patient eventually got back to his work, and continues well. He lives practically on the same lines of diet, taking no red meat or alcohol, and is now able to do as hard a day's work as he ever did, and when visiting Harrogate last year we found his pressure 140 to 145 mm. Hg. only. This is an instance of what can be done for some cases of high blood-pressure in the way of treatment when seen early. This patient had no nitrites after the first week's treatment, for they had no effect in reducing the pressure, neither did he have iodides, and we may attribute his recovery to rest, alteration in diet, and more or less free purgation with calomel and salines.

A natural question to ask is, How are we to determine the true position of a case of supernormal pressure? These cases always give us anxiety, and rightly so, for we are confronted with two dangers, and with one eye we see visions of a possible catastrophe through the rupture of a weak vessel by doing too little and with the other the danger of doing too much in lowering the pressure and weakening the cardiac force. My reply to this is, take a careful history of the case, and observe the condition of the cardio-vascular, the renal and alimentary systems, and try to find out the cause of the high pressure, such as errors in diet, alcohol, syphilis, lead, and excessive worry, remembering that supernormal pressure is not a disease *per se*, but a symptom only. If the left ventricle is hypertrophied it suggests the pressure has been persistently high for some months or years. Experience teaches us that with a restriction of red meats and alcoholic drinks from the dietary of those who take them freely, the pressure, if not a permanently high one, usually diminishes, and this argues that purin bodies and alcohol probably act as vaso-constrictors. Waste is constantly going on in the body, and it is important to get rid of the products of waste whatever they may be. The proof of the splanchnic vessels being some of the earliest to become affected in arterio-sclerosis points to our relieving congestion of the visceral vessels, which can be done by judicious purgation, and 1 gr. doses of calomel and saline purgatives are most useful two or three times a week. One of the greatest outlets for waste products is the skin, and when inactive a free action of this organ must be encouraged by artificial means, such as by massage douche, and Nauheim baths, when these can be obtained, and intestinal lavation is also very desirable in cases with constipation. It is a common observation that with a dilated periphery causing an active skin we have a lower range of blood-pressure, which leads us to suggest to those who can afford it to follow the sun. The cardinal principles of treatment, then, resolve themselves into a strict regulation of the intake and an active outlet for the waste products, and moderate exercise followed by rest is very desirable to give a better distribution of the blood, but sphygmomanometric observations must be made from time to time as a guidance in treatment.

If, in spite of our regime and treatment, the pressure continues high, after having watched the case carefully for some months we conclude that this is a case in which the pressure is permanently supernormal. In any case we recommend the general regime to be continued, and so long as no urgent symptoms arise the patient may do his work in life if he avoids excitement and sudden strain.

Lastly, we must not use any active means to reduce the pressure, remembering that it is probably an advantage and necessary that the pressure should be high for the maintenance of an adequate supply of blood to vital organs. As for drugs of the vasodilator type—I mean the nitrite group—I use them very little except in emergencies, for I believe it is sounder treatment to get rid of the bodies in the system which contract the periphery by the means I have mentioned, rather than by dilating the blood-vessels with the offending poisons still intact. I give iodides very carefully if at all, but if there is any syphilitic history they may be pushed. As for gypsin, this I have given a trial in some cases, but with no marked results.

DISCUSSION.

Dr. BUCKLEY expressed his hearty concurrence in the views which had been expressed by both Dr. Edgecombe and Dr. Mantle on the treatment of cases of high blood-pressure. He believed that in a considerable number of cases it was a mistake to direct the treatment to this particular symptom. Except where definite ill-effects were likely to result from the high pressure *per se*, treatment must be directed to the underlying conditions. He would like, however, to hear their views on a case of his own. A woman, aged 38, during the three years she had been under observation, had had a blood-pressure varying from 230 to 270 systolic and 160 diastolic, associated with obsessions and a generally nervous temperament. Gradually the heart had enlarged; she had palpitation and occasional faintness, and albuminuria had developed. He had tried drugs of all kinds, dietetic measures, and long spells of rest in bed with free purgation, but nothing had had any effect except possibly thyroid gland, and the effect of this was not marked. It would take too long to go into a full account of the case, but he would much appreciate any suggestions as to treatment. Another case he had seen in a woman, aged 55, whose general health was quite good: there was nothing amiss with heart or kidneys, but her blood-pressure during four years had never been below 196, and generally ranged from 230 to 260 systolic, 135 diastolic. One feature, however, had struck him, she had rapidly aged and now looked very much older than her years. Treatment had been quite ineffective, and further attempts did not appear to him to be indicated. He thought it was a mistake to say much to a patient about blood-pressure; already the general public was taking too much interest in the subject, and in a nervous patient much harm might be done by suggesting there was anything abnormal in this respect: so many of the cases appeared to be neurotic and did not call for any treatment in this direction.

Dr. W. **Bezly Thorne** endorsed the views expressed by both Dr. Edgecombe and Dr. Mantle, especially as to the danger of attempting to lower blood-pressure by means of vaso-dilators. That proceeding, he said, hastened the failure of cardiac compensation, increasing general weakness and dyspnœa, and encouraging œdema. In the course of fifteen years of sphygmometric observations he had never known digitalis to raise blood-pressure. On the other hand, he had found the solution of adrenalin, orally administered, to lower maximal readings, apparently by bringing the muscular tissue of the arteries into action and stimulating conductivity. He strongly advised making diastolic in conjunction with maximal records of pressure.

Dr. R. **Ackerley** (Llandrindod Wells) said he could not regard high blood-pressure as the cause of the symptoms associated with it. As regards food, he did not think red meat or any other form of nitrogenous food was necessarily dangerous. Few meals, moderate or small in quantity, avoidance of common salt, and free flushings with water apart from meals, were the essential dietetic rules. Starchy foods, imperfectly insalivated, were dangerous, as they fermented in the bowel.

Dr. C. O. **Hawthorne** expressed the opinion that to present sphygmometric readings in terms of blood-pressure was hardly justifiable. He agreed that a mere high reading was not a claim for active treatment. He doubted if any drugs had a sustained effect on high readings, and the action of nitrites was only short-lived.

Dr. F. **Howard Humphris** wished to express his appreciation of the scholarly papers of Dr. Edgecombe and Dr. Mantle, and to concur with the principle on which emphasis has been laid that in not every case of high blood-pressure was treatment advisable; but he rose to speak on one statement which Dr. Edgecombe had made, because it touched upon a point which he thought should be made clearer. Dr. Edgecombe had said, in speaking of cases suitable for treatment, that he had found in high frequency "a disappointing agent." Now this was a statement which should not go forth with the imprimatur of this Society. Before naming it as disappointing or unsatisfactory was it not pertinent to ask the cause of the failure? Were the apparatus and the application of it such as to ensure success? The high frequency current for the reduction of blood-pressure *par excellence* was that of d'Arsonval, and from his (Dr. Humphris's) experience in this country the dose administered was usually from 200 to 300 ma., whereas the therapeutic dose was 1,000 ma. or more. Apart from other considerations this alone would make a difference between success and failure. With a dose of 1,000 ma. or more, according to the case, the blood-pressure should drop from 10 to 20 per cent. Thus the blood-pressure, which was 200 at the beginning of the treatment, became 180 or 160. This would gradually rise till the following day (but not always, however, to its original point), when another treatment should be given, but after a time it would be observed that the pressure no longer rose between the treatments. This might be taken to be the patient's normal pressure, and to keep it at this he would require to come for further treatments from time to time, as the varying conditions might cause an increase of the pressure. With d'Arsonvalization, properly administered, much more could be accomplished than with most measures employed for treating hypertension. With it, except in the very advanced cases, a patient could be kept for years in a condition of safety—judicious attention, of course, being paid to general lines of alimentation and exercise.

In reply to the President (Dr. Percy G. Lewis) who asked if the effect of the current was due to the increase of metabolism which the current caused, Dr. Humphris replied that the theories which had been advanced to account for the lowering by the current were many. There was no doubt that the current did cause an increase in metabolism. This had been demonstrated both clinically and chemically. There was a gain in physical and mental strength and general improvement in the health, and the symptoms involved, whether insomnia, palpitation, or anxious fears, disappeared. Chemically, there was an increase of solids in the urine, and this had also been found on analysis of the perspiration.

The improvement in general metabolism was probably largely due to the effect on the vasomotor system, whereby the arteries, becoming relaxed, permitted of a freer circulation of the blood, and if this one fact were granted, then the beneficial effects of the current were easily explained. A subsidiary cause might be at work, and that was the thermic effect of the current. This was quite apparent to the patient, and it might be that this caused an increase of functional activity by which the elimination of the toxic materials was augmented.

Balneological and Climatological Section.

May 20, 1913.

Sir HERMANN WEBER in the Chair.

THE SAMUEL HYDE MEMORIAL LECTURES

The Place of Climatology in Medicine.

By WILLIAM GORDON, M.D.

LECTURE I.

SIR HERMANN WEBER AND GENTLEMEN,—I have first—and the duty is a pleasant one—to offer to the Council and members of the Section my best thanks for the honour they have done me in asking me to deliver these, the second, Hyde Memorial Lectures, and for the added honour, Sir, that you preside. When their kind invitation came I felt a good deal of misgiving as to whether I ought to accept it; it seemed no light matter to me, whose province is general medicine, to address men in their own special department of knowledge, who are so well fitted to instruct myself. But, as I thought over the possible reasons why I had been asked, one seemed to suggest itself which encouraged me to make the attempt. The opening lectures of a long series, such as we must suppose that these will be, are not unlike the introduction to a bulky volume, and may be expected to survey the whole subject from a general point of view. Such an introduction I thought a general physician might write: such, at all events, I now venture to place before you. My predecessor, Dr. Fortescue Fox, in his interesting lectures in 1911, dealt in the broad fashion I refer to with the subject of hydrology, the *περι ὑδάτων* of Hippocrates. To me this year have fallen the *περι ἀέρων καὶ τόπων*. I propose to consider their place in contemporary medical study, to indicate their immense real importance, and, whilst noting their general present day neglect, to suggest certain remedies which may tend to restore them to the place that is rightfully theirs. How far my performance in this programme is going to fall short of my opportunity I am only too painfully aware. But, as Browning puts it, "let your better wit mend all."

My second duty—and privilege—is to call to your recollection him in whose honour these lectures have been named. Dr. Samuel Hyde was born at Stalybridge, in Cheshire, in 1849, and received his medical education at King's College, London. He settled at Buxton in 1877 as medical officer of the "Peak Hydropathic and Thermal Establishment," and afterwards in private practice, acquiring the reputation of a skilful, observant physician, thoughtful and thorough, deeply imbued with the faith of fruitful experience in the efficacy of waters and climate in the treatment of disease. Literature on the subject was then limited, and his editings and writings added valuably to what was available. In 1889 he inaugurated a journal of British and foreign health resorts, and in 1895 he and Dr. Septimus Sunderland founded the British Balneological and Climatological Society, the parent of this Section, thus providing for the first time a common meeting ground for the medical observers at the various British health resorts, a notable service to the medicine of this country, and one we do well specially to commemorate. Hyde also became the editor of the quarterly journal of the Society. Dr. Fox, who knew him well, has described him as an indefatigable worker, patient and persistent, with the imagination to conceive and foresee, the constructive ability, courage, and perseverance to achieve, a personality of rare effectiveness and charm. When death claimed him at the early age of 50, his work was already complete—a British school of balneology and climatology had been created.

THE IMPORTANCE OF MEDICAL CLIMATOLOGY.

To realize the importance of medical climatology it is only necessary to define it. Yet this is not so simple as might at first sight appear; definitions are generally difficult, and this one seems to have been found exceptionally so. Modern authorities have commonly dealt with climates merely in terms of average local weather. But in its medical aspects, at all events, climatology covers more than can be so expressed. When we talk of a climate being good or bad in a medical sense, it is not only weather which we have in mind. Doubtless something may be said for the view that the effects of climate are ultimately results of meteorological conditions, at least where neither the inhabitants nor their habitations go barefoot on the soil, and if we exclude hydrology. But many of these meteorological conditions cannot at present be determined, of the existence of some of them we are probably unaware; so that we are obliged, for practical purposes, to include in our definition, as in our investigations, topographical factors whose meteorological consequences still remain to be unravelled. In making this compromise it is curious to observe that we are but following the lead of the "Father of Medicine," who summed up his subjects as "airs, waters, and places."

I would therefore define "climate" and "climatology" in their medical aspects thus: "The climate of a place, in a medical sense, is the sum of the influences upon human health and sickness of its geographical, and especially of its meteorological, conditions"; and "Medical climatology is the science of climates medically considered and of their variations in space and time." So defined, it is clear that climatology constitutes a great part of the environment of medicine, and that to neglect it is to ignore much of the natural history of disease.

Medical climatology may be divided into "descriptive" and "theoretical"; "descriptive," embracing "medical geography," which treats of the distribution of disease over the earth, and "medical history," concerned with records of epidemics and those changes in prevalence or type which diseases undergo; "theoretical," including what may be termed "geographical" and "historical pathology," sciences which should set forth the laws which govern these distributions and periodicities.

THE PLACE OF CLIMATOLOGY IN THE PAST.

No survey of my subject could be considered complete which did not take cognizance of the past. Nor will a brief retrospect be devoid of practical advantage.

In the dawn of European thought, during the epoch in which the Greek genius attained its zenith, that is to say, in the sixth and fifth centuries before Christ, men apparently looked on life more directly and comprehensively than we do now; and sometimes reached, as it were intuitively, simpler and saner views. Thus it has been held by no mean modern thinkers that in the great writings of twenty-four centuries ago guidance is still to be gained in those confused problems which our murkier mental atmosphere presents to us. So it seems to be in medicine. For the work of Hippocrates on Climatology, brief as it is, translatable in less than 9,000 words, takes a view of the subject whose width is only beginning to be understood. In that work seasons and soils, winds and water, aspects, shelters and exposures, altitudes, temperatures, vegetation, the habits, food and drink of the inhabitants, are all taken into account, and not merely as affecting disease but normal development, not only the physical characters of races, but their moral characteristics—even the excellence of their art and their success in war. "Whoever," says the Master, "wishes to investigate medicine properly" must attend to these things.

It would be rash to conclude that so wide a presentment of the subject was entirely original in the time of the writer, and, in fact, we have indications that his predecessors and contemporaries were accustomed to lay stress on such considerations. Whether this arose from the peculiarities of their training in natural health resorts, or from the marked differences which obviously existed between climates familiar to them, we do not know. But the story that Empedocles of Agrigentum improved the climate of his native town in Sicily by blocking up an opening betwixt two hills shows that the ancients realized how much depended on knowledge of this kind.

This primitive width of outlook was not, however, maintained. So far as I can discover, subsequent writers gave the subject narrower treatment; even Aretæus, regarded as the second most distinguished of the physicians of antiquity, dismissed climatology almost in a page. Perhaps the great anatomical researches of Alexandria had turned men's minds into different channels. Celsus and Galen apparently had little of importance to say on the matter. The great Arabic commentators, whether at Baghdad or Cordova, appear almost to have passed it by. Even the Renaissance gave it slight attention. Sydenham in England was exceptional in the stress he laid on the seasonal varieties of disease, following in this his favourite model, Hippocrates. But even Sydenham, so far as I can see, made no valuable advance; and, until the great medical era of the nineteenth century, the place of medical climatology was far more limited than it had been in primitive times. Then, however, a wider activity set in. The extension of facilities for travel was succeeded by the growth of medical geography; medical history followed in natural sequence; and a study of their theory began. Further than this, historians like Buckle and Draper, philosophers like Herbert Spencer, took up the thread of speculation on the influence of climates upon races, where Hippocrates had let it fall; so that once more, after so many centuries of comparative neglect, the whole fabric of ancient thought upon the subject rose into view.

The Place of Climatology in the Present.

And this brings us to a survey of the extent of our present knowledge.

At the outset one must distinguish the factors which enter into climatology, so far as they have hitherto revealed themselves. Sixteen may perhaps be enumerated, half of them *meteorological*, half *topographical*. These are set forth in Table I. All of them have been considered in one way or another in relation to disease, and already our gains are great, though modest indeed compared to what they may be. But for satisfactory knowledge of their action we require not only to be acquainted with their relations to human disease, but also to know their influence on human beings in health, as well as on the parasites which produce human disease, and on such non-human hosts as harbour them.

Table I.

Climatological Factors.

Meteorological	Topographical
Temperature.	Latitude.
Wind.	Geographical position by which is meant relation to land and sea, lakes, mountains, &c.
Rainfall.	Altitude.
Sunlight.	Soil.
Electricity.	Vegetation.
Atmospheric pressure.	Water supply.
Atmospheric humidity.	Wind shelter and exposure.
Atmospheric purity.	Aspect.

Other Factors sometimes requiring Elimination.

Race.
Closeness of intermarriage.
Sex.
Age.
Occupation.
Density of population.
Poverty.

Sanitation.
Preventive measures against disease.
Prevalence of other diseases connected in any way with the disease in question.
Progressive change in prevalence with the lapse of time.

Needless to say, it is not my purpose to weary you with a systematic outline of knowledge so familiar to you. I shall only recall what is necessary to illustrate my point, the immense importance of the subject, and if I dwell on this fact or on that at greater length, it is only because it bears on an illustration which I shall presently give of what I conceive to be a fundamental principle.

Influences on Healthy Human Beings.

Considering first their influences upon healthy human beings, the most powerful of all climatic factors seems to be *temperature*. Taken in conjunction with atmospheric humidity, it was suggested by the late Dr. W. R. Huggard, of Davos, as the most practical means of classifying climates, and although it may be questioned whether the time is yet ripe for any sort of classification to be satisfactorily laid down, whatever grouping may be ultimately adopted, temperature will almost certainly have to be one main means of climatic division. Ranke has made the pregnant observation that there seems to be an optimum temperature for human beings, which, he says, necessitates the least amount of metabolism compatible with healthy active life. He has placed this optimum between 59° and 68° F., within, in fact, about the limits of temperature which experience has shown us to be best for a pneumonia patient. In hot climates, where least metabolism is required, less food is consumed and there is a disinclination to exertion. The abdominal organs are hyperæmic, the skin acts more and the kidneys less than in temperate regions, and danger attends conditions which involve considerable heat production, such as fevers, physical exertion, and excess in eating. In cold climates, on the other hand, more food is requisite to maintain a healthy activity, but active exercise is commonly taken, and the skin acts less, the kidneys more.

Atmospheric humidity claims special attention from its important relations to temperature. The humidity reduces the tropical heat, but increases its oppressiveness, and people in hot damp climates become lethargic and relaxed. The effects of cold are also greatly modified by humidity; whereas in dry cold the removal of heat from the body is determined by the bodily needs, in damp cold there is a leakage of warmth which is difficult to wholly prevent. Clothes do not entirely control it, and wind, if it exists, considerably increases it. Humidity also acts importantly in lessening the intensity of light.

Atmospheric pressure has received a great deal of attention, chiefly because so many of those who have interested themselves in the effects of altitude have assumed that its influence is chiefly due to this factor.

If, however, we set aside special effects, such as mountain sickness (the outcome of a diminished intake of oxygen) and an enlargement of the thorax of a compensatory sort, the most interesting indisputable result of diminished atmospheric pressure seems to be compensatory increase of the colouring matter of both plants and animals—in plants of the chlorophyll, in animals of the hæmoglobin and red corpuscles.

Winds have received strangely little attention. In damp cold the leakage of heat from the body becomes much greater in wind. Then, certain winds are remarkably enervating, like the Föhn. East wind in Europe is detrimental to many persons, although we have no satisfactory knowledge of why this is so. East winds in these countries seem to have less ozone in them than south-westers, but what effect this difference produces we do not know.

Light increases colour and well-being, yet its precise action on human beings has received, I think, very little attention. *Of electricity* in its natural conditions we know practically nothing as a climatic factor. Yet recent experiments, in which it has been artificially used to stimulate plant and animal growth, suggest that electrical conditions may have powerful effects on climate. Of the influences on healthy men of *rainfall*, *soil*, *vegetation*, *wind shelter* and *wind exposure*, we know practically nothing.

Thus so far as what may be called "physiological climatology" is concerned, we know enough to indicate the importance of knowing more; yet we are still only on the threshold of the subject.

Influence on Parasites and their Non-human Hosts.

We have learnt for certain that the effect of climate on some of the parasites of man and on their non-human hosts is profound. The study of tropical diseases has made this plain. Certain disorders are confined to certain zones of *temperature*. Thus, whatever may be found to be the organism of yellow fever, we know that it does not flourish in temperate climates. The mosquito that carries it and the mosquito host of malaria become rare also at certain altitudes where the heat is less. Other diseases are modified. Thus, phthisis in the Tropics, whilst usually uncommon outside the towns, runs a more rapid course than in cooler latitudes. The gravity of type is probably due in part to the temperature, the rarity is perhaps a consequence of the intensity of light. We know there are optimum temperatures for organisms, as Ranke says there are for man. *Light*, again, has a profoundly destructive influence upon micro-organisms, especially direct sunlight. Apparently it is the blue, violet, and ultra-violet rays to which it owes this most important power. The comparative rarity of phthisis in the Tropics just referred to, and in some high altitudes as well, may owe not a little to the disinfectant power of light.

Rain is popularly supposed to wash the atmosphere, and, whilst it is raining, it doubtless does so. But it is sometimes forgotten that heavy rain after drought causes unusually active development of organisms in the soil, and that these, when the air dries again, enter it as dust. Long-lasting drought decreases the number and the vitality of organisms in the soil. Of the *action of wind*, *air pressure*, *natural electricity*, and *soil* on pathogenic microbes we are, I believe, without information. Here again, therefore, we have much to learn, and comparatively little has as yet been established.

Influence on Human Disease.

Dealing next with the influence of the factors of climate on human diseases, we enter on a field where remarkable progress has been made. Medical geography has become an imposing branch of knowledge. A great empiric acquaintance with the effect of places on disease is steadily growing up. Of this I need say no more. Similarly, medical history is becoming constantly more considerable and precise. But when we come to the theoretical side of medical climatology we find ourselves considerably worse off. It is not too much to say that the most striking characteristic of our knowledge in this department is its uncertainty.

Phthisis is the complaint upon which by far the most has been written in reference to its relations with climate, both in respect of frequency and course. *Altitude* is an ancient subject of discussion. In Peru its influence has been extolled for time out of mind. In my second lecture I shall deal very fully with the claims of the "altitude theory of immunity," and propose to show, in spite of the labour expended on the subject, that nothing has hitherto been proved. *Latitude*, strange to say, has attracted less attention, though its effects are more certainly important. *Soil* has been repeatedly investigated and, to those who have not read the original monographs of Bowditch and Buchanan, the relation of phthisis to dampness of soil is no longer open to doubt. To those, however, who have carefully sifted the evidence the question is still undecided. *Wind shelter* and *exposure* have engaged my own attention closely for the past fourteen years, but although the results of my work have left no doubt on my own mind, I cannot flatter myself that my convictions are shared by everyone. *Atmospheric pressure* is, with some authors, a synonym for altitude. Yet there are strong reasons for questioning its effect. *Temperature* is probably a notable factor in determining the influence of both altitude and latitude, and its variations may have something to do with the effects of wind. Yet nothing is definitely ascertained. About *rainfall, atmospheric humidity, atmospheric purity, sunlight, local aspect, geographical position, vegetation, water supply*, and *electricity* in relation to phthisis we know nothing positively, although with regard to several of these a good deal is sometimes written which can only be regarded as surmise. In fact, considering the amount of labour hitherto spent on the theoretical climatology of phthisis, the real outcome has been extremely small. A terrible vagueness lies like a blight over the field of labour.

Other Tuberculous Diseases.—Other tuberculous diseases have scarcely been investigated climatologically, except where they have been included in the same figures with phthisis. It is interesting and very curious to observe that the *actual sea-front*, so hurtful in cases of pulmonary tuberculosis (as Walshe, I think, first pointed out, and as Ransome has urged), is highly beneficial in certain localities to so-called surgical tuberculosis. Of this no explanation exists.

Pneumonia.—The frequency and severity of pneumonia at high altitudes have been as much insisted on as the rarity of phthisis. It seems, in fact, to be well established that in many mountainous regions pneumonia becomes commoner and more deadly as altitude increases. Whether this depends on increased exposure to certain winds is a question awaiting investigation. Some very small figures, which I am going to submit to you, suggest that this may be the case. The idea that exposure to cold, dry winds is an important cause of the disease is not new, and interesting instances of coincident prevalence of such winds and pneumonia have been given. The curious converse distribution of phthisis and pneumonia to which I have drawn attention might be due to differing wind exposures. For if phthisis mortality is increased in a given area by south-west winds, and pneumonia mortality by north-east winds, such a converse distribution might be expected. In the United States, according to the census of 1880, the generally converse distribution was most curiously marked. The following example of converse distribution may not be uninteresting to you. I have dealt elsewhere rather elaborately with the rural district of Okehampton, in Devonshire, where I mapped out the distribution of phthisis in the parishes, and the exposure and shelter of the parishes to different winds. The maps of phthisis distribution for the decade of 1890-99 were found to present a most striking correspondence with the maps of exposure and shelter in respect of west and south-west winds. I here present to you two tables showing the relation of pneumonia mortality to the exposure to north-east wind. The first, Table II, deals with all deaths between the ages of 5 and 70 during the years 1890 to 1899, the same as were investigated in respect of phthisis. Cases under 5 and over 70 were excluded as specially likely to introduce error from inclusion of bronchopneumonia and hypostatic pneumonia. The assessment of shelter and exposure was that originally made for the phthisis investigation, and confirmed by my friend, Dr. E. H. Young, Medical Officer of Health of Okehampton, who has kindly supplied me with the present returns. It is seen that whereas in parishes sheltered from north-east wind only 0·35 per 1,000 died from lobar pneumonia, in the parishes exposed to the north-east 0·55 per 1,000 died, and in the parishes partly so exposed

TABLE II. NORTH-EAST WIND AND LOBAR PNEUMONIA: ALL DEATHS AT AGES 5 TO 70, OKEHAMPTON RURAL DISTRICT, 1890-1899.

I.—*Sheltered Parishes (Total Population, 5,061).*

Name of parish	Population, 1891	Deaths
Iddesleigh	377	3
Hatherleigh	1,437	6
Monkokehampton	191	—
North Tawton	1,737	4
Bridestowe	586	3
Bratton Clovelly	487	2
Germansweek	211	—
Honeychurch	35	

Total deaths, 18 = annual death-rate (average) 0·35 per 1,000.

II.—*Exposed Parishes (Total Population, 5,796).*

South Tawton	1,264	11
Spreyton	388	4
Chagford	1,460	7
Gidleigh	129	—
Belstone	181	2
Okehampton (rural)	520	1
Ashbury	69	—
Inwardleigh	519	2
Beaworthy	268	2
North Lew	714	1
Highampton	284	2

Total deaths, 32 = death-rate (average) 0·55 per 1,000.

III.—*Imperfectly Sheltered Parishes (Total Population, 3,528).*

Meeth	203	1
Broadwood Kelly	261	—
Jacobstowe	222	—
Exbourne	355	2
Sampford Courtenay	866	9
Drewsteignton	751	4
Throwleigh	281	1
Sourton	448	1
Bondleigh	141	—

Total deaths, 18 = death rate (average) 0·51 per 1,000.

0·51 per 1,000. In the case of pneumonia, however, even more than in that of phthisis, it is desirable to consider separately the female death-rates, because the women are presumably generally near their own homes, whilst the men may be at work in other parishes, and because the women's occupations are more uniform than the men's. Accordingly,

TABLE III.—NORTH-EAST WIND AND LOBAR PNEUMONIA; FEMALE DEATHS AT AGES 5 TO 70, OKEHAMPTON RURAL DISTRICT, 1890-1899.

I.—*Sheltered Parishes (2,614 Females).*

Name of parish	Female population, 1891	Female deaths. 1890-1899
Iddesleigh	197	1
Hatherleigh	724	4
Monkokehampton	83	—
North Tawton	927	—
Bridestowe	309	2
Bratton Clovelly	255	—
Germansweek	102	—
Honeychurch	17	—

Total deaths, 7 = female death-rate (average) 0·26 per 1,000 per annum.

II.—*Exposed Parishes (2,916 Females).*

South Tawton	633	7
Spreyton	193	3
Chagford	728	2
Gidleigh	57	—
Belstone	89	2
Okehampton	300	—
Ashbury	37	—
Inwardleigh	258	—
Beaworthy	122	2
North Lew	364	1
Highampton	135	1

Total deaths, 18 = female death-rate (average) 0·61 per 1,000.

III.—*Imperfectly Sheltered Parishes (1,743 Females).*

Meeth	91	—
Broadwood Kelly	132	—
Jacobstowe	113	—
Exbourne	167	1
Sampford Courtenay	429	2
Drewsteignton	378	2
Throwleigh	149	1
Sourton	217	—
Bondleigh	67	—

Total deaths, 6 = female death-rate (average) 0·34 per 1,000.

Table III has been drawn up dealing exclusively with female deaths at the same ages, and in this the contrast becomes much more striking. Whereas in parishes sheltered from the north-east only 0·26 per 1,000 died, in the parishes fully exposed to the north-east 0·61 per 1,000 died, and in the parishes partly exposed 0·34 per 1,000. It must, however, be remembered that this result is from a single and rather sparsely populated sanitary area, and that the figures dealt with are very small. It is worth presenting to you only as a suggestion of the desirability of further inquiry and as an interesting corroboration of previous observations made by other methods. This relation of wind and pneumonia is one of which nowadays we hear very little. Yet it surely is one of some practical importance. The winds which appear to have the power of causing the disease seem almost everywhere to be cold and dry. *Temperature*, in fact, seems to enter largely into the causation of pneumonia, low temperature and wide variations appearing to promote the occurrence of the disease.

Bronchitis is often considered to be affected by the same influences as pneumonia. But this apparently is not altogether correct. The distribution of bronchitis in the United States in 1880 was by no means the same as that of pneumonia; also, Sturges stated that at Gibraltar different winds appeared responsible for the two diseases—the east, which is damp, seemed to promote the occurrence of bronchitis; the west, which there is dry, the occurrence of pneumonia. It would appear that whereas dry cold tends to cause pneumonia, damp cold rather tends to cause bronchitis. On the other hand, a warm moist atmosphere has undoubted therapeutic value in the drier varieties of bronchitis. In how many disorders can we so confidently recommend a health resort as we advise Torquay or Falmouth in bronchial irritation?

Cancer.—Turning to other diseases. Of the climatology of cancer we may certainly say we know nothing of importance. Haviland's suggestion that the disease is chiefly prevalent in river valleys which are periodically flooded is one which, in Devonshire at all events, I cannot substantiate. But I admit my endeavours have been very limited. In tropical countries I think the evidence is strong that amongst natives living under usual conditions it is relatively rare.

Heart disease has a climatology well worth looking into. Haviland held that it was most prevalent in places not well flushed by wind. But very little has been done on the subject. Only a few years ago authoritative statements were made in reference to a district well known to me which were the direct reverse of the facts.

Nephritis.—Here again very little has been done, although more information seems at our disposal than in the case of heart disease.

Some other Diseases.—For *asthma* we have a good deal of empiric knowledge, the chief fact being its capriciousness. *Gout and rheumatism, dyspepsia, anæmia, neurasthenia, neuralgia*, and *convalescence from acute* *diseases* have, apart from balneology, each a certain useful climatology of its own, but discordant statements are made in respect of them.

The importance of considerations such as the foregoing may be made still more obvious by also regarding them from the points of view of diagnosis, prognosis, prevention, and treatment.

IMPORTANCE IN DIAGNOSIS.

The question whether a patient has resided in the Tropics is a familiar one to all of us in dealing with a travelled person and an obscure complaint. Similarly, we recognize the value of knowing the haunts of malaria and of hydatid disease. But how often do we think it helpful to consider whether a case comes to us from a neighbourhood where we could easily ascertain that tuberculosis or cancer is exceptionally rife? Yet this inquiry is scarcely less reasonable than the question whether the family history of the patient presents an unusual frequency of tuberculosis or cancer. The undue commonness of cancer or tuberculosis in a family should, most people will agree, make us doubly careful in our search for it in a suspicious case. Equally I would submit that the fact of residence in a district where one or other is exceptionally frequent should lead to unusual care in setting aside its diagnosis. There are districts in England where phthisis is six times more prevalent, and districts where cancer is almost three times more prevalent, than in others.

Again, in estimating the liability of the individual before us to cancer, certainly in making the difficult and essential early diagnosis, when no element of probability should be beneath our notice, has it ever occurred to us to wonder if the so-called cancer age is the same in all parts of the country?

Cancer Ages in North-east Cornwall.

The following figures suggest that it is not. The north-east of Cornwall is a region of unusual longevity, and if senility of tissue is a more or less necessary condition for the commencement of carcinoma it might be reasonably expected that the cancer age, where longevity is relatively greater, might be more advanced than in the large centres of population, where, as I have shown, longevity is so remarkably less. My friend Dr. W. F. Thompson, Medical Officer of Health of Launceston, has furnished me with the returns on which the accompanying tables are based. (*See* Table IV, p. 116.) In the first I have compared the distribution of all cases of cancer in the successive decades from 35 onwards, in Launceston and Camelford Registration Districts, with the age-distribution in England and Wales generally. Clearly, in the Cornish districts the age of greatest frequency comes about a decade later than in England and Wales at large. The figures, however, are small for the Cornish districts, and I have therefore sought confirmation in a separate consideration of the ages in different organs. In their case I have compared the ages not with the country at large, but with those quoted by well-known authorities from great hospitals in crowded towns. In Launceston and Camelford districts it would appear that the ages of chief commonness of cancer of stomach, uterus, and breast are in each case almost twenty years later than those observed in certain great city

TABLE IV.—CANCER AGES IN NORTH-EAST CORNWALL: PERCENTAGES OF CASES DYING AT VARIOUS AGE-PERIODS.

All Organs.

	England and Wales (1891-1900), 221,169 deaths at 35 and over	Launceston and Camelford Registration Districts (1897-1907), 176 deaths
Age 35-44	10	4
,, 45-54 ...	22	12
,, 55-64	29	28
,, 65-74	26	29
,, 75 and over ..	11	25

Stomach.

	Various London Hospitals (Fenwick, 1902), [illegible] cases	Same districts and period, [illegible] cases
Age 30-40	14	—
,, 40-50	29	11
,, 50-60	33	13
,, 60-70 ..	18	34
,, 70-80	1·5	31
,, 80 and over ...	—	11

Uterus.

	Two Manchester Hospitals (Sinclair, 1890), [illegible] cases	Same districts and period, 19 cases
Age under 30	2	—
,, 30-40 . .	26	
,, 40-50	42	16
,, 50-60 ..	26	37
,, 60-70	3	31
,, 70 and over ...	0·7	16

Breast.

	Middlesex Hospital (Sheild, 1898), [illegible] cases	Same districts and period, 21 cases
Age 25-40	14	—
,, 40-50	32	19
,, 50-60 ...	29	19
,, 60 and over ...	25	62

hospitals. These differences are very remarkable, though such small figures can only be taken as suggestive. If their indications are true the probability of cancer in a patient of 40 years of age in such districts as these two in North-east Cornwall is definitely less than in these large centres of population. May not such differences be found to be partly climatic?

IMPORTANCE IN PROGNOSIS.

Turning next to prognosis, it is curious to contrast the stress laid on it by the ancients with our modern avoidance of it. Hippocrates attached as much importance to its correct estimate as our patients do now. We, on the other hand, think we show our wisdom by declining to prognose, and, as so generally is the case, atrophy tends to follow disuse. Amongst other conditions of which we may acquire knowledge is the difference in the expectation of life in some chronic diseases in different parts of this country. *Rheumatic fever*, for instance, is commoner in a certain Midland town than it is near Exeter. Therefore, the prognosis of a young valvular heart case should be better near Exeter than in that town. I have in mind a case of heart disease which got much worse in that place and greatly improved near Exeter, a case which led me to inquire as to the commonness of rheumatic heart disease in the neighbourhood I refer to. Again, *bronchitis* and *emphysema* used to form, I remember, a large proportion of the out-patient cases at University College Hospital, London, when I was a house physician there. On the other hand, I found it rather uncommon at the Exeter Dispensary and in my wards at the Royal Devon and Exeter Hospital. The prognosis of emphysema should therefore be presumably better in Exeter than in London. Similarly, if the pulmonic circulation is less liable in Exeter to the accident of bronchitis, the prognosis of *mitral disease of the heart* should also be better in Exeter than in London. I believe it to be so, and to this point I shall presently return.

But the local differences are probably much wider than these few examples indicate. I was greatly struck, a few years ago, by a statement published by a justly celebrated physician as to the prognosis of aortic regurgitation. His experience was drawn from a densely populated area, and he had never seen a case of aortic regurgitation which survived the discovery of the lesion for more than fifteen years. Yet much longer survivals are common enough in Devonshire. For instance, one young woman, with a well-marked aortic regurgitant murmur, which I heard twenty years ago, is still going about almost as well as ever.

although her heart is larger and the murmur louder and more widely audible. Had the statement I refer to come from a less reliable source I should probably have disregarded it. But, coming whence it did, it set me thinking. I knew that the longevity of our Devon country districts is remarkable, and it seemed to me worth inquiring what differences in general longevity existed between different parts of England and Wales. Accordingly, I calculated, for every registration district in England and Wales, and for the decade 1891-1900, the percentage of the total deaths, which were formed by those occurring at 75 and over, and found the most extraordinary disparities. Whereas in many districts 25 per cent. of the deaths occurred at 75 and over, in many others only 7 per cent. and less occurred at those ages. In one Yorkshire rural district the percentage was actually 29·4, whilst in one great industrial centre it was as low as 2·2. I tried to discover how far a difference in infantile death-rate would explain the disparity, and found it only went a very little way. It seemed almost impossible to explain away the differences by the movement of younger lives to the great centres of population from the rural districts. Both of these factors helped undoubtedly to account for the disparities, but to no very great extent. There seems no doubt that a very marked difference exists, in expectation of life generally, between the great towns and the country districts. If so, surely this must affect the prognosis of almost all chronic disease, and as most of our efforts at prognosis, inadequate as they sometimes have been, emanate from our crowded centres, there surely is need for extensive inquiry into the prognosis of these complaints in our rural areas, where I am sure the outlook is different. Moreover, if density of population has this effect, is it not possible that by comparing rural populations with each other we may come to discover climatic factors also which tend to modify longevity? This seems to me an investigation which promises to repay the trouble it would entail.

IMPORTANCE IN PREVENTION.

If it be true that a certain disease is specially rare in a certain place, and if good reason can be shown that this rarity is not merely fortuitous, may we not hold that a patient prone to that disease will, by residing in that place, have a specially good hope of avoiding the disease?

Such a consideration applies to tuberculosis, and one chief reason why I have devoted so much time in endeavouring to establish the value of shelter from rain-bearing wind in lessening the frequency of pulmonary tuberculosis is because I see, in places so sheltered, the most suitable place of residence for those in whom tubercle has become quiescent, or for those who belong to families whose proclivity to tuberculosis is pronounced. I feel sure that there are districts in England which patients with phthisis would be wise to avoid and where sanatoria ought not to be erected.

Similarly, I think that there are districts where old people with strong cancerous family history should not settle, and districts where those who have had repeated attacks of pneumonia run some risk in residing. One cannot be dogmatic yet on these two latter points, but would it not be well if one could?

IMPORTANCE IN TREATMENT.

Once upon a time the medical faculty of this country laid much stress on climate in the cure of consumption. Now the fashion has changed. For (softly be it spoken) there is a fashion in therapeutics not so very far removed from a fashion in frocks! And there is a type of mind which accounts for much of this fashion. It may be described as the uni-dimensional mind, a type of intelligence which only discerns one cause for a single effect. Thus, do open-air methods of treatment hold out new hopes? Then climate and drugs are to be discarded! Is tuberculin found to be valuable? Then sanatoria are to be decried! To the misapprehension induced by this sort of mind has climate, I believe, succumbed in the therapeutics of phthisis. What right have we at present to believe that the methods of open air, graduated exercise, and tuberculin administration cannot be aided by judiciously selected climates? It may be that they cannot. But it is wholly unjustifiable to assume. In his Harveian oration, only two years ago, the late Dr. Theodore Williams, whose practical comparison of climates remains as a model for us all, stated that the results of the Swiss high altitude sanatoria were twice as good as those obtained at ordinary levels. More recently, on the other hand, sanatoria results have been reported almost at sea-level as good as those in the Alps. Here is a divergence of opinion surely badly in need of being cleared up. The comparison of results from sanatoria is a peculiarly difficult one. So much depends on the type of case admitted, the class, the duration of stay, and, even more, on the mode of arriving at the diagnosis and the particular bias in classifying results. If, as in one recent instance, cases are discussed as phthisis in which bacilli could not be discovered, surely the door is open to errors of the gravest kind. If comparisons are made between different sanatoria, surely only cases should be taken into account in which bacilli have been demonstrated, and these should be grouped on some uniform plan according to stage and type. Again, it has perhaps been forgotten that no European resort nowadays claims such success as Dr. Fuentes, quoted by Archibald Smith, assigned in 1858 to Jauja, in the Peruvian Andes. 1858 is no doubt a very long while ago, and since then Koch's discovery of the bacillus of tubercle has drawn a barrier of demarcation across the history of phthisis investigation, so that all antecedent statements require confirmation. Yet it is worth recalling that in the interesting and valuable series of cases of high altitude treatment of phthisis published in 1869 by Sir Hermann Weber, in a paper read before the Royal Medical and Chirurgical Society, the most successful were those which were sent to the Andes. That high altitude has a value in other diseases, such as certain cases of asthma, of Graves's disease, and of malaria, is admitted: why not in phthisis?

If anyone is desirous of testing anew the value of the Peruvian Andes, the present time would appear to be propitious. The terrible Isthmus of Panama has been shorn of its malaria and "Yellow Jack." Good steamers carry the traveller from England to Callao in twenty-eight or thirty days, and the Oroya Railway traverses in forty-eight hours huge passes which formerly had to be painfully crossed on mule-back. Whether Spanish food and housing have progressed in the Andes, as they have in parts of modern Spain, I do not know. If so, not so very much has to be complained of. But it is unlikely, and those who go to Andean mountain towns will probably have to be prepared for roughing it in the way both of quarters and cuisine.

In short, taking into account all the foregoing, there can be no doubt of the immense importance of medical climatology however we regard it, whether from the standpoint of diagnosis, prognosis, prevention, or treatment. We already possess valuable information in all its various branches; if I have barely mentioned medical geography and history, it is because in those the importance of our possessions is unquestionable. As I have said, it is when we come to the theory of the subject, the scaffolding on which we depend so much for the further advancement of our building of knowledge, that we find our footing so seriously insecure.

THE PRESENT NEGLECT OF MEDICAL CLIMATOLOGY.

But if the importance of medical climatology is great, great also is the present general neglect of it. That more and more valuable information is continually becoming available only seems to interest the few; for the many, the references in the text-books are shrinking, the student hears less and less about the subject as time goes on, and the busy practitioner, so trained, naturally does not trouble himself very greatly in regard to it. As Sir William Whitla expresses it, in his chapter on phthisis in his recent work on the "Practice of Medicine": "The view is gradually gaining ground that there is no special curative element in any special climatic conditions" (p. 1353); and (p. 1297): "Climate on the whole must be said to play a minor rôle in its causation."

What is the consequence? The late Dr. Solly, of Colorado Springs, put it with such clearness in 1897 that I cannot do better than quote his words:—

> If we consider (he wrote) how great a sacrifice of time, money, inclination, and affection is involved when an invalid, under direction of a physician, leaves his home and journeys into another and perhaps a far country, we marvel at the small amount of thought and study that is bestowed by the majority of physicians upon the science of medical climatology: for without a fair knowledge and appreciation of this no rational selection of climate can be made.
>
> The deficiency begins (he continues) with the medical schools, which should teach at least the broad principles of climatology and the outlines of climatic therapeutics. What would be thought to-day of the physician who diagnosed and prescribed for a disease of some organ of whose structure and physiology he was ignorant, or of the surgeon who proceeded to operate on parts the anatomy of which he had not studied? Why, then, should a physician presume, as so many do, to prescribe a climate without having acquainted himself with the meteorological facts and climatic data, and with their meaning and significance?

It is sixteen years since these words were written. Yet they apply now just as much as they did then. There is no department of medicine in which so much ignorance prevails or in which such culpable carelessness is tolerated. If we accept the definition I have given of climatology, the seriousness of the existing point of view at once becomes manifest. Neglect on so large a scale must have more than trivial consequences, and it is not surprising to find that the consequences are common and disastrous. This is all the more regrettable at the present time, when the growth of the ancillary sciences, especially of geography and meteorology, the increasing number of stations at which weather observations are recorded, the publication of works of reference, and the multiplication of admirable maps, orographical, geological, and meteorological, are putting at our disposal invaluable means and methods of investigation.

SOME REMEDIES FOR THIS NEGLECT.

For such a state of affairs a remedy will have to be found. In what direction shall we turn with most probability of discovering it? As in physical disease diagnosis must precede treatment, so here our first inquiry must be, "What are the reasons for this neglect?" I believe that they will be found in climatology itself. Solly refers to one of the causes:—

> On turning to the mass of literature available upon climatological subjects, we find it largely composed of empirical and biased accounts of various health resorts and that these reports differ little in their statements of the advantages to be derived. Commonly each claims for its own resort the ability to cure all diseases, and the only invalids warned against coming are those in whom disease is far advanced. The facts given are few, and logical deductions from them are rare. In despair of making a choice from such sources, the physician is apt to take the casual opinion of patients or of other laymen who have visited certain resorts and to select the climate accordingly. He cannot, however, form a correct judgment from such information unless he has previously grounded himself in the fundamental principles of climatology and studied the recorded facts. In this desert of rubbish there are, nevertheless, bright oases of truth and reason, such as are to be found in the writings of Weber, Hirsch, Jourdanet, Lombard, Vivenot, Rohden, Copland, Davidson, Denison, Yeo, the Williamsons (father and son), and others.

Here in the department of medical geography there is clearly room for reform. And there is every indication that workers are moving towards it. In recently dealing with one small section of Dr. Neville Wood's valuable volume on the "Health Resorts of the British Islands," I was much impressed by the sincere desire evinced throughout the health resorts of the south-west of England to present to the medical public a faithful picture of their merits and demerits. Too sanguine statements of my own, from limited central experience, were ruthlessly cut down from closer knowledge by my local correspondents. Rarely did it seem to me that even local enthusiasm has stepped ahead of certainty. The method pursued in that work of combining a central observer with local reporters seems to me one which may be widely extended with advantage. Moreover, a centrally placed writer dealing with very large numbers of patients spread over many health resorts, as was the case with the late Dr. Theodore Williams, can furnish invaluable dispassionate comparisons of results obtained in one place with those gained in another. Williams's careful observations upon selected cases disposed of the claims of Pau and went far to establish the value of Davos.

Of such summaries we have far too few. It was Sir Hermann Weber who, by this sort of comparison, drew widespread attention in this country to high altitudes, and particularly to the Peruvian. Needless to say, the utmost care must be taken to contrast only cases which are actually comparable, and not to draw conclusions from numbers which are inadequate. Moreover, the summaries must, of course, be unbiased. As I have already indicated, our theories of climatology are far too immature to allow them to colour our judgment of the facts. For years to come we must be satisfied to patiently record observations, however unexpected, however inexplicable, some of us may consider them to be.

But more than this is necessary. It is beginning to be suggested that climatology should be specially taught, that there should be special chairs of climatology in our medical schools. That must ultimately be so, and although the time seems scarcely ripe for a separate chair of climatology, a combined chair of what Dr. Fortescue Fox has well called hydrology and of climatology should undoubtedly be established somewhere in this country. Not only is the time ripe for it, but England has already fallen behind other countries in this respect. This is especially unfortunate, since the materials for climatic investigation in England are second to none in richness and accuracy. Such a chair would go far to stimulate much-needed investigation, and it is to be hoped that its establishment will not be long delayed.

But we must learn as well as lecture. And the question arises, in what way? Shall I be pardoned if I insist upon a principle about which I have recently written? It seems to me to go deep towards the foundation of a sound science, not perhaps affording a sufficient remedy for our uncertainties, but possessing merits which cannot be overlooked. I allude to what I have called "the principle of approximate isolation of influences." Hitherto it has only been imperfectly recognized, if indeed it can be said to have been definitely recognized at all. Yet without it I can see no escape from the theoretical chaos which confronts us. By such approximate isolation I mean not merely the happy-go-lucky elimination of this or that influence in the investigation of a third, but the systematic enumeration of all the known influences which seem to affect the field of inquiry besides the influence in question, and their successive elimination from the problem in one way or another, so far as that is possible. The process naturally is neither easy nor short.

Let us ask ourselves what progress would have been possible in bacteriology if investigators had continued to work with the mixed broth cultures of Pasteur? What precision of knowledge could we have attained as to the life-history of the majority of micro-organisms? Modern bacteriology may be said to date from Koch's discovery of isolation methods. Similarly (though unhappily no such absolute isolation is here possible), if climatology is to take its true place in medicine, some sort of isolation of influences will have to be attempted.

The older literature of the influence on phthisis prevalence of altitude or of soil is fairly comparable to the mixed broth culture of the micro-organisms. In neither was a serious systematic attempt made to eliminate other known influences. Müller's work on altitude and phthisis in Switzerland, which I shall discuss in detail in my next lecture, goes furthest in this direction, but not far enough. He eliminated the influence of occupation by considering separately agricultural, industrial, and "mixed" populations, and got rid of the effect on the figures of imported cases of the disease by deducting the percentage so imported in each locality. But although he gave interesting notes on soil and social conditions other than occupational, he made no attempt to eliminate their effect, nor do his notes suffice for us to do so.

Buchanan, in his Reports to the Privy Council on the relations of phthisis to dampness of soil, eliminated no other influence. Density of population, difference of occupation, and other social factors, all obviously liable to affect his figures, were allowed to confuse his problem; and his method of manipulating his figures by guesswork, to correct for imported cases and for the presence of institutions, cannot be too strongly condemned.

In the second lecture I propose to furnish illustrations—first, of the systematic application, and second, of the neglect, of this principle. For its systematic application, I am sorry I can only refer to my own work, and I shall do so very briefly, as it has already been sufficiently enlarged on elsewhere. The results of the neglect of the principle I shall illustrate by a revision, in the light of it, of the so-called "altitude theory of phthisis immunity," and show that, so reviewed, the existing evidence in favour of that theory absolutely crumbles to pieces.

Balneological and Climatological Section.

May 21, 1913.

Dr. F. Parkes Weber in the Chair.

THE SAMUEL HYDE MEMORIAL LECTURES.

The Place of Climatology in Medicine.

By William Gordon, M.D.

LECTURE II.—THE PRINCIPLE OF APPROXIMATE ISOLATION OF INFLUENCES.

Illustration I.—The Influence of Rain-bearing Wind on the Prevalence of Phthisis.

Dr. Parkes Weber and Gentlemen,—My first illustration of the value of the "principle of approximate isolation of influences" must be drawn from my own work. I shall deal with it very briefly, as it has been so fully discussed elsewhere (1). I cannot exaggerate the feeling of security one gains from it in climatic investigation. In addition to the factors of climate, which I have already enumerated, there are a number of other influences which have to be eliminated. These are set forth in the second half of Table I. They were dealt with as follows:—

Race has undoubtedly a very strong influence on phthisis, and to eliminate it only populations of the same race were compared with one another. With regard to *sex*, female death-rates for several reasons afford a better criterion than male, and only female death-rates were used when possible. *Occupation* has a marked effect, and therefore localities where special industries were almost universal were excluded from consideration. To eliminate *density of population* town and country were not compared with one another. To get rid of the effect of *preventive measures against the disease*, the figures referring to periods previous to the introduction of these were used when possible where preventive measures had been adopted. *The progressive change in prevalence with lapse of time* was prevented from introducing error by the comparison of only contemporary records. The other influences were shown to be so much less powerful than that of rainy wind that, where they could not be separately investigated, they might rightly be regarded as most unlikely to introduce error.

ILLUSTRATION II.—THE HIGH ALTITUDE THEORY OF PHTHISIS IMMUNITY.

To illustrate the consequences of the neglect of this principle I have taken the work hitherto done on the "high altitude theory of phthisis immunity."

There is no more fascinating chapter in medicine than that which deals with altitude and phthisis. Its setting is superb. The most magnificent regions of the earth, some of the most famous, form the scenery. The names on its pages are amongst our most honoured. It ranges over the most diverse climates and raises the most fundamental questions of climatology.

Originating in the vast valleys of the Andes, where from time immemorial consumptives from the coast have been restored to health, and where the natives were believed to be practically immune, the theory that altitude was in some way antagonistic to the disease was first announced to Europe by Dr. Archibald Smith [2] in his writings from 1840 onwards. His statements were subsequently confirmed by Tschudi [3] and by Guilbert [4], whilst evidence of similar rarity at almost equal altitudes was furnished by Jourdanet [5], from the high plateau of Mexico. German observers took up the idea at home, and endeavoured to show, at much lower elevations, a diminution in the prevalence of phthisis with increasing height. Brockmann [6] and Fuchs [7] in the Hartz, Virchow [8] in the Spessart, Virchow and Brehmer [9] in the Silesian valleys, von Corval [10] in Baden, and Merbach [11] in Saxony, are all said by Hirsch [12] to have made this diminution probable. In Switzerland, dealing with altitudes more comparable to those of the Andes and over a much larger area, Lombard [13], Muller [14], and others published statistics of the greatest interest.

Sir Hermann Weber [15] was one of the first to test the value of both Alpine and Andean stations, and the remarkable success which he reported, especially from the Andes, drew general attention in this country. The late Dr. Theodore Williams [16] furnished striking evidence of the value of the Swiss stations in the treatment of early cases, but was one of the first to throw doubt on the truth of the theory. This doubt has been increased by the discovery that in cities like Quito, formerly said to be almost immune, consumption is common, and the recent results of sanatorium treatment at low levels have (not quite logically) diminished the belief that altitude has anything to do with either the prevalence or the course of the disease. The general failure of the Himalayas as a health resort in consumption, even at heights of over 6,000 ft., contributes to the increasing doubt, and it might almost be said at the present time that the high altitude theory is moribund. But it is by no means dead, and as lately as 1906 the late Dr. Huggard [17], of Davos, in his "Handbook of Climatic Treatment," quoted a table published in 1895 by the Sanitary Department of the Grisons. The summary of this table (Table IX) seems to show very clearly a decrease of phthisis incidence with increasing height. To it, and to the arguments adduced in the extensive literature which preceded it, no satisfactory answer has hitherto been given. It is such an answer that I am now endeavouring to provide. *I propose to show that no valid evidence exists that altitude, per se, has any influence upon the prevalence of phthisis.*

But it is necessary at the outset to lay down certain limitations. First, no one now claims that any altitude confers an absolute immunity from phthisis; that, therefore, needs no discussion. Secondly, the earlier statements as to the rarity of the disease in crowded cities at great heights have been shown to hold true no longer—if, indeed, they ever were correct. They will, therefore, not be now considered, except very briefly as follows: At Mexico, Jourdanet [18] never claimed more than a relative rarity as compared with the coast and placed the death-rate at 2·1 per 1,000, and Le Roy de Méricourt [19] gave it at 4·7. In Bogota, according to Restrepo [20], and at Quito, according to Jacoby [21], phthisis enters largely into the mortality lists. This is remarkable, seeing that in 1878 Gayraud and Domec [22], writing of Quito, said "our personal experience allows us to affirm that phthisis is so rare that we may say that it does not exist there, at least as a malady originating locally. . . . The fact is then indisputable for us: one does not become phthisical at Quito" (Hirsch). Restrepo gives details which perhaps throw light on this remarkable statement. At Bogota about 50 per cent. of the cattle coming from the lower country are tubercular and the diseased parts are eaten by the very poor. It is amongst these very poor that the disease is common, and the form of the disease is peculiar. The intestines are always affected and, in the lungs, the apices are not oftener attacked than other parts. There is little tendency to the formation of cavities or of fibroid tissue, and the symptoms differ strikingly from those of phthisis as we know it. With a very chronic course, a usually subnormal temperature, no night sweats, very rarely cough, sputum, or hæmoptysis, the picture presented is one of intractable diarrhœa, digestive troubles, and emaciation. Such a disorder would scarcely have suggested pulmonary consumption to observers who in 1878 knew nothing of the tubercle bacillus. But their mistake must render us cautious in accepting the earlier views. Restrepo's account, however, by no means disproves a real rarity of ordinary phthisis from inhalation of the human form of the bacillus.

Lastly, a sharp distinction must be drawn here, as in all investigations of the climatic relations of tuberculosis, between an influence on prevalence and an influence on course. I am only here discussing the influence on prevalence. These separate influences have, I think, been too much intermingled in discussing the altitude theory—as the brief outline of its vicissitudes has indicated. It might perhaps be expected that places where phthisis is exceptionally rare would be generally beneficial to imported cases; yet this is by no means true. In tropical countries, away from the crowded towns, consumption, though rare, is usually rapid in its course; and in the Peruvian sierra Archibald Smith has carefully indicated localities where, though phthisis was practically non-existent amongst the natives, the developed disease did badly. The converse proposition, however, that places where phthisis is exceptionally common might be expected to prove unfavourable to imported cases, seems to have more to be said for it. I know of no place where consumption is rife yet where imported cases do well. In dealing, therefore, with altitudes, such as the Himalayas, where it is particularly difficult to obtain direct evidence regarding the prevalence of the disease, it may be held permissible, in absence of direct information (always remembering the uncertainty of such reasoning), to make use of authoritative statements as to their effect upon its course. Otherwise I shall confine all I have to say strictly to the influence on prevalence.

I shall deal first with the Andes, not merely because the theory arose there, but because the climate is in several respects unique;

secondly, with the Swiss Alps, where the most important work has been done on the subject; thirdly, with the German uplands and other European elevations; then with the Himalayas and other Indian altitudes; and, finally, with the tablelands of South America, of South Africa, and Persia.

I must again point out that Koch's discovery of the tubercle bacillus in 1882 has made a landmark in the history. Before 1882 diagnosis was definitely more doubtful than it has since become, and instances will be noticed in which serious misstatements have consequently arisen—in fact, one such has probably been already mentioned, since it is unlikely that the frequency of pulmonary tuberculosis now recognized amongst the poor of Quito is of only recent development. Some of the statements date back to the first half of last century, and it might be held that such antiquated opinion should be altogether neglected. But, as it forms part of the existing argument, and as it may be justly urged that phthisis has so long been a clinically well recognized disease, not usually mistakable at its actual termination, this can scarcely be done. In all cases, however, I shall qualify my information by adding its date.

The Andes.

There can be no doubt that the Peruvian Andes, in the latitude of Lima, present a striking contrast to the adjacent coast in relation to phthisis prevalence. Dr. Campodonico says in a valuable letter written to me in 1905: "In Lima the death-rate from tuberculosis is very high, as is seen from the enclosed statistics" (it was 7·7 per 1,000 in 1904); but "it is perfectly true that the inland plateaux of Peru are almost absolutely free from phthisis."

It must also be borne in mind (it is proper to allude to this here) that, correctly or not, the claims made for the valley of Jauja as a sanatorium for phthisis considerably exceed any claims which have been advanced elsewhere. Whereas in Lima the disease runs a rapidly fatal course, at Jauja, at an elevation of about 10,000 ft., Dr. Fuentes (23) stated in 1858 that over 79 per cent. of the patients recovered, and, of the recoveries quoted by Sir Hermann Weber (24) in the paper I have referred to, some of the most remarkable took place in these mountains. We have thus definitely in this part of Peru a remarkable contrast between adjacent lowland and highland in respect of the relation to phthisis. It remains to be decided upon what peculiarity this contrast depends.

In the first place, it must be observed that the climatic contrast is equally pronounced. Lima is only 565 ft. above sea-level in a climate by no means usual in the Tropics, sheltered by the Andes from the south-east trades and very damp but almost rainless, and chilled by the great Antarctic current which, running northwards along the Pacific coast and carrying, from Southern Chili to some distance north of the Equator, a vast body of cold water, cools the atmosphere of both sea and land and frequently covers both with a roof of cloud.

For more than half the year—says Mr. Bryce [25]—Lima has a peculiar climate. It is never cold enough to have a fire, but usually cold enough to make you wish for one. It never rains, but it is never dry; that is to say, it is not wet enough to make one hold up an umbrella, yet wet enough to soak one's clothes. September was as dark as a London November and as damp as an Edinburgh February, for the fog was of that penetrating and wetting kind which in the case of Scotland they call a "haar." For the other half of the year there is tropical heat. When to this we add that this climate is very enervating, that the inhabitants are remarkable for their effeminacy and delicacy of constitution, the men especially, with their contracted chests, who find shaving and washing the face in cold water sufficient to cause catarrh (!) [26]; that the city is a large one containing over 100,000 inhabitants, of all sorts of races; that the death-rate from all causes in 1840 was about three times that of London or Manchester, and was over 35 per 1,000 in 1903 [27], it need cause no surprise that the tuberculosis-rate is a high one.

On the other hand, the climates and conditions to which Dr. Archibald Smith specially drew attention are entirely different. As it is necessary to be precise, I must quote his own statements as they stand:—

> Certain states of the air of the atmosphere, depending on different degrees of altitude, appear to be either hostile or favourable according to the particular locality in which the patient happens to reside . . . Thus on the coast it is a common disease, but on the intermedial mountains and in the temperate valleys of the interior it is rare [28].

He elsewhere carefully refers to the "deep warm glens of the interior" as the favourable localities [29].

> Huarriaca is in climate very like Obrajillo, on the western slope of the Andes [elsewhere he says that Obrajillo is in a hollow locked in by hills], and

> in one of those recesses in the Andine glens and defiles very productive of maize, &c., &c. Such, indeed, are the marked localities, blessed with a steady, temperate climate and a dry air of about 60° F. in the shade, as well as sunny, cheerful sky throughout the greater part of the year. Such are the localities where phthisis proper, or tubercular disease of the lungs, is only known as an exotic . . . The sky of the dry, intermediary valley . . . is generally throughout the whole year remarkably pure, bright, and serene [30].

Of Jauja he says: "With a sky always bright and sunny and an atmosphere pure and bracing which invites to outdoor exercise and enjoyment" [31]. In confirmation of Smith's statements Campodonico writes of the territory of Junin where Tarma, Jauja, and Huancayo are situated:—

> These towns, protected as they are by the lofty range of the Andes, which deflect the trade winds, can hardly be said to have any regular winds. The climate is very dry, although the rainfall reaches a high point in the months of December, January, and February, when sudden and frequent showers occur, the water dries away rapidly—iron tools are very rarely oxidized [32].

He attributes the rarity of phthisis to several causes; for instance, "dryness of the air, the effectual Andean screen sheltering from humid southern winds, sparseness of population, with a consequent less pollution of the air." It must also be noticed that the towns are small and that the occupations of the people are pastoral and agricultural, "the harvest being home, the rural population rest from their agricultural labour for eight months in the year, which they give up to amusement and feasting."

Dr. Theodore Williams carefully described the valley of Jauja [33]:—

> The valley of Jauja is a plateau forty-four miles long and seventeen wide, separating the two ranges of Andes . . . entirely drained towards the east by the Mantaro. . . . The soil is alluvial with a calcareous substratum. The year is made up of two seasons: the dry season, which is the coldest, extending from March to August, in which last month frost occurs, and the rainy (called the winter), which is the hottest, extending from September to February. Thunderstorms occur in January and February, and wind is most felt in July and August (i.e., one observes it in the dry season) . . . . The climate does not admit of great extremes, and the thermometer falls sometimes to 28° F., seldom rising much above 57° F. (in shade). The sun's rays are powerful, and so great in their direct influence that in the full sunshine the temperature may be 122° F. and in the shade 50° F. The atmosphere is very clear, partly on account of rapid evaporation, and partly because of the rapid drainage by the

River Mantaro. Iron and steel are stated never to rust, and such is the transparency of the atmosphere that stars are seen by day. The prevailing winds are south-west in the evening and the north-east in the morning, and occasionally they blow sufficiently to cause a tempest.

It should be observed that at the silver mines of Pasco, over 14,000 ft. above sea-level, "with a wet season like a Scotch winter, and though in the dry season it shines brightly and warmly at noon, yet in the shade the air is chillingly cold and the nights always frosty," although imported cases did not improve, Smith only once in a year's residence met with a case of hæmoptysis.

In a most interesting account of Bolivia kindly sent me six weeks ago by Dr. Ramsay Smith, British Consul at Orura (to which I trust to do fuller justice in a future paper), he emphasises the extreme rarity of any sort of tuberculosis amongst those born and bred in his district, which lies about 12,000 ft. above sea-level.

It should be noted that no one has tried to show, so far as I am aware, any *increasing* rarity with increasing altitude in the Andes. The optimum height for treatment is stated to be between 5,000 and 10,000 ft.

Now race cannot account for the difference. For instance, Campodonico tells me "that inlanders may easily contract tuberculosis when they leave the tableland of the Andes and come to the coast; in fact, on the coast of Peru the largest tribute to mortality from tuberculosis is paid by them." So also the chest enlargement on which stress has elsewhere been laid affords here no adequate explanation, since these inlanders exhibit it. Differences in occupation and in density of population may go far of themselves to account for the difference of prevalence. For in the sheltered tableland of Kashmir Dr. Arthur Neve tells me that whereas in the insanitary, overcrowded city of Srinagar phthisis "is not rare," "it is almost unknown" outside it. In Kashmir no great difference of altitude enters into the problem, yet the contrast between town and country is apparently the same.

When we add together differences in climate which produce an enervated, delicate, and narrow-chested population in Lima, and a healthy broad-chested people in the Sierra; between a very damp and a very dry climate; between a sky overcast for half the year and the nearly continuous brilliancy of a tropical sun, through a most diaphanous atmosphere; between conditions which are described by Archibald Smith as depressing to appetite and hindering to digestion, and conditions which he found to be exactly the reverse—differences, moreover, whose effects we have been afforded no means of separately estimating and eliminating; it is clear that we cannot assert that the lesser prevalence on the heights is in any direct way determined by mere altitude.

The Andean valleys, indeed, present an almost unique climate—well within the Tropics, only about 12 from the Equator, yet singularly equable and temperate, with a remarkably dry, still atmosphere, with the disinfecting presence of great intensity of light, yet without the excessive shade temperature which incubates bacilli; and when we add to these conditions sparseness of population and pastoral and agricultural occupations, with no overwork and with healthful outdoor life, we shall find little difficulty in explaining even a unique rarity of phthisis within them without invoking altitude or its equivalent in diminished barometric pressure to account for it.

In short, the Andean valleys described by Archibald Smith furnish no valid evidence in favour of the theory he propounded. As regards the therapeutic effect of these altitudes, the mere reduction of temperature from that of the Tropics for part of the year on the coast to that of temperate climates in the Sierra must be an agent of immense importance.

The Swiss Alps.

The work done in Switzerland was the most important in Europe. The altitudes dealt with were great, the area extensive, the material carefully scrutinized, and the methods used more thorough than elsewhere. I shall therefore discuss it in some detail.

Dr. Lombard, of Geneva, in his charmingly lucid "Climats de Montagnes," stated that at the higher inhabited altitudes in the Alps phthisis was comparatively rare. "I have shown," he wrote in 1873, "that the rarity of phthisis on the heights is an indisputable fact," and later on, "One may take it that phthisis is almost unknown above 2,000 metres." But he insisted on a very interesting and important point which Hirsch has not noticed—viz., that at intermediate heights a belt of increased phthisis normally exists, "phthisis zone," (34) above and below which the disease is less prevalent, a zone placed, he thought, approximately between 1,300 or 1,600 ft. and about 3,000 or 4,000 ft. of altitude. It was also, he found, a zone of greater frequency of "scrofula." This belt roughly corresponded with another belt of increased humidity, greater rainfall, damper soil, more mist and cloud, and more frequent thunderstorms, which reached, he said, from about

1,600 or 2,000 ft. above sea-level to about 3,000 or 5,000 ft. He also described a belt of forest and meadow in which herbaceous and arborescent plants took on more luxuriant growth, stretching from about 1,600 ft. to about 6,000 ft.

Now, this point of a "phthisis zone," if correct, is of the greatest moment, as it at once casts doubt on all the work which has been done at levels which do not reach its higher limit. Thus Lombard goes on to say:—

> But is this so (the lessening of phthisis prevalence) for all mountain regions? We do not think so, and we shall see that, if a belt exists where phthisis is nearly completely unknown, there are other regions where the disease acquires a degree of frequency much greater than that observed in the surrounding plains.

A large number of papers, he tells us, exist to show this. Dr. Locher-Balber found tubercular disease twice as common in the mountain districts of Canton Zürich as down by the lake. He makes the interesting statement that a Dr. Mansford published in 1818 a work intended to show that in England phthisis patients were more numerous in proportion as their dwellings were more elevated, a conclusion which my own observation bears out, when the localities examined are exposed to westerly winds. Lombard explains the apparent contradiction by pointing out that the localities quoted were within the belt of increased phthisis mortality. My observations would suggest that the increase of their phthisis death-rate was due to their elevation increasing their rainy wind exposure.

And here it will be well to glance at the climatic influences exerted by mountains and to try and get some rational notion of how mere altitude might act upon the prevalence of phthisis. The feature which presents the greatest regularity with increase of altitude is decrease of barometric pressure, and this has been supposed by some to be the factor which tends to produce a relative phthisis immunity. Roughly speaking, at 6,000 ft. the air pressure is about 25 per cent. less than at sea-level. At that level the effect of the decrease in oxygen pressure becomes noticeable to the newcomer, and a condition which Jourdanet named anoxyhæmia develops. To compensate for this a rapid increase of hæmoglobin and red corpuscles occurs, so that Viault, who investigated it in Peru, found that in two weeks his red corpuscles increased from 5 to 7 million per cubic millimetre, and in another week to 8 million. The amount of iron in the blood rose in the ratio of 4 to 7.

Also the chest measurements increase, so that the natives of these high altitudes have larger chests than those who have lived below. Both of these compensatory changes have been given as explanations of the supposed immunity.

With increasing altitude the intensity of insolation also increases, so that at the summit of Mont Blanc the intensity of solar radiation is 26 per cent. greater than at the level of Paris, and a marked increase in the intensity of the ultra-violet rays exists at great altitudes and an increase in the chemical effect of sunlight. Nevertheless, the air temperature falls and both annual and daily range of temperature considerably diminish, the climate therefore becoming cooler and more equable.

An intermediate belt does exist, such as Lombard described, of greater humidity, rainfall, and cloud. In the Tropics this is at an altitude of between 1,300 and 1,600 metres. In higher latitudes it varies with the season, in winter being lower, in summer higher. The rainfall increases with the increasing altitude up to a certain point, at which it again decreases. It should be borne in mind that the central mountains of Germany and those of England are below the altitude above which the rainfall lessens. In the north-west Himalayas, Hill [35] found the zone of maximum rainfall during the monsoon was about 1,270 metres above sea-level, and if the rainfall on the plains be taken as 1, the amount in this maximum zone was 3·7, but at 3,000 metres only 0·2. Season considerably modifies the position of these zones.

That increased chest capacity does not prevent phthisis we have already learnt from Dr. Campodonico's statement, as well as from general experience of relapses on patients leaving higher levels. The increase in red corpuscles and hæmoglobin might conceivably tend to hinder the development of phthisis; the increased solar radiation, and especially of the ultra-violet rays, would destroy bacilli exposed to them, and at great heights the dryness of the air should be prophylactic. On the other hand, the zone of increased rainfall and cloud would have the contrary effect. The proof, however, here, as always, must depend not on theoretical expectation but on ascertained facts.

In 1863 a Commission was appointed by the Swiss Natural Science Society to investigate the distribution of phthisis in Switzerland, with a view to determining the influence of altitude. The five years 1865 to 1869 were selected as the period of observation. Dr. Muller, of Winterthur, acted as secretary. In his careful and elaborate report

Müller points out that the period of observation chosen was too short. It was, however, the utmost possible, and in many places—indeed, in some entire cantons—it was found unattainable, returns being only procurable for four, three, or even two years. Moreover, the figures, he says, were not free from omissions and errors, though he indicates those which he thinks definitely open to doubt. Great pains were clearly taken to make the best of the materials, and the statistics were drawn from a very wide area and great population. This report, therefore, is entitled to the most attentive consideration. We shall see that it does not prove that phthisis prevalence necessarily diminishes as altitude increases.

Müller eliminates the disturbing influence of occupation, which obviously greatly affected the death-rates, by grouping the localities as "industrial," "agricultural," and "mixed," and by considering each separately. Table V gives the relation of phthisis to altitude in each of these groups. The industrial districts do not seem to prove any relationship, nor do the agricultural districts above the height of 700 metres (about 2,300 ft.). But the "mixed" cantons might be held to suggest a relation (if we assume that Lombard was right in his opinion about an intermediate belt of greater mortality and, therefore, neglect the heavier and increasing death-rates between 700 and 1,300 metres).

TABLE V.—DEATH-RATES FROM PHTHISIS AT SUCCESSIVE ALTITUDES IN SWITZERLAND (MULLER) PER 1,000 PER ANNUM.

Altitude in metres	Altitude in feet	Industrial districts	Mixed districts	Agricultural districts
200-499	656-1,639	2·7	1·85	1·4
500-699	1,640-2,295	3·0	1·55	1·2
700-899	2,296-2,951	1·35	1·7	0·7
900-1,099	2,952-3,608	1·5	1·9	0·7
1,100-1,299	3,609-4,264	2·3	2·3	0·7
1,300-1,499	4,265-4,920	—	1·4	0·6
Over 1,500	4,921-5,905	—	1·3	0·7

But Müller further provides another set of statistics (which Hirsch has overlooked) eliminating a source of error, which in Switzerland, where so many of the inhabitants go to other countries to work and return home when ill, is particularly liable to mislead—viz., the inclusion of cases which originated elsewhere. Table VI embodies these figures. Here, again, the industrial districts give no definite indication, and the agricultural above 700 metres show no signs of decrease, but the "mixed" districts suggest even more strongly than before a decrease with increasing altitude, except for a passing increase between 700 and 1,100 metres. It must, however, be remembered that of the three groups the most indefinite as regards occupation is the mixed, for the amount of industrial occupation, of course, varies from place to place.

There is, however, another powerful influence which has to be taken into account, of which Müller was not aware—viz., the influence of prevalent rain-bearing winds. These are the north-west, west, and south-west for the greater part of Switzerland, with perhaps the south-east for the lower valleys which run down into Italy. Of the westerly winds the chief rainy wind appears, throughout Central Europe, to be the north-west.

To examine the effects of these winds I have constructed from Müller's work Tables VII and VIII, excluding all towns of more than 10,000 inhabitants (in order to lessen error from density of population), all districts where a hospital was said to exist, all where the period of observation had been less than five years, and all where the number of cases really of local origin could not be determined. Here, again, the agricultural group over 700 metres shows no signs of a decrease in phthisis with increase of height, whilst both the "mixed" and industrial groups indicate an *increase* of phthisis, which in the latter is actually progressive over 500 metres. On examining the individual places as regards their exposure and shelter from westerly winds we see at once an easy explanation of a greater death-rate at lower levels where it occurs; below 700 metres the proportion of places exposed is considerably greater than above. It becomes, therefore, clear that the apparent influence of altitude may be readily explained as an indirect effect of wind exposure or shelter. The industrial districts happen to be almost all exposed, and increasing height merely carries them into regions of greater wind and rain; the

TABLE VI.—MÜLLER'S TABLE AFTER OMISSION OF IMPORTED CASES.

Altitude in feet	Industrial districts	Mixed districts	Agricultural districts
656-1,639	1·8	1·4	1·2
1,640-2,295	2·1	1·2	1·1
2,296-2,951	—	1·3	0·6
2,952-3,608	1·3	1·3	0·5
3,609-4,264	2·2	1·1	0·7
4,265-4,920	—	1·0	0·6
4,921-5,905	—	0·8	0·7

TABLE VII.—BEING MÜLLER'S TABLE XVI MODIFIED AS STATED.

X = Exposed to S.W., W., or N.W. winds.
S = Sheltered from all of them.

District	Canton	Population	PHTHISIS OF LOCAL ORIGIN		Exposure or shelter to westerly winds
			Deaths in 5 years	Annual death-rate per 1,000	
		ALTITUDE 200-499 METRES (656-1,639 FT.).			
		Industrial.			
Mendrisio (town) ...	Tessin	2,200	26	2·3	**X** S.W.
Zug (town)	Zug	4,066	38	1·8	**X** W., N.W.
Diessenhofen (town) ..	Thurgau	1,595	12	1·5	**X** ,,
Lenzburg (town)... ...	Aargau	2,297	15	1·3	? **X** W., S.W.
Coldrerio, Salorino .	Tessin	1,018	3	0·5	? **S**
Totals	...	11,176	94	Av. 1·6	
		Mixed.			
Lachen	Schwyz	1,532	18	2·3	**X** N.W.
Sargans, Mels	St. Gallen	3,997	39	1·9	**X** ,,
Cully (town)	Waadt	1,089	8	1·4	**X** S.W.
Anières, &c. .. .	Genf	3,472	24	1·3	**X** ,,
Liestal, Seltisberg ...	Baselland	4,027	25	1·2	**X** N.W.
Lausen	,,	781	5	1·2	**X** ,, ?
Ormalingen, &c.... ...	,,	1,199	6	1·0	**S**
Colombier, &c.	Neuenburg	2,355	12	1·0	? **X** S.W.
Gelterkinden	Baselland	2,198	10	0·9	**S**
Tenniken, &c.	,,	1,164	2	0·3	**S**
Sissach, &c.	,,	3,087	4	0·2	**S** ½ ?
Totals	...	24,901	153	Av. 1·2	
		Agricultural.			
Unterhallau ..	Schaffhausen	2,370	30	2·5	?
Begnins, &c. . ..	Waadt	1,977	18	1·8	**X** S.W.
Erlach Lüscherz, &c. ..	Bern	2,684	23	1·7	**X** ,,
Maisprach, &c.	Baselland	1,213	9	1·4	**S**
Jussy	Genf	873	6	1·3	**X** S.W.
Wintersingen, &c.	Baselland	753	5	1·3	½ **S**
Ins, &c....	Bern	2,835	19	1·3	**X** S.W.
Walchwyl . .	Zug	1,055	6	1·1	**X** N.W.
Basadingen	Thurgau	2,095	10	0·9	**X** W., N.W.
Satigny	Genf	3,546	17	0·9	**X** S.W.
Andelfingen .	Zürich	5,851	19	0·6	?
Risch ...	Zug	960	2	0·4	**S**
Arisdorf, &c. .	Baselland	1,097	2	0·3	**S**
Totals	...	27,309	166	Av. 1·2	

TABLE VII.—BEING MÜLLER'S TABLE XVI MODIFIED AS STATED—(*continued*).

X = Exposed to S.W., W., or N.W. winds.
S = Sheltered from all of them.

District	Canton	Population	PHTHISIS OF LOCAL ORIGIN		Exposure or shelter to westerly winds
			Deaths in 5 years	Annual death-rate per 1,000	
ALTITUDE 500-699 METRES (1,640-2,295 FT.).					
Industrial.					
Unterseen	Bern	1,732	20	2·3	**X** W.
Bauma ...	Zürich	2,939	27	1·8	? **X** N.W.
Totals	...	4,671	47	Av. 2·0	
Mixed.					
Meyringen	Bern	4,739	46	1·9	? **X** W.
Diegten, &c.	Baselland	1,623	13	1·6	?
Laüfelfingen	,,	734	6	1·6	?
Neuheim	Zug	700	5	1·4	**X** W., N.W.
Wenslingen	Baselland	634	4	1·2	**X** N.W.
Hutwyl (town)	Bern	3,254	21	1·2	**X** ,,
Rümlingen, &c.	Baselland	1,387	6	0·8	½ **X**
Zeglingen, &c.	,,	1,245	5	0·8	½ **X**
Totals	...	14,316	106	Av. 1·4	
Agricultural.					
Ringenburg	Bern	1,123	14	2·4	**X** S.W.
Grandvaux	Waadt	1,713	17	1·9	**X** S.W., W.
Bremgarten	Bern	2,125	15	1·4	**X** S.W.
St. Saphorin, &c.	Waadt	2,868	20	1·3	**X** S.W., W.
Leissigen, &c.	Bern	800	5	1·2	**X** N.W.
Kirchlindach	,,	809	5	1·2	**X** S.W.
Valle Capriasca	Tessin	2,967	14	0·9	**S**
Wohlen ...	Bern	3,211	16	0·9	**X** S.W.
Gsteig ...	,,	7,418	33	0·8	½ **X** S.W.
Rapperswyl	,,	1,888	6	0·6	?
Totals	..	24,922	145	Av. 1·1	
ALTITUDE 700-899 METRES (2,296-2,951 FT.).					
No Industrial.					
Mixed.					
Menzingen	Zug	2,248	33	2·9	**X** N.W., W.
Couvet ..	Neuenburg	2,102	17	1·7	?
Oberoegeri	Zug	1,905	6	0·6	**S**
Unteroegeri	,,	2,492	3	0·2	**S**
Totals	..	8,747	59	Av. 1·3	

TABLE VII.—BEING MÜLLER'S TABLE XVI MODIFIED AS STATED—(*continued*).

X = Exposed to S.W., W., or N.W. winds.
S = Sheltered from all of them.

District	Canton	Population	Phthisis of local origin		Exposure or shelter to westerly winds
			Deaths in 5 years	Annual death-rate per 1,000	
ALTITUDE 700-899 METRES (2,296-2,951 FT.)—(*continued*).					
Agricultural.					
Arzier	Waadt	358	4	2·2	**X** S.W.
Gimel, &c.	,,	1,823	12	1·3	?
Pfäffers	St. Gallen	600	4	1·3	**X** N.W.
Trois Torrens	Wallis	1,313	6	0·9	**S**
Dürrenroth	Bern	1,408	6	0·8	**S**
Bassins, &c.	Waadt	912	3	0·6	?
Totals	...	6,414	35	Av. 1·09	
ALTITUDE 900-1,099 METRES (2,952-3,608 FT.).					
Industrial.					
Sternenburg	Zürich	1,038	11	2·1	**X** S.W., W., N.W.
Les Bayards	Neuenburg	958	9	1·8	**X** S.W.
Totals	...	1,996	20	Av. 2·0	
No Mixed.					
Agricultural.					
St. Georges, &c.	Waadt	720	6	1·6	**X** S.W.
Vättis	St. Gallen	350	1	0·5	**S**
Boltigen	Bern	2,010	6	0·5	**S**
Frutigen, &c. ..	,,	5,224	15	0·5	**S** ("? if complete")
Lenk	,,	2,288	5	0·4	**S**
Weisstannen ..	St. Gallen	471	1	0·4	**S**
Ayent. Vex ...	Wallis	1,962	2	0·2	**S**
Totals	...	13,025	36	Av. 0·5	
ALTITUDE 1,100-1,299 METRES (3,609-4,264 FT.).					
Industrial.					
St. Croix ..	Waadt	4,574	50	2·1	?
No Mixed.					
Agricultural.					
Revereulaz, &c.	Wallis	273	7	5·1	? **X** N.W.
Agettes and Hérémence	,,	1,368	1	0·1	**S**
Gsteig, &c.	Bern	1,415	2	0·2	**S** ("? if complete")
Totals ...	.	3,056	10	Av. 0·6	

TABLE VII.—BEING MÜLLER'S TABLE XVI MODIFIED AS STATED—(*continued*).

X = Exposed to S.W., W., or N.W. winds.
S = Sheltered from all of them.

District	Canton	Population	Phthisis of local origin		Exposure or shelter to westerly winds
			Deaths in 5 years	Annual death-rate per 1,000	
ALTITUDE 1,300-1,499 METRES (4,265-4,920 FT.). *No Industrial or Mixed.* *Agricultural.*					
Adelboden	Bern	1,544	5	0·6	**S** (" ? if complete ")
Nax, St. Martin	Wallis	2,734	3	0·2	¾ **S**
Totals	...	4,278	8	Av. 0·3	
ALTITUDE 1,500 METRES AND UPWARDS (4,921-5,905 FT.). *No Industrial or Mixed.* *Agricultural.*					
Champéry	Wallis	548	2	0·7	**S**

TABLE VIII.—SUMMARY OF AVERAGES IN TABLE VII.

Height over sea-level in metres	Phthisis of local origin. Average death-rates per 1,000		
	Industrial	Mixed	Agricultural
200-499 (656-1,639 ft.)	1·6 ...	1·2 ...	1·2
500-699 (1,640-2,295 ft.)	2·0 ...	1·4 ...	1·1
700-899 (2,296-2,951 ft.)	-- ...	1·3 ...	1·09
900-1,099 (2,952-3,608 ft.)	2·0 ...	— ...	0·5
1,100-1,299 (3,609-4,264 ft.)	2·18 ..	— ..	0·6
1,300-1,499 (4,265-4,920 ft.)	— ...	— ...	0·3
1,500 and over (4,921-5,905 ft.) ...	— ...	— ...	0·7

"mixed" districts vary in death-rate apparently from the same cause; whilst the uniform lowness of death-rate in the agricultural localities of over 700 metres altitude seems to depend on their almost uniform shelter. Below 700 metres a considerable proportion of them are exposed, which accounts for their higher death-rates at the lower levels. It seems, in fact, as if, with increasing altitude, the increasing bleakness of the exposures had forced the inhabitants to build their villages in shelter, and the effect of this shelter in lowering phthisis death-rate had come to simulate a diminution in the prevalence of the disease directly due to altitude.

These tables conclusively dispose, I think, of any reasonable claim that in Switzerland during the five years selected an influence of altitude on the prevalence of phthisis, apart from increased or decreased exposure to rain-bearing wind, could be discovered. We have, however, in Müller's report still further evidence in the same direction, below the upper limit which Lombard assigned to his belt of increased prevalence. I lay particular stress on it because of its antagonism to more northern statistics presently to be quoted. The Canton of of Zürich was one of the most satisfactory in the series, because its returns were for five years throughout and appeared to the reporter exceptionally trustworthy.

Table XIX gives the thirteen highest districts, the ten lowest and the fifteen districts with the least mortality from phthisis. It will be seen from these figures that in this canton, where elevation almost always means increased north-westerly exposure, increasing altitude is associated not with lower but with very definitely higher mortality from phthisis. The populations given lend no colour to an idea that density of population has a determining influence. Occupation, however, evidently has an effect, and, in fairness to the upholders of the altitude theory, one must protect them by precautions which all of them do not themselves adopt. Yet even when only agricultural districts are compared with each other it is seen that the lower localities still have the lower death-rates.

But we can deduce more from Müller's figures than a mere negation. It is plain that, in shelter amongst agricultural populations, increase of altitude has no evident influence on phthisis prevalence; but what about populations exposed to rain-bearing winds? Of these we have here no certain examples above the limit which Lombard sets to his humid zone. Up to that limit, however, the phthisis prevalence appears to actually increase as we ascend, and we may reasonably ascribe this increase to the increased incidence of rain-bearing winds, since in shelter the same humidity at the same height may reasonably be assumed to exist, although it created no increase in the phthisis death-rates.

We may, in fact, state our conclusions so far thus: (1) In shelter from rain-bearing winds there is no evidence of any influence of altitude on phthisis prevalence; (2) in exposure to rain-bearing winds the prevalence of phthisis appears to increase with increasing altitude within the limits of Lombard's zone of increased humidity and rainfall.

All the foregoing, however, is based on figures compiled before Koch's discovery in 1882. We now turn to the more modern, and presumably more exact, statistics published in 1895 by the Sanitary Department of the Grisons. They were held by the Department strongly to support the idea that increasing altitude decreases the prevalence of tuberculosis. Three-quarters of the cases were pulmonary, and the figures may be reasonably dealt with as furnishing similar arguments for phthisis as for tuberculosis generally. For I have shown elsewhere that other forms of tuberculosis are affected by rainy wind exposure in much the same way as phthisis is. They express morbidities, not mortalities, and are, therefore, considerably larger.

The Grisons offers an exceptionally favourable field for investigating the question, as Müller had pointed out, since it provides such wide differences in inhabited altitude. Unfortunately, Müller had found it impossible to obtain satisfactory information from it. It also affords exceptionally marked contrasts of wind exposure and shelter. The west wind must be much broken and dried by its passage over the Western Alps, the south-west seems only to gain free access to the Upper Engadine. The south-east may have some effect as a rainy wind on the highest villages of the valleys which run down into Italy. North-westerly winds probably affect the canton most. They are especially prevalent in summer, the time of the chief rainfall. The table published by the Sanitary Department (Table IX) was, however, arranged in unequal divisions of altitude, and it was first necessary in examining it to arrange it in equal divisions. I did so in 200-metre divisions (Table X). So arranged, it by no means made clear a definite effect of

TABLE IX.—ISSUED BY THE SANITARY DEPARTMENT OF THE GRISONS (1895).

Number of communes		Metres above sea		Population		Tubercular morbidity per 1,000
15	...	285-599	...	20,369	...	12·18
40	...	600-999	. .	20,935	. .	9·74
64	...	1,000-1,499		25,346	. .	7·18
19	..	1,500-1,880	...	10,291	. .	5·64

TABLE X.—THE SAME RETURNS REARRANGED AT UNIFORM INTERVALS OF 200 METRES

	Morbidity, per 1,000
Places up to 600 metres high ...	12·08
,, from 601-800 metres high	8·8
,, ,, 801-1,000 ,,	12·5
,, ,, 1,001-1,200 ,,	7·1
,, ,, 1,201-1,400 ,,	7·5
,, ,, 1,401-1,600 ,,	4·2
,, ,, 1,601-1,880 ,,	[illegible]

altitude, and at each successive height pronounced exceptions were found to occur, which suggested some more potent influence in operation. It seemed possible that this influence was that of rain-bearing wind.

To ascertain whether rain-bearing winds had any influence on the figures given, a table was constructed showing the tuberculosis morbidity in places exposed to various winds, and it at once became obvious that a higher rate accompanied exposure to any westerly winds (Table XI). Another table [36] was drawn up of localities with morbidities under 5 per 1,000 and over 10 per 1,000. The localities with lower rates were then seen, with few exceptions, to be sheltered from all westerly winds, whereas those with the higher rates were, with two exceptions, exposed to one or other of them. The winds causing increased frequency of tuberculosis were generally north-westerly. So another table was drawn up comparing at each altitude the morbidities in the populations sheltered from north-westerly winds with those exposed to them. The result was very remarkable (Table XII). At every level the exposed populations suffered far more (twice to seven times as much) from tuberculosis than the sheltered. And one other important fact also became obvious—viz., that as altitude increased a greater proportion of the people tended to live in places sheltered from these winds, as if the exposures become too bleak for habitation. In neither exposed nor sheltered places was there evidence that altitude lessened the morbidity; on the contrary, in exposure the effect of altitude was not to lessen, but to increase it.

TABLE XI.—TUBERCULOSIS IN THE GRISONS. SUMMARY AS REGARDS SHELTER AND EXPOSURE.

Shelter and exposure	Population	Tuberculosis morbidity per 1,000
Sheltered from all	18,734	5·3
Exposed only to W.N.W., N.W., or N.N.W.	9,256	13·2
,, N. or N.E.	6,043	6·9
,, E.N.E., E., or S.E.	6,173	5·8
,, W.	280	14·2
,, S.W. or W.S.W.	118	19·7

It should be observed that the populations exposed to W., S.W., and W.S.W., are very much smaller than the others.

The incidence of westerly winds, therefore, not altitude, appeared chiefly to determine the distribution of the morbidity. To make quite sure, however, a table was made out showing the morbidity at successive heights in places sheltered from all westerly winds and from the south-east (Table XIII). From this table the conclusion seems inevitable that altitude of itself has little influence, if any, and only acts indirectly, in exposed places, by increasing the exposure. In fact, from these tuberculosis morbidities in the Grisons we arrive at precisely the same conclusions as from Müller's phthisis mortalities, of thirty years earlier, dealing with other cantons.

TABLE XII.—THE N.W., N.N.W., AND W.N.W. WINDS. EFFECT OF EXPOSURE TO, AND SHELTER FROM, THEM AT VARIOUS HEIGHTS (SUMMARY).

Altitude in metres	Altitude in feet	FULLY EXPOSED TO N.W.		FULLY SHELTERED FROM N.W.	
		Population	Morbidity per 1,000	Population	Morbidity per 1,000
Up to 600	1,968	14,039	14·4	4,874	5·3
601-800	—	1,937	12·4	4,004	5·2
801-1,000	—	199	25·1	2,369	6·3
1,001-1,200	—	260	19·2	7,212	5·6
1,201-1,400	—	2,092	15·3	5,237	2·4
1,401-1,600	—	270	18·5	8,273	3·8
1,601-1,880	5,250-6,160	0	—	4,966	8·6

It should be observed that the exposed populations at 801-1,000, at 1,001-1,200, and at 1,401-1,600 metres are very small.

TABLE XIII.—SHELTERED FROM ALL WESTERLY WINDS AND FROM SIROCCO (SUMMARY).

	Morbidity	Population
Up to 600 metres	3·9 per 1,000 ..	2,898
601-800 ,, ...	4·7 ,, ...	3,173
801-1,000 ,, ...	4·1 ,,	971
1,001-1,200 ,, ...	2·8 ,,	4,254
1,201-1,400 ,, ...	2·3 ,,	5,059
1,401-1,600 ,, ...	1·9 ,,	3,693
1,601-1,880 ,, ...	6·7 ,,	3,538

It should be noted that we have in these later statistics no means afforded us of eliminating errors due to imported cases or to differences of occupations, as we had in Müller's report. In my study of the district the shelters and exposures were deduced with great care from the contoured Swiss Government survey maps, kindly lent me by the late Dr. Huggard.

As to Dr. Lombard's humid belt, it is interesting to see in Table XII between 800 and 1,200 metres a definite increase in the morbidities both in shelter and exposure, especially in exposure, although the populations in exposure are too small for us to dare to draw any positive conclusions.

From these Swiss investigations, older and newer alike, therefore, I think we are entitled to affirm—

(1) That altitude of itself has no evident effect on the mortality from phthisis, such as has been supposed to exist—in other words, that increasing altitude does not here of itself appreciably decrease phthisis prevalence.

(2) That altitude has, however, in exposure to rain-bearing winds, just the reverse effect, the prevalence increasing with the altitude, this being presumably due to increasing exposure to these winds.

(3) That there is some support for Lombard's view that an intermediate zone of increased phthisis prevalence exists, roughly corresponding to the zone of maximum humidity and rainfall, at least in exposure to rain-bearing winds.

The Mountains of Germany.

Taking next the work done on the German mountains, there are some general points which have to be remembered. In the first place, all the localities dealt with are much lower than the highest inhabited Alpine, the higher ones coming, in fact, within Lombard's belt of increased phthisis prevalence. In the Hartz the heights dealt with are less than 2,000 ft. above sea-level, in Saxony the highest are a little over 2,000, and in Baden not much over 3,000 ft.; also, they are all below the level at which rainfall ceases to increase with altitude, and probably also suffer more from wind in exposed situations at higher than at lower levels. The claim, therefore, that phthisis prevalence becomes less as elevation increases is evidently not the same thing as in the very high altitudes we have been discussing. Moreover, the shelter afforded in the recesses of these hills is increased by the forests with which they are so often clothed. Again, all the work on German heights was done before Koch's discovery in 1882, and consequently when diagnosis was less certain than afterwards. Brockmann's conclusions, in fact, rest on erroneous diagnosis; nor was such care taken in any of the papers to avoid error as characterized Müller's inquiries; soil and shelter here, as there, were omitted from consideration; and in addition it will be seen that no such satisfactory means was adopted to prevent confusion from such sources as differences of occupation, density of population, defective notification, and imported cases. The earliest work was Brockmann's [6] on the Hartz in 1843. He alleged that pulmonary tuberculosis was less prevalent on the plateau of the upper Hartz than below. In support of this he only quoted two considerably different levels—Clausthal, including Zellerfeld, at a height of about 2,000 ft., and Lerbach, a mere village lower down, in a narrow valley running due south-west. He definitely said that he based his statements as to rarity of phthisis on Clausthal. Now Clausthal is a mining town, with silver, lead, and copper mines, and he admitted that a form of consumption, which in his opinion was not tubercular, was very common there. This other form was evidently, from his description, miner's phthisis. Statistics of phthisis in a mining community, from which miner's phthisis is excluded, cannot be regarded as convincing, and his paper must be dismissed as valueless.

Von Corval's [10] work in Baden dealt with four years, 1869 to 1872; he allowed that his material was defective; all cases were not medically certified (he made no estimate of the proportion) and he found it necessary to include all cases certified as chronic pneumonia (Table XIV). Six groups of places were compared in ascending order of altitude, but as the names of the places were not given it is impossible to examine them further. He claimed to show that in higher altitude alone is to be sought one of the most important factors in hindering the development of consumption. Except for a partial elimination of density of population by tables from which towns of over 10,000, 5,000, and 3,000 inhabitants were successively excluded, no attempt was made to get rid of any conflicting influence. To do him fuller justice than he has done himself, or than Hirsch and Schlochow [37] have done him, I have calculated from his figures Table XV, which eliminates density

TABLE XIV.—VON CORVAL'S TABLE FOR BADEN, 1869-72, INCLUDING ALL TUBERCULOUS DEATHS AND DEATHS FROM "CHRONIC PNEUMONIA."

Altitude in feet	Number of towns or villages	Population, average of four years	Death-rate per 1,000
330-1,000	750	933,773	3·36
1,000-1,500	337	224,210	2·75
1,500-2,000	160	81,066	2·60
2,000-2,500	190	104,287	2·75
2,500-3,000	97	59,155	2·33
Over 3,000	47	20,367	2·17

of population down to a certain point, which is as far as he gives the means of doing.

Upon his work certain comments seem justifiable. It is true, as von Corval claims, that the last column in Table XV suggests a certain tendency in the phthisis mortality to diminish as altitude increases, and this is not negatived by the other columns. But the figures show equally clearly an influence of size of town, and inasmuch as it is evident from his tables that the places over 3,000 ft. have the largest percentage of small villages, decreased density of population may alone account for the lowest figure. Again, it was stated that by far the greatest number of inhabitants are occupied in agriculture and wine-growing, &c.—open-air occupations—but that a certain proportion are employed in manufactures, not only in the largest towns, but even in the narrowest valleys of the Schwartzwald. No attempt, however, was made to separate these, so that we are free to surmise that probably as altitude increases open-air employments become relatively more common. Further, in the highest villages it is likely that the percentage not medically certified increased, and that the apparent lessening of phthisis death-rate may be due to this. These three considerations alone would invalidate any conclusion founded on his tables. But we have another reason for rejecting them as proof of an influence of altitude. Hirsch supplies a table [38] showing the mean annual phthisis death-rate per 1,000 for the different circles of Baden only a few years later than von Corval's work—namely, 1874 to 1881 (Table XVI). Here we observe a still steeper decline in

TABLE XV.—CALCULATED FROM VON CORVAL'S FIGURES.

		Tuberculosis death-rate per 1,000 living in towns of populations of			
Group	Height in feet	Over 10,000	5,000-10,000	3,000-5,000	Under 3,000
I	330-1,000	4·5	3·4	3·6	3·1
II	1,000-1,500	—	2·9	3·5	2·7
III	1,500-2,000	—	—	4·9	2·5
IV	2,000-2,500	—	—	3·2	2·7
V	2,500-3,000	—	—	3·0	2·3
VI	Over 3,000	—	—	—	2·1

TABLE XVI.—BADEN AVERAGE ANNUAL PHTHISIS DEATH-RATES PER 1,000 INHABITANTS IN THE VARIOUS "CIRCLES," 1874-81 (HIRSCH).

Mannheim	3·87	Constance	2·65
Karlsruhe	3·41	Lörrach	2·54
Baden	3·28	Mosbach	2·3
Freiburg	3·05	Villigen	2·3
Heidelberg	3·04	Waldshut	2·24
Offenburg	2·89		

values, and if we examine the orographical map of Baden we shall see that these areas stand very much in their order of exposure to westerly, and especially to north-westerly, winds. Taking into consideration, therefore, the effect of these four factors alone, which have not been eliminated in von Corval's work, his figures, I submit, lose all significance.

Merbach's [11] table for Saxony (Table XVII), if we exclude the places under 200 metres high, only proves, if indeed it can be held to prove anything, that altitude has no effect whatever on phthisis prevalence. But he gives the names of the towns, their populations, altitudes, and phthisis death-rates. So that it is possible to examine them more fully and see if, in reality, any evidence of an influence of mere altitude exists. The highest town was only 2,066 ft. above sea-level. Only towns were dealt with, but no distinction was drawn on the score of density of population or prevailing industry. The number of cases not medically certified was considerable, in some actually so many as 40 to 60 per cent. of the total, but the percentage is given for each town. A survey of the map of Saxony will quickly show that the higher death-rates occur chiefly in the more open country, the lower amongst the hill valleys of the south-west. In Table XVIII I have

TABLE XVII.—MERBACH'S TABLE FOR SAXONY, 1873-75, DEALING WITH TOWNS OF OVER 5,000 INHABITANTS ONLY AND DEATHS FROM PHTHISIS BETWEEN THE AGES OF 16 AND 60 YEARS.

Elevation in metres		Phthisis death-rate per 1,000
100-200	... ...	4·9
200-300	... ...	3·3
300-400	... ...	3·2
400-500	... ...	3·5
550-650	... ...	3·2

TABLE XVIII.—SAXON TOWNS ACCORDING TO DIRECTION OF THE RIVERS ON WHICH THEY STAND. ANNUAL PHTHISIS DEATH-RATE PER 1,000 AT AGES 14 TO 60.

	Averages			
↗	3·2	...	3·1, 3·7, 2·1, 2·7, 3·8, 3·6, 2·5, 2·6, 2·4, 2·7, 4·5, 3·4, 4·2, 3·8, 4·1	
↑	3·6	...	3·7, 3·9, 3·7, 3·1, 4·4, 3·6, 3·0, 3·6, 3·3, 3·5	
↖	4·7	...	4·2, 3·2, 3·9, 4·3, 3·3, 5·3, 6·1, 4·6, 7·4, 4·5, 5·6, 4·9, 4·2	
←	3·6	...	3·6	much westward shelter.
↙	3·6	...	3·8, 3·4	

Or only considering Towns where at least 80 per cent. of the Cases were Medically Certified.

	Averages		
↗	3·2	...	3·7, 2·1, 2·7, 3·8, 3·6, 2·5, 2·6, 4·3, 4·1
↖	4·7 nearly*	...	4·2, 4·9, 5·6, 4·5, 7·4, 4·6, 5·3, 3·3, 4·3, 3·2, 4·2

* I.e., nearly 80 per cent. were.

put a column of arrows to show the direction of the rivers on which the towns stand, as a rough means of gauging their exposure. It is interesting to observe that the towns on rivers which flow towards the north-west, and are therefore exposed at least to winds from that quarter, have an average phthisis death-rate 50 per cent. higher than the towns which, standing on rivers flowing north-east, are more or less sheltered from all westerly winds, since these latter lie almost entirely amongst the hills. The variations in his figures, therefore, are presumably due in part to wind. With this and other influences uneliminated all significance of his work disappears.

Lübben [39] in 1880 dealt with the distribution of cases of tuberculosis in Thuringia (using morbidities, not mortalities) and only dealing with individual practices in towns here and there, his phthisis-rates being expressed in percentages of all sorts of kinds of illness. Such material is obviously most unsatisfactory. No attempt was made to eliminate other influences and the separate town morbidities were not stated. All that his figures show, if indeed they can be admitted to show anything, is a certain rarity of phthisis in the Thuringenwald. Certainly no claim for altitude can be made from them.

Virchow's [8] statement that in the Spessart phthisis is uncommon is only what one would expect from a sparsely inhabited, densely forested hill country, and is explained by himself solely on the grounds of occupation and open air. Virchow himself made no suggestion that altitude had anything to do with the low death-rate, although Hirsch quotes his statement in support of this theory. Brehmer [9] found phthisis non-existent in Görbersdorf at the height of about 1,700 ft.; but Görbersdorf lies in an exceptionally sheltered valley on the east of the Riesengebirge amongst their foothills.

With these observations in Germany should be compared Table XIX, Müller's figures for the Canton of Zürich (already alluded to) at much the same heights, but where every death certificate had been medically signed, and where, for every parish separately, the necessary particulars of height, population, occupation, and number of cases originating locally had been carefully given. The result, as we have seen, was the exact reverse of the result in almost neighbouring Baden and the rest of the German districts examined.

Fuchs's [7] comparison of the great town of Hamburg with the sheltered little mountain town of Brotterode need not detain us, nor Küchenmeister's [40] now untenable generalization. The German mountains, in short, afford no valid evidence whatever of an antagonism between altitude and phthisis.

Other European Heights.

In England I have carefully examined the registration districts to discover whether difference in altitude has any effect on the disease—although our altitudes, needless to say, in no way compare with those which we have been discussing. The only fact I can trace is the same that I have traced abroad—namely, an increasing liability to phthisis

TABLE XIX.—CANTON ZÜRICH (THE CITY OF ZÜRICH EXCLUDED) (MÜLLER).

Highest Parishes.

Population		Phthisis death-rate	Metres high
1038 I	Sternenberg	1·2	900
2228 I	Fischenthal	3·4	781
2990 I	Bäretsweil	2·9	700-800
1096 I	Hirtzel	2·5	700
4713 I	Wald	2·0	650-870
1726 I	Hittnau	2·0	640-770
1435 I	Schönenberg	1·9	700
2662 I	Hinweil	1·5	540-780
659 A	Hutten	1·5	740
690 A	Zumikon	1·5	700
2203 ?	Turbenthal	1·4	556-812
2382 A	Egg	1·3	551-800
672 A	Aeugst	1·2	712

Lowest Parishes—under 400 Metres (1,312 Ft.).

Population		Phthisis death-rate	Metres high
731 A	Weiach	1·9	389
733 ?	Schlieren	1·6	395
2288 A	Rorbas	1·3	396
1390 A	Flaach	1·3	374
998 A	Rheinau	1·2	394
1555 ?	Dietikon	0·9	392
1503 A	Eglisau	0·8	340
1486 A	Glattfelden	0·8	369
2822 A	Andelfingen	0·4	395
502 A	Dorlikon	0·4	390

Altitudes in Metres of Parishes with Phthisis Death-rates under 0·7 per 1,000.

		Per cent.			Per cent
A 390	Dorlikon	0·4	A 446	Regensdorf	0·5
A 395	Andelfingen	0·4	A 446	Stadel	0·6
A 413	Marthalen	0·6	A 532	Buch	0·3
A 415	Laufen	0·5	? 560	Uitikon	0·6
A 422	Rümlang	0·5	? 600	Riffelweil	0·4
A 428	Dorf	0·5	A 617	Regensberg	0·6
A 434	Wyl	0·3	? 630	Kyburg	0·5
A 439	Dällikon	0·3			

I means industrial. A means agricultural.

where altitude increases exposure; and it is curious to note that in 1818, nearly a century ago, as already mentioned, an increase of phthisis with increase of altitude in England was recorded by Dr. Mansford, whose papers, unfortunately, I am unable to trace.

In the Pyrenees, Eaux-Bonnes, Eaux-Chaudes, Cauterets, and other places have been mentioned by Sir Hermann Weber [41] as freer from consumption than other localities in the plains below, but these towns are wonderfully free from wind and have been specially described as sheltered by Dr. Burney Yeo [42], who writes of Eaux-Bonnes: "There is exceedingly little wind, and I was assured by an excellent authority that the air is often so still that one may pass days without seeing a leaf stir on the trees." In the Alpes Maritimes, Briançon, the highest town in Europe, has been quoted as singularly free from consumption, but it is shut in and thus greatly sheltered by mountains.

The Indian Hill Stations.

In the Himalayas the high altitude theory met with its first obviously serious reverse. It is true that Hirsch [44] has stated that immunity from phthisis came out decidedly on the northern and southern slopes of the Himalayas at the elevated points of the Western Ghats on the Nilgherri Hills, and on the Mount Aboo, but he relied upon very old statements, some going back to 1823, which do not tally with more recent information. Some excuse for this lies in the wonderful difficulty of obtaining reliable knowledge of the frequency of phthisis in these regions. But the following statements seem sufficiently definite to quote.

Davidson [45] states that between 1867-76 among the Goorkha regiments stationed at Dehra (2,232 ft.), Dhurmsala (4,500 to 6,600 ft.), Abbottabad (4,120 ft.), Almora and Bakloh (both at considerable elevations), all on the south-westerly slopes of the Himalayas, the proportions of deaths from phthisis to the total mortality was as high as 20·3 per cent., whilst the proportion among the native troops stationed in the Gangetic provinces below was only 10·6 per cent. It should, however, be here observed that racial proclivity to phthisis appears to differ considerably in India, and the Goorkhas seem to be particularly liable to the disease. Davidson states that the highest elevations of the Western Ghats and of the Himalayas are not free from consumption, though it is there comparatively rare; but in Coorg, at an elevation nowhere under 3,000 ft., it is said to be very common.

Dr. Alexander Crombie [46], reporting at the Congress of Tuberculosis at Berlin in 1899, on the prevalence of consumption in India, quotes Webb as stating "amongst the natives inhabiting the lower range of the Himalaya Mountains I have seen scrofulous swellings and ulcers of the neck common." The figures for the ten years ending 1896 showed that amongst native troops the higher rate of admissions for phthisis was in the hill regions. The three places where phthisis appears to be rare—namely, Kashmir (in which it undoubtedly is so, as I learn from Dr. Neve), the valley of Nepal, and Manipur [47]—are all in exceptionally sheltered situations. Kashmir is comparatively windless, and phthisis is almost unknown outside its capital, Srinagar, but, in it, it is by no means rare.

It should be remembered that the hill stations of India have in the rainy season an extremely heavy rainfall. In the absence of direct information regarding prevalence, it may be held permissible to record, as bearing indirectly on the point, comparatively recent experience on the effect of Indian mountain resorts on the course of the disease. Their unsuitability has scarcely received the attention which the great experience of those who have insisted on it should have ensured.

Sir Joseph Fayrer [48] referred to them as follows: Simla (6,953 ft.) "should be avoided by the phthisical." Ranikhet (6,086 ft.) is not of much real service. Darjeeling (6,912 ft.), more useful than the foregoing, enjoys considerable immunity from wind and storms. Pachmari (3,564 ft.) is ineligible as a resort for phthisis. Mount Aboo (3,945 ft.), phthisis is not benefited there. Mahableshwar (from 4,500 ft. to 4,700 ft.), after the mists set in in May, is prejudicial to phthisis. The Nilgherris, with their average elevation of station of about 6,500 ft., are not advantageous in phthisis; and Newera Eliya (Ceylon) (6,150 ft.) "has an evil reputation of phthisis." At Dalhousie (6,800 ft.), in 1897, Surgeon-General Keogh informed me that six admissions for phthisis in the early stages were returned to the plains because the climate was too severe for them. Here, therefore, we have very high altitudes which are detrimental to phthisis patients and in some of which the disease appears to be common. These, however, are situations open to wind and with a heavy rainfall.

The Rockies, South Africa, and Persia.

Next, taking the Rocky Mountains, there is no doubt of the rarity of consumption in Colorado, New Mexico, and Arizona. From a study of Solly's "Medical Climatology," and Davidson's "Geographical Pathology," there can be no doubt of the rarity of phthisis in the eastern foothills of the Rockies and in their valleys and tablelands, in spite of some prevalence of wind: but then the climate is very dry. The rainfall in places is as low as 9 in. annually. It does not seem, in fact, that, as regards the prevalence of phthisis, these districts differ much from lower countries which are similarly dry, like the Darling Downs of Australia, or Upper Egypt. Similar remarks apply to the high tablelands of South Africa and Persia. In none of these situations has any attempt been made, so far as I know, to prove increasing rarity of phthisis with increasing altitude. The statements regarding Abyssinia are conflicting.

Taking all this considerable mass of information into account, and it is all that is at our disposal, it seems to me impossible to arrive at any other conclusion than that no evidence of any value exists in favour of the high altitude theory of phthisis immunity. Whether a fuller investigation on the lines of the principle which I have suggested will furnish any such evidence remains to be seen. To set the matter finally at rest it should be undertaken.

I trust that I have given sufficient illustration of the principle on which I have ventured to lay such stress—sufficient proof of its extreme importance. In closing my survey of the subject I would repeat my belief that the place of climatology in medicine should be, and is destined some day to be, a very great one, and, as a means of placing it there, I would once more urge the adoption of this principle.

THE MEDICINE OF THE FUTURE.

Thus far, however, I have dealt only with medicine as at present understood, but in the progress of civilization will there not also be an expansion of its scope? Shall we rest content with the advance only from what one may call reparative medicine to preventive medicine? Will not the tendency be to proceed to what may some day be described as "constructive medicine"—that is to say, to the actual use of medical science in building up a more perfect organism than man at present is? With this in view we come back to the ideas mooted by Hippocrates, and it would be unfitting for anyone dealing in these days broadly with the subject of climatology to seem to fall behind the sandal-prints of the great Asiatic Greek. More than two thousand years ago he pointed out the advantage of living in a city which faced the morning sun, and though the greatest apparent exception to the rule he laid down was even then rising on its Seven Hills, his view has received remarkable confirmation since. Capital cities have curiously often stood upon an east coast, not merely in States with which he was acquainted, but in what were then the distant Western Isles and unimagined lands beyond the Pillars of Hercules.

Kindred questions, too, perhaps unknown to him, have since suggested themselves. Why, for instance, does empire usually establish itself most securely westwards? Why in the Lampadephoria of the nations has the torch of culture been passed almost continually towards the Pole? To such questions we have no answer. Yet may they not indicate profound climatological truths which we would do wisely in trying to understand?

CONCLUSION.

Gentlemen, I have done, and I am under no misapprehension in respect of my shortcomings. I have not even offered you a completed sketch. The outlook in some directions has seemed to me so wide that I have not ventured to delineate it. Only this I will say in conclusion to those who, upon the same road, are about to pass beyond me. Bearing always in mind the infinite care and patience which alone can lead to certainty, have that faith in your findings that needs no explanations, and lay aside expectation, which is the veil that hides the obvious; so shall you come into a new realm of intellect in which humanity has more than it knows to hope for.

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Balneological and Climatological Section.

May 31, 1913.[1]

Dr. PERCY G. LEWIS, President of the Section, in the Chair.

Some Phenomena connected with the Passage of Electricity through Rocks and its Relation to Atmospheric Electricity.

By A. G. S. MAHOMED.

WHEN I had the honour of addressing you before on this subject I gave a short general outline of the facts of atmospheric electricity, and described some recording apparatus I had designed, a part of this being portable, so that observations might be made afield. I also gave some results I had so obtained. I began by observing that the earth is always negatively electrified, that the atmosphere was nearly always positively electrified, and that the higher levels of the atmosphere showed a stronger electric tension until a height was reached—about 4,000 metres—where it was believed the electric condition was permanent; while the greatest range was discovered near the earth, negative conditions being sometimes encountered. I also drew attention to the daily range from maximum (about 8 a.m. and 8 p.m.) to minimum (which is about 2 p.m., and 3 to 4 a.m.) and to the seasonal variation (which is greatest at the equinoxes and least at the solstices), and mentioned its relation to the auroral frequency, magnetic storms and sun-spots. The reason why the atmosphere should be positively electrified has provoked many scientific speculations. One is that it originally received a positive charge at the creation of the earth, and has kept it ever since. Of course we are dealing with *static* electricity, but that seems rather a long time to resist dissipation. Another explanation is that snow, rain, hail, fog and dust storms continually take place in the lower strata of the atmosphere, and since at the disruption of an electric corpuscle the negative ions are more quickly and easily carried down by these particles than the positive, the earth is continually reinforced by negative ions, while the positive remain in the atmosphere. That theory, I believe, was propounded by Lord Kelvin. There are other theories, some depending on the influence of the sun. I hope, however, to demonstrate to you to-day some phenomena which may provide a foundation for another theory, which, if it does not explain everything, may have such local significance as to interest you as medical men.

---

[1] Provincial Meeting held at the Mont Dore Hotel, Bournemouth.

I have here a primitive kind of electrical machine, in which catskin is made to revolve against a piece of vulcanite. This is a ridiculously powerless machine; nothing would elicit a spark from it. It is the same thing as rubbing a vulcanite knitting-needle on your coat sleeve, but it is continuous, and it has the merit that it develops the same kind of weak electrification at a high potential as you meet with in the atmosphere. I have also a copper plate insulated on ebonite legs, and another copper plate suspended above it by a balance, so that by drawing on a silk thread it can easily be raised or lowered. Now, if we put the two plates in contact, connect the lower plate with the electrifying machine, and the upper with an electroscope, you may observe that on turning the machine the gold-leaf moves out to a certain point, representing a tension of some 400 or 500 volts. If the upper plate is raised it retains the charge, and this is found by testing to be a negative one.

I now interpose a piece of stone, Purbeck marble, between the plates. On turning the machine you will notice that the gold-leaf again moves out (though a little more slowly), showing that the electrification has passed through or over the stone; then raise the top plate so that it just swings clear of the stone—earth it, the gold-leaf drops; give a few more turns and gently raise the plate still higher. The leaf moves out a little way as the plate ascends, which is curious, as it is being removed from the source of electrical energy; moreover, on bringing a negatively excited rod near it the leaf droops, and on removing the rod it again moves out. It is obvious that the upper plate has received an induced charge of the opposite sign—it is now positively electrified. We will now place a piece of chalk between the plates and proceed as before. After earthing, it will be noticed that while the machine is in motion the leaf moves slightly out, but on raising the plate it falls. It has not received a positive charge. If we remove the chalk and substitute a layer of sand from the cliffs, dried and flattened down to the thickness of the chalk, the same result follows—it is not positively electrified.

What is the explanation of these phenomena? The chalk from its porous nature is incapable of being well dried, it is consequently more or less of a conductor; it therefore admits the passage of negative electricity through it, and the copper plate above it receives that kind of electrification. The sand has been dried, but the discrete, pointed particles of sand also afford an easy passage to electricity. The Purbeck stone, on the contrary, is dense, hard and smooth, and acts like an insulator; it acts like a Leyden jar. There you have tinfoil, glass, and tinfoil again. The electric condition on the outer foil is of the opposite kind to that on the inner foil. The glass is of high dielectric capacity. Here you have copper, stone, and copper. The electric current is torsed and broken. The electric corpuscle breaks down, the negative ions remain on the lower plate, the positive seek the higher. The dielectric capacity of Purbeck stone is demonstrated to be higher than that of chalk or sand.

What is the relation of this to atmospheric electricity? I would call to your remembrance that when platinum, copper, or iron is heated to redness, the air in the neighbourhood is found to be positively electrified; the platinum is negatively electrified. This was Professor Thompson's observation. We believe that the interior of the globe is still hot; the deeper you go down the higher the temperature rises. From volcanoes and geysers, hot air, steam, gases, and lava arise. There is, therefore, reason to suppose that the molten metals in the interior cause a flow of negative ions outwards, and I suggest that when these come to the cooled crust they induce a positive charge in the atmosphere above it. I have endeavoured to represent this in the diagram I now show you. Near the earth the electrification is not very strong, but in the higher portions of the air the positive potential increases. That, I should think, is what anyone would expect.

Diagram 1 is intended to represent a section through a part of the earth core, the crust, and the superjacent atmosphere. The curvature of the earth is increased so as to bring the diagram into convenient size. The section might represent a portion of the Bournemouth plateau, the Isle of Wight (too sheer), and the cliffs at Swanage. I have endeavoured to show the interior of the earth as molten or flaming, which is a little picturesque; above it are first plutonic and other rocks, then sedimentary deposits, and finally sand outcropping at Bournemouth, the various strata twisted up at the Island, and the Purbeck stone at Swanage lying upon ferruginous masses. You observe that the upper part of the atmosphere is of the deepest blue, to indicate its high potential. Over the Bournemouth area it is nearly colourless just above the pines in the depression (it appears quite colourless in the photograph, and the pines can only be seen by magnification). A higher potential is indicated over the rise at the cliff. Over Swanage the potential is high above the mass of limestone rock, and the lines

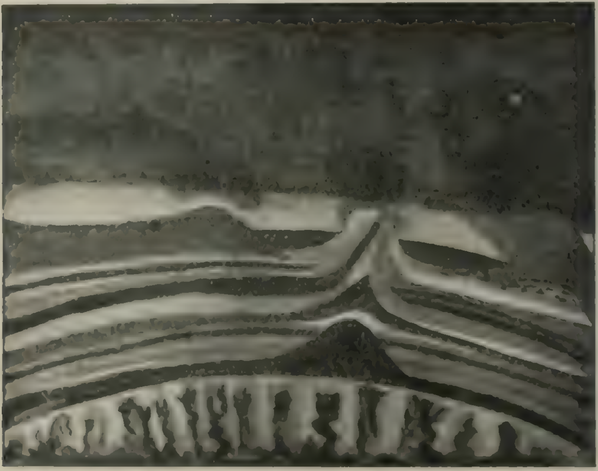

Diagram 1.

Section through earth crust. Lines indicating direction of negative electrification require a magnifying glass to become visible.

of negative electrification are deflected in their passage through the dielectric.

It may be objected by some of you that it is only in those portions of the crust where rocks of high inductive capacity abound that this positive induction would take place, but I would remind you that rocks of low inductive capacity are placed on others that have a higher capacity. Our sand rests on clay and gravels, so that everywhere the sum of the rocks composing the crust are such as to build up a dielectric or insulator, which probably presents a tolerably even average, though no doubt where granites and slates outcrop the local electrical condition is one of higher potential than where sands, peat, ferruginous rocks, alluvium, or chalk occur. This no doubt has a relation to health. It is the influence, or rather part of the influence, which older physicians described as *telluric*. No doubt humidity or dryness is another factor. I spoke of these matters to a German officer some time ago. He said: "I believe what you say. Years ago I was in garrison in ——, in Silesia. We never felt so very fit while we were there, but only a mile or two off, on the plain, there were stones everywhere and a small village. All the fellows there were as healthy as anything. I used to ride over there because the air was so good."

I will now call your attention to diagram 2, which represents observations made on the same day on two occasions, one in October, 1911, the other last Sunday, during trips by motor over a track of country where the change from heath and Bagshot sands to chalk and meadow land occurs. You will see that the change is marked on each occasion, but as the change is exactly opposite in character some of you will think no useful deduction can be made therefrom, but I do not think this is so. On the second trip, certainly, no meteorological change appeared to be in progress; it was cloudless, hot, and with a slight breeze from south-east. It is, therefore, probable that the variation is due to telluric differences. And this change may be due to the more rapid drying out of chalk or sand on the two occasions, making one or other a better dielectric. Moreover the variation in the readings is progressive with the change of strata on both occasions.

Dr. Le Cadet observes: "The negative tension at a point of the surface of the earth is normally equivalent to the sum of the masses of positive electricity spread in a certain thickness of the atmosphere above that point."

It appears to me possible that we might amend this apothegm thus: "The positive tension of the lower part of the atmosphere at a point above a certain part of the earth's surface is normally proportional to the dielectric capacity of the earth's crust at that point." I say normally, but you will readily see that to obtain a normal at any point is very difficult on account of the varying meteorological conditions, particularly in these islands: clouds, rain, hail, snow, fog, haze, and wind modify them profoundly.

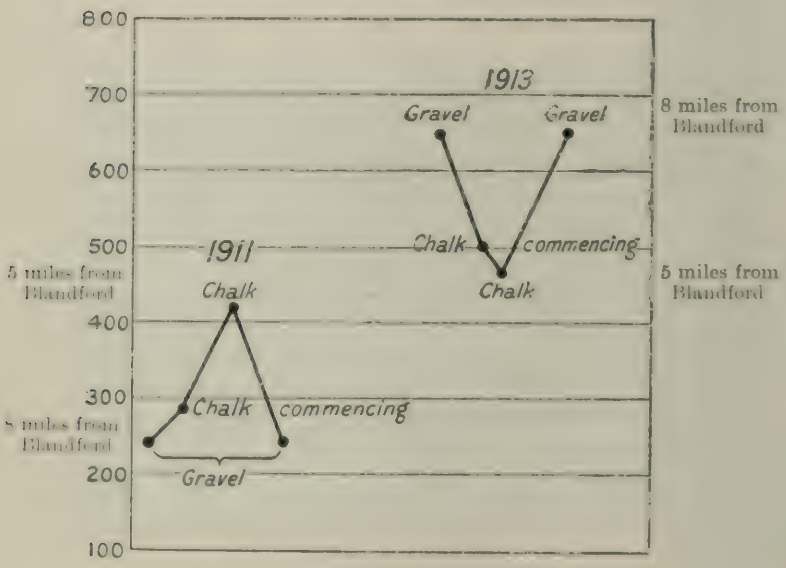

DIAGRAM 2.

These observations were made at identical spots on the Hamworthy-Blandford road in 1911 within three hours, in 1913 in a less space of time. The figures on left indicate the potential in volts. The first observation point was on bracken and heath at a higher level than number two; the third spot was intermediate.

I have long tried to compare the potential at Swanage, and at Bournemouth: at first, by making an observation here and then one at Swanage as soon as a steamer would take me there. Then, by getting someone to remain at the hut and record the readings there, while I went to Swanage and made an observation by my portable apparatus. Only last month was I able to set an observer to make an observation at 10.15 every morning while I made one here. These observations are too few to be very useful, but on the day of the storm last month we both observed an oscillating condition in the morning, but Swanage was 200 or 300 volts higher than here. This agrees with the observation of a friend who lived on an island in Poole Harbour. He said: "Storms begin at Swanage, pass over the harbour, up the valleys to Blandford or Salisbury. That is the usual course. I have, however, seen storms travel towards Bournemouth." This also agrees with the common knowledge that Bournemouth has comparatively few electric storms. Other factors are the condensation of rain or fog over the Purbeck hills during south-west winds; another is the influence of pine trees on the electric conditions. When you consider the influence of radiation of electricity from points, you will notice how great must be the radiation of negative from the numerous pine needles. There is no doubt that a plantation of pines, more than of any other tree, must exercise the protective influence of lightning conductors which are often made with numerous metallic points, as in the Palais de Justice at Bruxelles, in order to get rid of accumulated negative electricity. I have on one occasion found a high negative at Swanage on a bright day with very little cloud. Just before hail or electric storms it is common to read a negative.

Some other records were shown which seemed to prove that a local change of weather can be sometimes prognosticated by a drop in the potential occurring several hours before any barometric change. A chart was shown of daily readings during January to April, 1912, to which were added the mean readings at the same hours on the self-recording instruments at Kew. They approximated fairly closely. The readings at Bournemouth were very low for a long period, though there were several spells of dry weather. During a frost the readings at both stations rose suddenly, dropping as the frost continued. In April the readings rose again with some abruptness, and thereafter remained fairly high.

Note.—The day after the meeting at which this paper was read I showed about a dozen of the visitors the method of taking observations by lamp at my hut. Just before they arrived the gold-leaf dropped from

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Section for the Study of Disease in Children.

October 25, 1912.

Mr. A. H. TUBBY, President of the Section, in the Chair.

Case of Tetanoid Spasms.

By EDMUND CAUTLEY, M.D.

MALE child, born September 4 at term, without difficulty, and brought up on milk and barley water. Since 10 days of age he has had attacks of rigidity about every half hour. The mouth is said to be clenched, the neck rigid, but the head not retracted, and the hands clenched. He takes food with difficulty and regurgitates some through the nose during the attacks. The child's nutrition is good. There is no indication of sepsis, and only a few curds are present in the stools. During the attacks the whole of the upper half of the body becomes rigid, the neck muscles extremely so, and the head is slightly retracted. The eyes are closed and the mouth can be opened very little. The fontanelle does not bulge. The hands are flexed at the wrists and the fingers and thumbs hyperextended, not assuming the attitude characteristic of tetany. The toes are hyperextended. Since admission the attacks have become less frequent and less severe.

Dr. CAUTLEY said that the chief difficulty concerned the diagnosis. Possibly there was some error of cerebral development.

Case of Partial Aphasia.

By EDMUND CAUTLEY, M.D.

FEMALE child, born March 25, 1909, was admitted to the Belgrave Hospital for Children on July 29, 1912. She was one of four healthy children, breast-fed as a baby, and had had varicella in her third year. For twelve days she had had slight cough, and for six days drowsiness and anorexia. On July 28 there was general twitching, possibly a fit. On admission she was drowsy and uttered occasional cries. There was no fever. The pupils were dilated and sluggish and there was slight convergent squint. The head was a little retracted and the neck muscles rather stiff. The legs were flexed at the hips and knees, and the child resented them being straightened. There was moderate general bronchitis.

August 5: Flaccid paralysis of the legs. Knee-jerks normal. Plantar reflex extensor on the left, flexor on the right side. Cerebrospinal fluid was under considerable pressure, clear, and contained very few cells. The child uttered an occasional meningeal cry.

August 12: Legs and arms apparently in a state of flaccid paralysis but moved in response to repeated stimuli. No head retraction or spinal rigidity. Unable to sit up or hold up her head. Kernig's sign present. Knee-jerks absent. Triceps and supinator reflexes present. Cerebrospinal fluid as before.

August 19: Condition unchanged. Convergent squint more marked. Occasional cries.

August 26: Took more notice and moved arms about. Could not speak.

September 1: Able to sit up. Fed herself fairly well. Still unable to speak.

September 7: Could stand with some support. Attempted to articulate and said "yes" and "no" fairly distinctly. Habits not yet clean. No ocular palsy or fundus change.

September 12: Could stand alone and walk with assistance. Said "yes" and "no" distinctly in a drawling manner. Clean in her habits. The supinator reflex and the knee-jerk were more marked on the right than the left side. Both plantar reflexes were flexor.

September 15: Walked without support but unsteadily, and made more attempts at speech.

Since the last note the girl has steadily improved and gained weight. She now speaks fairly well, in a somewhat drawling manner, and is quite intelligent.

The diagnosis rests between encephalitis and serous apoplexy.

DISCUSSION.

Dr. CAUTLEY expressed his preference for the term "amnesic aphasia." It depended on an acute cerebral illness, and was due to loss of memory for words. That memory was now gradually returning. The question was as to the condition at the time of the acute illness. During it the child was more or less unconscious, and drowsy for some days, and lost the use of her limbs. The cerebrospinal fluid was under increased pressure, but it was clear and contained no excess of cells. The child was now in much better health, and could walk about normally. She could say many words clearly, but rather slowly. Possibly the case was one of serous apoplexy. One could exclude encephalitis because of the absence of high temperature and paralysis of cranial nerves. The left squint was due to congenital defect of refraction. There was no history of headache, but probably she had had headache, as she uttered cries like those of a meningeal case. There was an excess of exudation of serous fluid in the ventricles of the brain, and perhaps outside it, which later was re-absorbed. Similar cases had been described under the term "meningismus," but that was a vague name, indicating simply that there was some irritation of the meninges.

Dr. PORTER PARKINSON agreed with Dr. Cautley that the condition was probably not due to encephalitis, for one other reason besides those he had given—namely, that it was clearing up without leaving any permanent damage. Encephalitis left such permanent damage. A boy, aged 6 years, had been sent to him from the country who, three years previously, had had an illness as to which the doctor only remembered that he was very constipated and that the trouble was a nervous one. Though previously the child had spoken well for its age, there was, after this illness, complete loss of speech, and when he saw the patient there was motor aphasia. He understood what was said, and the nature of objects shown to him, but could not say a word. That was more like the condition of encephalitis than the present case.

Case of Congenital Syphilis; Hæmaturia.

By J. PORTER PARKINSON, M.D.

THE patient is a girl, aged 8 years. Father and mother considered healthy. There are seven other children: the first was born dead, the second was a seventh-month child and lived seven hours, the third child suffers from epileptic fits, and the others are presumably healthy. The child has a healthy history. She had scarlet fever a year ago, and measles and chicken-pox previously, but no other illnesses. There has been a squint for six years. Three weeks before admission the patient had jaundice, vomiting, and diarrhœa, the urine was red in colour, and there was some swelling of the face and limbs. This passed off, but reappeared, and the child was brought to the hospital a week later.

On admission she was pale, with a slight earthy tinge of face. Slight œdema of the eyelids and ankles. The heart, lungs and abdomen were normal. The blood-pressure was 115 mm. The urine was reduced in quantity, 8 to 10 oz. daily, specific gravity 1020, a considerable quantity of blood and a small amount of albumin. Microscopically no casts were seen, only blood cells and a few epithelial cells. There is a small linear scar on the lower lip. The Wassermann reaction of the blood is strongly positive, on this account she is having mercurial inunction. The blood in the urine rapidly diminished, and neither blood nor albumin was present after first week in hospital.

Mr. Stephenson reports that the squint is a constant convergent concomitant squint and has no connexion with syphilis, of which there is no sign in the eyes.

The cerebrospinal fluid is negative to the Wassermann test.

I am showing the case to elicit the opinion of Members of the Section as to the cause of the hæmaturia, whether it is syphilitic or not. My own view is that it is probably non-syphilitic.

DISCUSSION.

Dr. PORTER PARKINSON added that he would like to know the opinion of members as to the production of hæmaturia by syphilis. Syphilis produced various forms of kidney disease, and he had previously shown a case in which the urine was loaded with albumin, and in which there were various manifestations of syphilis, including infantilism.

Dr. CAUTLEY remarked that congenital syphilis produced hæmorrhagic nephritis in the early weeks of life, and then often proved fatal. There seemed no reason, therefore, why congenital syphilis should not cause hæmorrhagic nephritis of a milder type later in life, and hæmaturia as an accompaniment. He did not think hæmaturia occurred in congenital syphilis apart from nephritis.

Dr ROBERT HUTCHISON said he supposed there was no doubt that it was hæmaturia. Everyone knew that hæmoglobinuria was a common result of congenital syphilis. Recent investigations in Glasgow had shown that paroxysmal hæmoglobinuria had a syphilitic basis more often than had been supposed by many. He was inclined to agree with Dr. Parkinson that in this case the hæmaturia had nothing to do with the syphilis, because there was a history of the child having had jaundice, vomiting, and diarrhœa three weeks before admission, with some swelling in the face and limbs. That looked like acute nephritis of infective origin. Everyone must have seen blood persist in the urine after these acute hæmorrhagic attacks of nephritis in children even after the other signs of nephritis had disappeared.

Dr. THURSFIELD said that some years ago he had under his care a child who was a congenital syphilitic. For five or six years, while under his observation, she passed blood in the urine without any other evidence of nephritis. The general opinion of those who saw the case was that the hæmaturia was due to her congenital syphilis. In the present case he agreed that the hæmaturia was probably an acute attack, unconnected with the specific disease.

Mr. ALFRED L. SACHS said he did not think it had been clearly shown whether the hæmaturia was of renal origin, whether from one kidney or both, or whether it was due to some disease of any other part of the urinary tract. Schede had reported cases of renal gummata which he had found situated in the pyramids of one or both kidneys, and other similar cases, some congenital, had also been recorded (Legrain, Israel). He (the speaker) had never met with a case, and could not declare whether such gummata would cause hæmaturia. If syphilis was the cause in this child, the precise origin of the blood should be first conclusively proved by cystoscopy.

Dr. F. PARKES WEBER asked whether the "few epithelial cells" mentioned as present in the urine of the child were supposed to be renal epithelial cells and indicative of nephritis. If so, that was strong evidence that the case was one of the kind sometimes met with both in adults and in children, in which nephritis showed itself chiefly by the passage of blood in the urine. He asked Dr. Parkinson whether he thought that a congenital syphilitic taint in children sometimes predisposed to attacks of ordinary nephritis.

Dr. PARKINSON, in reply, drew attention to the fact that the patient had some anasarca and albuminuria as well as hæmaturia. He meant that there was more albumin in the urine than the blood would account for, and this pointed to the renal origin of the hæmaturia. The cells described as epithelial cells were renal cells. He was aware that a connexion existed between syphilis and hæmaturia; indeed, he showed a case of that nature two years ago.

Case of Congenital Family Cholæmia.

By F. J. POYNTON, M.D.

[*With some Further Investigations into the Condition of the Blood by* T. S. LUKIS, M.D.]

S. S., FEMALE, aged 8½ years, was admitted to hospital for an exacerbation of a jaundice from which she had suffered since birth. She has always been delicate and subject to these exacerbations on slight indisposition, particularly in cold weather. Her father has also been jaundiced from birth and was treated for many years for gallstones before the condition was recognized as a hæmolytic jaundice. He has been a great sufferer from minor indispositions associated with bouts of jaundice and anæmia which have greatly crippled his activity. His family is as follows:—

(1) Born jaundiced; died of "convulsions" at 4 months.

(2) Twins; lived seven and eight hours only; cause of death unknown.

(3) I. S., female, aged 10½ years, treated recently in hospital for incontinence of urine. A healthy-looking, bright, intelligent child. While in hospital spleen was noticed to be enlarged slightly. Soon after admission she suffered from a very slight attack of jaundice, only noticeable in the conjunctivæ; this was associated with slight indisposition, and was followed by a noticeable enlargement of the spleen, which subsequently regained its former dimensions. This was the first time jaundice had been noticed in this patient.

(4) S. S., aged 8½ years; the patient.

(5) Dead child; six months, premature.

(6) M. S., aged 7 years; healthy; no enlargement of spleen now, and jaundice never noticed. Three years ago slight splenic enlargement.

There is no collateral family history of jaundice on the father's side, and the mother and the mother's family are free from the complaint.

The patient is a vivacious, rather precociously intelligent child, but delicate-looking, and below the normal in physique. There is marked pallor of the skin and mucous membranes, with an icteric tinge that is variable in its intensity. The spleen is palpable but not markedly enlarged. The motions are normal in colour. The urine occasionally contains urobilin, but has never shown bile pigment. The blood serum examined during an exacerbation of the jaundice was free from urobilin or bile pigment. The Wassermann test on the blood was negative, as it was also on that of her sister, I. S. The fragility of the red blood cells, tested on four separate occasions, gave readings of 0·6, 0·65, 0·65, and 0·65. It is interesting that her father's fragility, on the last occasion that it was tested, stood at 0·65 also; her sister's at 0·6.

A blood count on September 28 gave the following: Red cells, 3,711,250; white cells, 11,200; hæmoglobin, 56 per cent.; colour index, 0·75. Differential count: Small lymphocytes, 12 per cent.; large lymphocytes, 15·3 per cent.; large mononuclears, 14·7 per cent.; transitional cells, 2 per cent.; polymorphonuclears, 53·3 per cent.; eosinophiles, 2 per cent.; myelocytes, 0·7 per cent. Many blood platelets present.

No nucleated red cells seen. An interesting feature of the case has been the demonstration in the blood by Pappenheim's "vital" staining process of "hématies granuleuses" or "reticulated red cells" in considerable numbers. A microscopic preparation of the blood showing these cells is being shown with the case.

Very scant attention, if any, has been paid in English medical literature to these cells, which were first described by Pappenheim and Israel in 1896. They occur in normal blood in a proportion of 1 to 2 per cent. of the red cells, but are very numerous in fœtal blood, diminishing rapidly towards term. They are found to be increased in numbers in many forms of severe anæmia, and are a particularly constant feature of the blood picture in acholuric family jaundice. In the patient's blood they have totalled 10 per cent., 8·4 per cent., and 11 per cent. of the red cells on three occasions. In her father's blood they stood at 6 per cent., and in her sister's at 5 per cent. In her sister, M. S., who is free from jaundice, only 0·2 per cent. of reticulated red cells were found. These reticulated red cells are to be regarded as evidence of very rapid blood regeneration.

The patient has been treated by the application of X-rays to the splenic region three times weekly, and has appeared to be much improved, the spleen becoming distinctly smaller and the jaundice less. Her mother testifies that she has never seen her so free from jaundice.

DISCUSSION.

Dr. POYNTON added that his case was a classical instance of the condition, and he was showing it in consequence of the further investigations which had been done for him by Dr. Lukis. The chief point was the demonstration of reticulated red blood corpuscles, about which Continental authorities had written so much, and Dr. Lukis exhibited a microscopical specimen demonstrating that point. The effect of X-rays was now being tried on the spleen, but he had not yet anything to report.

Dr. LUKIS said Dr. Poynton had asked him to say a few words about reticulated red cells, because he did not think they had yet been described in this country. These cells were first described by Pappenheim in 1896, and they were demonstrable only by the "vital" staining method. These cells were found in normal blood in only small quantities, but in large quantities in fœtal blood. They diminished rapidly in numbers up to term. They were found in excess in the blood of newly born children for the first twenty-four hours, and in abnormal numbers in icterus neonatorum, and in the graver anæmias.

They were not the same as the granular red corpuscles described in connexion with pernicious anæmia and other serious blood conditions. There was still much controversy as to their nature and significance: as to whether they were nuclear or protoplasmic. Pappenheim regarded them as protoplasmic, and as a sign of rejuvenescence of the blood, not of a degeneration of it. The significance of the cells in the present case was that they were nothing more than evidence of a rather large and rapid hæmopoiesis following on the hæmolysis associated with this form of jaundice. These cells were, according to Chauffard's experiments, less fragile than ordinary red cells, and he regarded them as the type which was developed with increased resistance for the purpose of defence against the toxin, whatever it might be. They would be glad of suggestions as to the pathology of the disease, and as to how far the disease was congenital. Several cases had occurred in this one family. If it was congenital, what process was congenital? Was it a congenital deficiency of the blood or of the liver? Rist and Ribadeau-Dumas, in France, had experimented with poisoning animals by bile salts, producing anæmia in them comparable to that in this present patient. These animals could be immunized to large doses of sodium taurocholate, but if splenectomy was performed, they lost their immunity. He understood that splenectomy had been performed in some of these cases of jaundice as a curative measure. He did not know what the theory of that procedure was.

Case of Excision of Spleen for Congenital Family Cholæmia.

By C. R. Box, M.D.

A. J., AGED 12 years. This patient is the subject of congenital cholæmia. Her mother (who was shown at the same time) has suffered from the same disease ever since she was a girl. A sister has anæmia and splenic enlargement. A younger brother also has an enlarged spleen, but shows slight polycythæmia. The patient was jaundiced for six weeks after birth and is known to have had an enlarged spleen since infancy. She has always been delicate. Five years ago a purpuric eruption appeared on her legs, and this recurred the following year. During the purpura she vomited some blood and also passed some bright blood by the bowel. For the past four years there have been recurrent attacks of persistent vomiting accompanied by pain in the left side of the abdomen, splenic swelling, and jaundice. Latterly the jaundice has been constant, although varying in depth. There is no bile in the urine but plenty in the stools. Her blood has been frequently examined; it always showed a moderate anæmia, with a low colour index. Anisocytosis has been noticed, but is not marked. Normoblasts have been seen on one occasion (when the purpura was present). The leucocyte count was 15,800 in her second year, the polynuclear cells and lymphocytes being approximately equal. More recent counts are given below. Five weeks ago the spleen was excised by Mr. E. M. Corner. It weighed 390 grm., and showed moderate perisplenitis. The splenic tissue had undergone some fibrosis, and the sinuses were dilated. There was no evidence of increased splenic activity and no reaction for free iron could be obtained.

A week after operation the patient's condition became critical and remained so for another week. Her temperature gradually rose to nearly 105° F.; vomiting of bilious matter was incessant, the pulse was very rapid, and complaint was made of severe pain in the joints, but there was no joint swelling. She is now convalescent, and the daffodil tint of her skin has quite disappeared.

BLOOD EXAMINATIONS.

(Made in the Clinical Laboratory of St. Thomas's Hospital by Dr. Weir under the direction of Dr. Dudgeon.)

	Before splenectomy	Twelve days later	Five weeks after operation
Red cells	3,337,500	4,950,000	3,868,750
White cells	10,640	6,220	21,280
Hæmoglobin	65 per cent.	80 per cent.	75 per cent.
Colour index	0·9	0·8	0·9
Polynuclear cells	50·0 per cent.	76·75 per cent.	50·25 per cent.
,, eosinophiles	2·25 ,,	3·75 ,,	13·5 ,,
Small lymphocytes	40·0 ,,	14·0 ,,	25·25 ,,
Large ,,	3·75 ,,	0·75 ,,	3·75 ,,
Large hyaline cells	3·5 ,,	5·75 ,,	6·0 ,,
Mast cells	—	—	1·25 ,,

Hæmolysis still begins with 0·5 per cent. saline.

[*Supplementary Note.*—On October 30, after albumin and casts had been noticed in the urine for a few days, uræmic convulsions set in and soon terminated fatally. Post mortem a small abscess was found in the splenic stump and acute inflammation of the kidneys. The liver contained much free iron.]

DISCUSSION.

Dr. SUTHERLAND considered that the question of excision of the spleen in connexion with this disease was one of great interest. His own experience of excision of that organ was limited to two cases of primary splenic anæmia, and it was the only effective treatment at their disposal. He did not know whether it had been done previously for cholæmia. He asked whether there was hæmatemesis before the operation in Dr. Box's case. He understood that the result of the operation had been not only improvement in the blood, but complete disappearance of the symptoms of cholæmia. If so, it seemed to point to the spleen being the primary organ concerned, as in primary splenic anæmia. Before the excision of the spleen there seemed to have been no leucopenia at all, but after excision there was diminution of the white cells; whereas in primary splenic anæmia the converse was true.

Dr. F. PARKES WEBER said he believed there had been one or two operations of splenectomy on the Continent in cases of chronic enlargement of the spleen in which the red blood cells had shown so-called "fragility" in regard to hypotonic saline solutions; at all events, splenectomy for "hæmolytic" splenomegaly had been tried, with good result, by Banti recently.[1] Another question was as to whether the anæmia in such cases of hæmolytic splenomegaly could be relieved by intramuscular injections of defibrinated blood from a healthy person; he believed that that treatment had been successfully employed in one case. With the improvement of the anæmia in the same class of cases there might still remain a deficient resistance of the red cells to hypotonic salt solutions. In fact, one could really speak of a condition of *hæmolytic polycythæmia* supervening, when the patient's anæmia had been turned into polycythæmia under treatment, though the peculiar fragility of the red blood cells remained.[2]

Dr. ROBERT HUTCHISON said it was difficult to understand the rationale of splenectomy in such a case as the present one, because he understood that in these cases the red corpuscles were more fragile, even in the presence of ordinary serum; hence one could not think that the spleen was producing a hæmolytic poison. If the spleen were producing a poison, one could understand removal of that organ doing good, but if the red cells were congenitally friable through some defect of bone-marrow, it was difficult to see why splenectomy should do good. The whole matter, however, was as yet so obscure that one had to abide by the results of experience.

Dr. GORDON R. WARD said the first two cases of excision of spleen for this condition were Australian cases, and were successful. They were published in 1904. He reminded the meeting of some early experiments of Hunter, in which he injected one of the blood poisons and produced the usual hæmolytic anæmia, and in other cases he excised the spleen first, and injected the same amount per body-weight, and the anæmia was less. The experiments showed that removal of the spleen might ultimately cure the disease, and yet the disease might not originate in the spleen, but merely expressed itself by altering the splenic function. The cases he referred to were those of Springthorpe and Stirling.[1]

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[1] On this subject see Guido Banti, "La Splenomegalie hemolytique," *Sem. Med.*, Paris, 1912, xxxii, p. 265.

[2] See especially in regard to this subject: Renon and Ch. Richet fils, Societe medicale des Hopitaux de Paris, meeting of July 26, 1912; also Widal, Abrami, and Brule, "A propos des rapports entre la fragilite globulaire, l'anemie et la polyglobulie," Societe medicale des Hopitaux de Paris, meeting of October 11, 1912.

Dr. THURSFIELD, referring to Dr. Ward's remarks, said that as the disease under discussion was not known in 1904, the excision of the spleen, then recorded, must be referred to some unknown condition.

Dr. SUTHERLAND agreed that the reference to cases in 1904 could not be accepted in this connexion, unless the clinical details definitely established the disease to be the same.

Dr. POYNTON, in reply, said he was in a difficulty at present in deciding whether the spleen was diminishing in size as part of the natural course of the disease or not; and he had no right, at present, to say that X-rays had done any good or any harm in this case. The question of operation interested him very much, because this child had scarcely any bad symptoms, but among the four families under his care some had had violent attacks of paroxysmal pain, sometimes over the liver, sometimes over the spleen. The Australian cases could, he thought, be verified, and an opinion arrived at as to whether they were congenital cholæmia, for the disease was a fairly definite one.

Dr. BOX, in reply, said there was a history of hæmatemesis in his case, and on two occasions there had been purpura: some years ago she passed some bright blood *per rectum*. The spleen removed showed peri-splenitis and a good deal of fibrosis. The Australian cases were reported by Springthorpe and Stirling, and the operations were done in 1903. There had been severe anæmia, in one case with "chlorotic tinge and an anxious expression." Removal of the spleen in this case was curative. A severe case in a sister without cholæmia was also cured by splenectomy. There was no subsequent lymphatic enlargement in either patient. Another sister, not operated upon, had jaundice with enlarged spleen. The blood of these patients was not tested for hæmolysis; the question had not arisen at that date.

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[1] "Six Cases of Splenic Anæmia in one Family," Springthorpe and Stirling, *Lancet*, 1904, ii, p. 1031.

[There were an uncle, three aunts, a nephew and a niece affected. All had large spleens and a "chlorotic complexion." There were signs of hæmolysis in the two spleens removed, and these were very marked. Hæmolysis is also suggested by the reaction of the blood which showed many normoblasts in two cases, and a few in another. There was also a colour index above 1·0 in all cases examined at one time or another, and excessive anisocytosis, presence of megalocytes and microcytes, and polychromatophilia. There is an absence of the leucopenia associated with the more usual types of splenic anæmia. The course of the disease showed frequent exacerbations of the anæmia followed by comparative recovery, thus agreeing with cholæmia. The exact meaning which the authors attach to the word "chlorotic" is doubtful, but the use of the expression "she was anæmic but not chlorotic" suggests that the tint so described was different from that of anæmic pallor, and possibly the peculiar primrose colour of cholæmia. Thus the family incidence, the splenic and the hepatic (in two cases) enlargement, the evidence of hæmolysis, the high colour index and absence of leucopenia, and some constant change in the complexion other than that of simple anæmia, are all suggestive of cholæmia and differ from the state of affairs met with in other types of splenomegaly with anæmia.—G. R. W.]

Two Cases of Genu Valgum due to Rarefaction and Deformity of the Shaft of the Femur.

By P. MAYNARD HEATH, M.S.

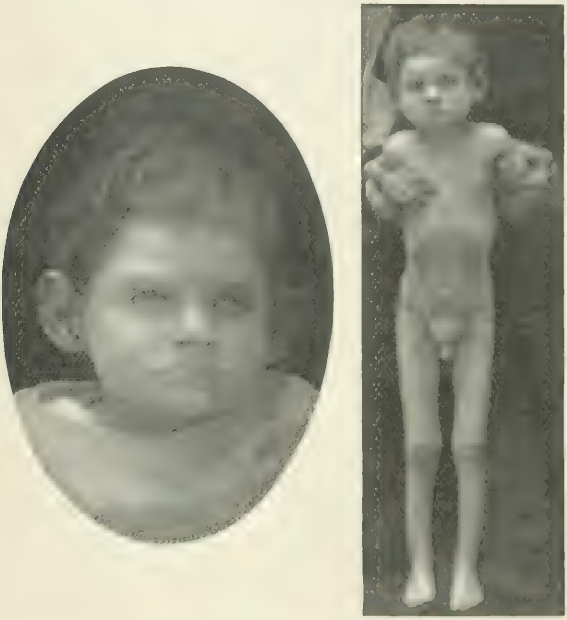
Case I.—Girl, aged 5½ years. She suffers from severe right genu valgum but can walk. There is no history of injury, but she was very much neglected in early childhood. She shows obvious signs of rickets (shape of the head, rosary, enlarged epiphyses). She is very pale and nervous. The chief deformity is due to a sharp bend in the shaft of the right femur about 4 in. above its lower extremity. An X-ray photograph shows that at this joint the bone is so rarefied as to be practically translucent. The rarefied area is not sharply outlined, and there is no evidence of cyst formation. There is some laxity of the ligaments in the right knee, and a well-developed Macewen's spine at the point of attachment of the internal lateral ligament to the tibia. There is very little deformity about the left knee, but well-marked coxa vara on each side and some deformity of the upper extremity of the right humerus are present. X-ray pictures in these situations do not show areas of rarefaction.

Case II.—Boy, aged 5¾ years. He is stunted, and shows well-marked signs of rickets. He suffers from bilateral genu valgum, most marked in the left limb. The deformity is similar to that in the first case, but not so great, and the rarefaction is not so marked. Macewen's spines are developed on the tibiae, and there is an exostosis at the upper end of the left humerus.

Cerebral Maldevelopment (? Sclerosis), with Infantilism and Idiocy.

By REGINALD MILLER, M.D.

BOY, aged 8½ years; born at full term by instrumental labour; first child. Appeared normal at birth but did not develop properly; grasped nothing in fingers until 3½ years old. Teeth erupted from twelfth month to third year, but rapidly decayed and most were extracted when 4 years old. Height 35 in., weight 22¼ lb., circumference of head 17½ in. Face senile in appearance from falling in of mouth. Testicles partially undescended; left small. Cannot walk or talk; never cries. Feet tend to cross, hands and fingers hyperextended; tremor of lips and hands. Dirty in habits, very destructive. By retching efforts brings food up into mouth, and unless controlled produces vomiting by forcing fingers down his throat; the amount of this vomiting daily is considerable. Wassermann test negative. Urine normal.



Case of cerebral maldevelopment.

Skiagrams (Dr. Harrison Orton): Long bones small, but otherwise normal. Mandible small and senile in type; only five teeth of second dentition visible unerupted.

DISCUSSION.

Dr. SHUTTLEWORTH, with reference to the ætiology, asked if there were any elucidating circumstances in the family or personal history of the case. He asked on what grounds it was thought to be one of sclerotic idiocy; had there been, since birth, any symptoms in the brain or membranes of an inflammatory character? He had tried to glean some of those particulars from the mother, but she gave no such history; she did not even try to account for the child's condition by a pre-natal fright. He had seen similar-looking cases, but they were usually syphilitic, of which condition there was no suspicion in this one.

Dr. MILLER, in reply, said he could not trace any other cases in the family. The diagnosis of cerebral sclerosis was suggested, believing that to be a congenital condition, because the child showed certain spastic changes, cross-legged progression, with tremor of lips and hands such as might be associated with a lesion of the red nucleus or its tracts. If it was not congenital—and the mother was rather anxious to say the child was born normal—the only other explanation of the infantilism was the vomiting. For years vomiting had occurred after everything the child took; this would account for much wasting, and possibly for lack of growth. The patient did not resemble the pictures of progeria, with the marked baldness and the arterial changes found in old age. He thought the senile appearance was due merely to the mandibular alteration from the loss of teeth.

Dr. GILFORD confirmed the impression of Dr. Miller that it was not a case of progeria, as described by his brother. The aspect of the child suggested marasmus.

Case of Coxa Vara.

By DUNCAN C. L. FITZWILLIAMS, F.R.C.S.

BOY, aged 9 years. Has limped ever since starting to walk, and the right leg was said always to have been shorter than its fellow. The apparent shortening was about 4 in., the real shortening fully 1½ in. The condition was extremely like a congenital dislocation of the hip, for the whole of the right limb was smaller than the left; this is especially well seen in the feet. The X-ray shows an extreme degree of coxa vara, probably congenital in origin. An attempt to place the limb in an abducted position after sub-trochanteric division of the bone has only been partially successful, but the shortening is much decreased.

Case of Traumatic Pancreatic Cyst after Operation.

By T. H. KELLOCK, F.R.C.S.

THE patient, a girl, aged 11 years, was knocked down by a horse in March, 1912, and said she was kicked by the horse in the abdomen. She was admitted to hospital almost immediately afterwards, somewhat collapsed and complaining of pain and tenderness in the left hypochondrium. The muscles in that region were very rigid, and it was thought that there was some fulness in the region of the spleen; there were no external marks of injury. The case was taken to be one of slight rupture of the spleen. Nothing was done surgically, the child gradually improved, and was sent to a convalescent home about a fortnight later, there being still a little increased resistance to palpation in the left hypochondrium. She remained at the convalescent home for three weeks, and then returned to her home and attended school apparently quite well. With a number of other children she was, one day, examined by the school medical officer, who found she had an abdominal tumour; of this the child was unaware.

Readmitted to hospital September 14, 1912, apparently in perfectly good health, a tumour was visible as she lay on her back or stood up, occupying the left hypochondrium; it was tense, fluctuating, fixed, and not tender on manipulation; extended to the right beyond the middle line of the body; was resonant above, but quite dull on percussion; at its central part resonance could be obtained behind it in the left lumbar region. The temperature was normal, and there were no abnormal constituents in the urine.

A few days after admission an incision was made through the upper part of the left rectus muscle into the abdomen and the cyst exposed. It was found to be retroperitoneal, pushing the stomach upwards and the transverse colon downwards. A trocar and cannula were inserted, and 50 oz. of greenish, opalescent fluid escaped; it was alkaline, and on examination proved to be pancreatic. A finger passed behind the cyst detected the left kidney in its normal position; there were thought to be a few adhesions round the spleen. The opening in the cyst wall was enlarged and a finger passed into it; the interior of the cavity felt rough and granular, and the finger could be made to reach in front of the spinal column and beyond the middle line. The edges of the opening into the cyst were sutured to the abdominal wall and a large drainage-tube inserted, the edges of the abdominal wound being closed round this; a long tube was added to the drainage-tube leading into a bottle by the side of the bed.

During the following twenty-four hours the temperature rose to 102° F., and on the third day to 103° F., but then rapidly fell to normal. Only 3 oz. of fluid escaped by the tube into the bottle, and as none appeared to be collecting in the cyst, the tube was shortened on the fourth day, and removed altogether two days later, when the opening rapidly closed; the rest of the wound having healed by first intention.

The patient was discharged on October 8.

Mr Kellock added that the interesting points were the patient's age, the frequency with which a kick from a horse was the cause of this condition, the rapidity with which the child got well, and the small quantity of fluid that drained from the cyst after the operation. The cyst was probably a true pancreatic one situated in the substance of the gland and not a pseudo-cyst, as was often the case where the fluid collected outside the gland in the peritoneal cavity. The very little inconvenience that this cyst caused the patient was remarkable when one remembered it contained 50 oz. of fluid. Up to the present there was no sign of any re-collection of the fluid.

Case of Ocular Torticollis.

By Sydney Stephenson, C.M.

In 1873 Cuignet[1] drew attention to certain deviations of the head, which had been treated mechanically, or even surgically, but which in reality were due to strabismus, and were often capable of cure by operations upon the muscles of the eye.

The following is a case in point. It differs, of course, from cases where patients tilt their heads to one side in order to get rid of or to modify the influence of an astigmatism with oblique meridians, so-called "head-tilting." It is strictly comparable with cases where patients endeavour to eliminate the diplopia produced by paralysis of one of the external muscles of the eyeball by a compensatory carriage of the head, which may be so characteristic that the muscle affected can almost be diagnosed from that symptom alone.

G. C., aged 9½ years, was brought to the Eye Department of Queen's Hospital for Children on August 29, 1912, with the history that she had been treated for some months by electricity for torticollis. The child had constantly carried her head on one side since she was 12 months of age, and the condition had not become worse since then. She had had no illness other than measles at 2 years of age. She belonged to a family of nine children, of whom one died of pneumonia at 3 years, one is mentally unsound, and a third suffers from "rheumatism and a bad heart."

On admission: In the child's habitual attitude the head is inclined towards the right shoulder, forming an angle of about 30° with the vertical. It can be straightened instantly at the child's will. There is no tension on the sternomastoid muscle, no twisting of the head, and little asymmetry of the face, points in which the condition offers an instructive contrast with cases of ordinary surgical torticollis. There is no deformity of the skull. The spinal column is slightly inclined to the left in the cervical region, to the right in the dorsal region, and to the left in the lumbar region. The condition shows the usual accompaniments of a right dorsal curve.

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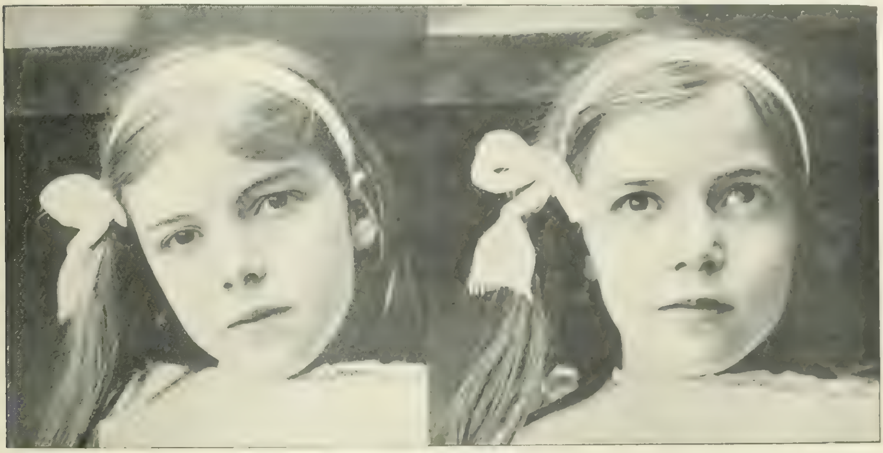
[1] Ann. d'Ocul., Par., 1873-74, i, p. 338.

With the head in the abnormal position, the right eye (as shown in the photographs) is usually free from squint, but at other times it is inclined downwards for from 8° to 10° (strabismus deorsumvergens). But as soon as the head is straightened the right eye squints downwards or the other (left) eye deviates upwards to a corresponding amount (strabismus sursumvergens). The investigation of double images is rendered untrustworthy by the nervousness of the child, but, as far as can be made out, when the head is tilted there is usually no diplopia, and when it is straightened double images are seen. Apart from the squint the eyes are healthy. Hyperopia, 0·25 D. Right vision, $\frac{6}{6}$ (two letters); left vision, $\frac{6}{6}$ (two letters). Visual fields for white full in both eyes.



Case of ocular torticollis.

DISCUSSION.

Mr. STEPHENSON said the explanation seemed to be that, possibly as a congenital defect, or at all events in quite early childhood, the child had paralysis of one or other of the muscles which elevate the right globe, with consequent formation of double images. The patient discovered that by inclining her head to the right shoulder she could do away with the double images. Although she was now 9½ years of age that posture of the head had become stereotyped.

The PRESIDENT (Mr. A. H. TUBBY) alluded to the interesting fact that, though the condition had lasted eight years, the sternomastoid had not become contracted. This seemed to him to throw some light on the causation of ordinary torticollis. It had been said to be due to malposition *in utero*, but if eight years' malposition outside the uterus would not produce contracture of the muscle, it was not likely that eight months inside the uterus would. The case was also valuable as showing the position of the spine which the position of the head had produced; the cervical column was deviated to the left and the dorsal spine to the right. It was a class of case with which he had been previously unacquainted, and the Section was much indebted to Mr. Stephenson for having brought it forward.

Dr. SUTHERLAND said he was also struck by the absence of change in the sternomastoid, and thought it might be due to the fact that the malposition of the head was not continuous, but only intermittent. During sleep the torticollis was probably absent and the spasm of the sternomastoid muscle subsided.

Case of Torticollis of (?) Ocular Origin.

By A. S. BLUNDELL BANKART, M.C., and A. H. PAYAN DAWNAY, F.R.C.S.

W. W., MALE, aged 7 years, attended the Orthopædic Hospital in May, 1912, for left wry-neck. The mother said that she had noticed the deformity from earliest infancy; the child was born after a long labour, but without instrumental aid. The child has a lateral flexion of the head to the left, which can be corrected voluntarily; there are no contractures or asymmetry of development. There is no ocular paralysis; the left eye converges slightly and is amblyopic; estimation of the refraction under mydriatic shows oblique hypermetropic astigmatism in both eyes, greater in the left. He has been wearing the correcting glasses (right, +0·75D, cyl., axis 60° down and in—vision, $\frac{6}{9}$; left, +3·0D, cyl., axis 60° down and in—vision, $\frac{6}{36}$) for four months, but he still holds his head to the left, unless told of it.

Mr. STEPHENSON suggested that this case had not the same ætiology as his own. Mr. Dawnay's case was an example of that fairly common condition in which the patient tilted the head so as to eliminate the influence of an oblique meridian astigmatism. Astigmatic patients nearly always assumed some curious attitude of the head when being tested for glasses, so that one was perpetually checking them and asking them to sit straight.

Case of Lateral Sinus Thrombosis followed by Thrombosis of the Facial Vein; Operation; Recovery.

By PHILIP TURNER, M.S.

T. S., AGED 14 years, was admitted on July 8 for headache and discharge from the left ear. The discharge from the ear had been present for eight years, and had appeared during an attack of typhoid fever. The acute trouble appeared about a week before admission after a visit to a swimming bath. The left side of his neck became stiff and painful, and he had a shivering fit. Since then he had one or more rigors daily until admission. When seen his temperature was 104° F., and pulse-rate 128. A diagnosis of lateral sinus thrombosis was made. The antrum and mastoid cells, when opened, contained pus, and the posterior wall of the antrum was found to be destroyed, opening the posterior fossa. A radical mastoid operation was rapidly performed, and the posterior fossa thoroughly exposed. The wall of the lateral sinus was sloughing and pus was oozing from a small perforation in this. The sinus was opened for an inch, and, after septic clot had been scraped away, bled freely from its upper end. The internal jugular was then ligatured. After the operation rigors occurred daily for seven days, while for a further two weeks there was a daily rise of temperature to about 103° F., with a feeling of chilliness without shivering. After the operation the left cheek became swollen and tender; two weeks later this was incised and about 2 oz. of pus were evacuated, the facial vein being seen traversing the abscess cavity. Double optic neuritis was present at first, but this had completely cleared up by the time he left the hospital. The temperature fell to normal twenty-three days after the operation, from which time convalescence was uneventful. For three weeks there was a cough with muco-purulent expectoration and physical signs of bronchitis. Cultivations of the pus showed the presence of *Bacillus pyocyaneus*, *pneumococcus*, and *Streptococcus longus*.

Case of (?) Anterior Poliomyelitis.

By T. R. WHIPHAM, M.D.

THE patient is a girl, aged 7 years, who presents an atrophy of the muscles of the forearms, especially on the left side. The wasting is more marked in the flexors than in the extensors. The hands are small, and there is marked wasting of the muscles of the thenar and hypothenar eminences. The interossei are also affected. The reflexes and sensation to all stimuli are normal. The upper arms are unaffected, and there is no abnormality elsewhere. No electrical reactions can be obtained in the flexores sublimis et profundus digitorum and in the small muscles of the thumbs. The condition was first noticed when the child was 3 months old, and does not seem to have progressed since. It was not ushered in by any illness. The patient was a full-term child—vertex presentation. The labour was easy and without instruments. There are no other cases of muscular wasting or paralysis in the family.

DISCUSSION.

Dr. WHIPHAM said that the interest of his case lay in the diagnosis between congenital myopathy and anterior poliomyelitis. If the lesion was congenital, the condition of the hands might not have been noticed until some time after birth when the child began to grasp objects. If it were anterior poliomyelitis, it must have commenced at a very early age, and have attacked chiefly, if not entirely, the upper extremities. The fact that the lesion was limited in extent, and that it had not progressed since it was first noticed, might possibly favour that view.

Dr. HUTCHISON did not regard the case as one of poliomyelitis if the history was accurate; the fact that it commenced when the child was 3 months old made that diagnosis unlikely. He had seen one case very much like it in which they were able to prove that the condition was due to the congenital absence of muscles, and that might be the explanation in this case.

Malformation of the Heart—Foramen Primum.

By Alexander Morison, M.D.

R. I., male, aged 5 months, was admitted into the Great Northern Central Hospital on April 29. He was suffering from difficulty of breathing, with signs of bronchitis, and a systolic cardiac bruit which was regarded as denoting cardiac malformation. The pulse-rate was 132, and the heart's rhythm regular; the respiratory rate was 32-38, and the temperature normal. The notes of the case are meagre, but one made on May 13, 1912, states that inspiratory suction was well marked over the lower ribs; that the respiratory rate was 60; that auscultation revealed fine crepitations; that the heart's rate was 114; that there was a systolic bruit over the cardiac apex, but not so loudly heard here as at the base of the left lung posteriorly. A cantering rhythm was also heard at the apex, and there was no cyanosis or clubbing of the fingers. There was little change in the child's condition during his stay in hospital, and he died on May 29, 1912.

The body was examined on the same day by the pathologist. It was well developed for the age; the head was not examined; the organs generally presented nothing of special interest, merely exhibiting the passive congestion to be expected from the lesions about to be described; the pleuræ were normal; the lungs showed numerous large and small patches of broncho-pneumonic consolidation, with areas of collapse in both lungs; the heart was increased in size and rather rounded in form; all the chambers of the heart, and especially the right auricle, were dilated and hypertrophied; the conus arteriosus and pulmonary artery were well developed, and the ductus arteriosus patent. It is described in the pathological report as widely patent, but has shrunk with immersion in formalin solution. The record states that there was a "very large foramen ovale." On this statement I shall comment presently. The auriculo-ventricular valves of both sides of the heart were continuous; the septum ventriculorum was perfect; the aortic orifice somewhat contracted, and at both the pulmonary arterial orifices and that of the aorta the full complement of valves was present.

The malformation described in the record as a "very large foramen ovale" was, in reality, a defect of the lower part of the auricular septum—the septum primum—one described by Professor Keith in his Hunterian Lectures[1] as "foramen primum." His lectures were based upon a study of a large number of cases from various sources, and he stated that he had seen five cases of this defect, but that the Museum of the College contained no example of it. He describes the defect as "one in which the endocardial cushions have fused, but are still separated from the crescentic lower margin of the interauricular septum by a space at which the blood in the two auricles may become mixed." He adds that it is not necessarily accompanied by any grave disturbance in the action of the heart.

On first examining this case I was inclined to regard it as an example of the absence of the septum ovale, as it is termed by Professor Keith in his lectures, but I have had the advantage of examining the specimen with him, and he regards it as clearly an example of foramen primum. Clinically the case was interesting as evincing the systolic bruit which accompanied the heart's action, most distinctly in the back, which, from the nature of the lesion discovered, might have been anticipated. When Professor Keith states that the lesion in question need not be accompanied by any grave disturbance of the heart's action, I presume he means that with normally developed arterial outlets and a wide interauricular opening there need be no evidence of the cyanotic engorgement often observed in some other congenital malformations. The condition is, however, scarcely compatible with prolonged existence, and the persistent ductus arteriosus and dilated and hypertrophied chambers in the present case, together with the non-febrile scattered consolidation and collapse of lung present, argue that the heart had to act at grave disadvantage, notwithstanding the absence of cyanosis.

Specimen of Tuberculous Right Kidney from a Child, aged 12 Months.

By EDMUND CAUTLEY, M.D.

SUCH a marked specimen of the disease is uncommon at this early age. It came from a girl, who was born on September 22, 1911, and died on October 14, 1912. She was the seventh child—three having died—of apparently healthy parents, and had been breast-fed for eleven months, and then brought up on milk and a proprietary food. At 6 weeks she had bronchitis. Previous to admission to hospital on September 23 she had had pertussis for three months and she still whooped. For two weeks the breathing had been more difficult. She took food badly, vomited on cough, and had a daily action of the bowels. Examination revealed a large, soft, tense, elastic tumour in the right lumbar region, extending down as far as the anterior superior iliac spine, and pushing the liver forward and to the left. The liver, especially the left lobe, seemed enlarged. The spleen was easily felt. A few small glands, anterior to the tumour, were distinctly palpable. The child was much wasted, had four teeth, and was slightly rachitic. No pus or tubercle bacilli were found in a catheter specimen of the urine, and von Pirquet's reaction proved negative. The tumour increased rapidly in size and presented a soft, fluctuating swelling over the end of the last rib. An incision was made in this region and about 14 oz. of sweet, sterile pus evacuated. Death ensued a few days later from asthenia. The temperature was irregularly febrile throughout.

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[1] *Lancet*, 1909, ii, p. 435.

The case was admitted as one of renal sarcoma. Alternative diagnoses were tuberculous kidney, pyonephrosis, perinephritic abscess, and localized tuberculous peritonitis.

Post mortem there was found extensive caseation, not breaking down, of the anterior half of the upper lobe of the right lung, and two small, softened, caseous bronchial glands, as well as some other small caseous mediastinal glands, but no general dissemination The liver and spleen were enlarged. There was no peritonitis. The right kidney was much enlarged and the upper half extensively caseous. It communicated with the remains of a post-nephritic abscess cavity. Two mesenteric glands were markedly caseous and a few others distinctly enlarged.

The PRESIDENT remarked that the case might have been suitable for partial extirpation of the kidney.

Notes on a Case of Precocious Development in a Boy, aged 6 Years.

By E. Cecil Williams, M.B.

W. J., aged 6 years, was admitted into the Bristol Children's Hospital on May 10, 1912. About six months prior to admission his parents noticed he was developing quickly.

On admission he weighed 4 st. 2 lb.; height, 4 ft. 2 in. Has a slight moustache; voice deep, like an adult's; muscles of arms and trunk well developed, can lift heavy weights; is slow of intellect and movement, cannot run as other boys of his age do; inclined to knock-knee, tibiae are also inclined to be curved; there is abundance of pubic hair, no axillary hair; external genitals fully developed; no bronzing of skin; heart and lungs normal; no tumour to be felt in abdomen; pulse varies from 72 to 88; blood-pressure, 110 mm. Hg.; fundi healthy; no sugar or albumin; circumference of head, 21½ in. Boy remained in hospital for five weeks, during which time he put on 11 lb. Weight equals that of a boy aged 13 years.

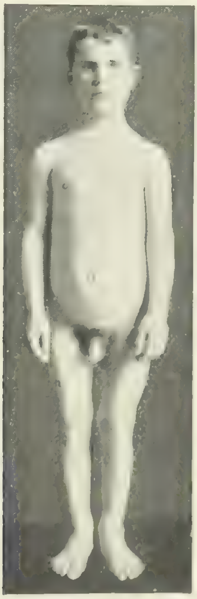
Bullock and Sequeira [1] have collected twelve examples of sex abnormalities in children, associated with adrenal hypernephromata, and verified post mortem; to this number Glynn [2] has added five more cases. A study of these seventeen cases shows: (1) that all except two were observed before 7 years of age (my patient is 6 years old); (2) that only three of the seventeen were boys (Mr. Hugh Lett [3] showed a living example of precocious growth in a boy aged 4 years); (3) a tendency in the case of males towards an increase in the male characteristics—e.g., in my case, the boy's muscular power—whereas in females there is a tendency to increase the male primary and secondary sexual characteristics at the expense of the female—e.g., appearance of beards, hypertrophy of clitoris.

I had an opportunity on October 15 of examining this boy again and comparing his weight and other measurements with those taken last June:—

	June 15, 1912	October 15, 1912	Normal boy, aged 6 years
Weight	4 st. 13 lb.	5 st. 5 lb. 5 oz.	3 st. 2½ lb.
Height	4 ft. 2 in.	4 ft. 4¼ in.	3 ft. 7 in.
Neck	12½ in.	13 in.	11 in.
Chest	26½ in.	29 in.	22 in.

This increase of weight and height, together with the advanced ossification shown in the skiagram of his hands, compared with those of an ordinary boy of 6 years, are striking evidence of his advanced skeletal development. There does not appear to be any further increase in his sexual development. No tumour can be felt; he is in excellent health.

Adrenal hypernephromata in children tend to grow slowly and do not all tend to disseminate. Other cases of precocity in children have been reported in connexion with the pineal gland. In order to ascertain whether there might be any pituitary enlargement, I have had a skiagram taken to show the base of the skull. I have the permission of Professor Fawcett to say that from the skiagram he thinks the "sella turcica" and pituitary fossa are enlarged. There is now a considerable amount of evidence of a correlation between sexual development and the various ductless glands. Bullock and Sequeira [1] refer to several cases of retarded sexual development in association with a hypoplastic or atrophied condition of the adrenals. They also refer to the "shrivelled" post-mortem appearances of the suprarenals in one of Hastings Gilford's cases of premature senility. The observations of



Precocious development in a boy, aged 6.

Gottschau [2] on the increase of cortical cells in pregnant rabbits at the expense of the medullary cells, and the observations of Stilling [2] on frogs during the pairing season, that the medullary cells disappear and characteristic cells called summer cells take their place, are adduced as evidence that the cortex is the portion of gland more intimately connected with growth and development. The blood-pressure of 110 mm. Hg. is certainly high. J. D. Rolleston [4] states that the blood-pressure rarely exceeds 105 mm. Hg. before 12 years of age. Dr. A. T. McCaw has taken a large number of blood-pressures at the Bristol Children's Hospital, which confirm Rolleston's figures. May this high blood-pressure be due to an excess of suprarenal or pituitary extract in the blood, possibly the result of glandular irritation due to a new growth?

REFERENCES.

[1] Bullock and Sequeira. *Trans. Path. Soc. Lond.*, 1905, lvi, pp. 189-208.

[2] Glynn. *Quart. Journ. Med.*, Oxf., 1912, v, pp. 157-92.

[3] Lett Hugh. *Rep. Soc. Study of Dis. in Child.*, 1905-06, vi, p. 200.

[4] Rolleston, J. D. *Brit. Journ. Child. Dis.*, 1911, viii, p. 436.

DISCUSSION.

The President said the X-ray picture seemed to show that the sella turcica was unusually large, and that there was an early development of the sphenoidal sinuses.

Dr. Thursfield pointed out that Dr. Williams did not mention the paper by Guthrie and Emery, published in the *Clinical Society's Transactions*.[1] In that paper the authors showed there were two distinct types: (1) the precociously obese type, not sexually precocious; (2) the "infant Hercules" type, muscularly big for its age, and always sexually precocious. The latter class was confined to males, the former in both sexes. He believed the present case belonged to the "infant Hercules" type.

Dr. Leonard Guthrie said Dr. Thursfield had expressed the view taken by Dr. Emery and himself (Dr. Guthrie) in the paper referred to. The present case could scarcely be said to be precociously obese, neither was it very muscular, therefore he would not like to place him in any one category. The skiagram was useful, as suggesting that a pituitary tumour might have some bearing on the condition.

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[1] *Trans. Clin. Soc. Lond.*, 1907, xl, pp. 175-202.

Section for the Study of Disease in Children.

November 22, 1912.

Mr. A. H. TUBBY, President of the Section, in the Chair.

Congenital Syphilitic Infant treated by Intravenous Injection of Neo-salvarsan.

By J. L. BUNCH, M.D.

THE child, a girl, was admitted under my care at the Queen's Hospital for Children on October 28, 1912. She was 2 years old, and was suffering from an offensive muco-purulent discharge from the nostrils, malnutrition, and well-marked condylomata round the anus. The Wassermann reaction was found by Dr. Woodforde, Pathologist to the Hospital, to be markedly positive, and on October 30 she was given a dose of 0·45 grm. neo-salvarsan intravenously into the median basilic vein. The neo-salvarsan was dissolved in 75 c.c. of 0·4 per cent. saline solution at room temperature, the water having been freshly distilled. The patient's temperature did not rise above 99° F., and although the dose was a considerable one for a child of this age, no ill-effects of any kind followed the injection. The child left the hospital two days afterwards with the syphilitic symptoms greatly improved, and a week afterwards the condylomata and muco-purulent rhinitis had disappeared.

The child was again taken into the hospital, and on November 13 a second injection of neo-salvarsan was given, this time intramuscularly into the right buttock, some minutes after the injection of 5 c.c. of a ½ per cent. novocain injection, the cannula being left in situ until the neo-salvarsan injection. This was followed by the injection of a few cubic centimetres of normal saline solution. The amount of neo-salvarsan so injected was again 0·45 grm., dissolved in 9 c.c. of freshly distilled water. The child has steadily improved in general health and has put on weight.

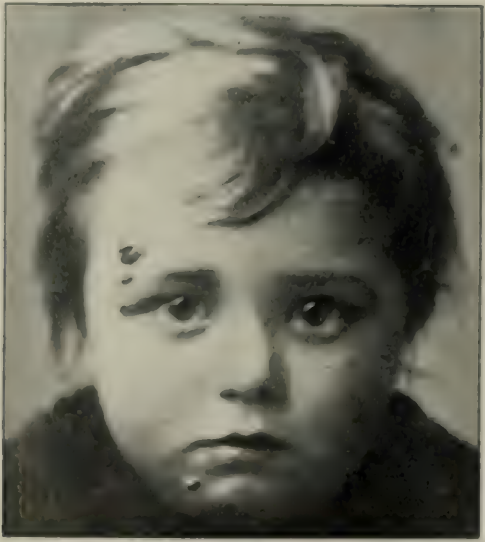
The Wassermann reaction is still positive.

The youngest child which I have treated by intravenous injection of salvarsan was 8 weeks old.

Case of Molluscum Contagiosum.

By J. L. Bunch, M.D.

Some weeks ago two or three small, shiny, raised lesions showed themselves on the right cheek of the patient, a boy, aged 2 years. These small swellings gradually increased in size until they resembled a split-pea, with a semi-translucent appearance, and an umbilication in the centre. Other similar lesions followed, also on the same cheek, and also one above the right orbit, which rapidly increased in size. This soon showed signs of redness at the base, and formed a well-marked projection about the size of a hazel-nut. The central depression was clearly defined in this swelling, as in all the others at first, but as the redness increased a firm, yellowish substance was extruded from it, which differed entirely from the translucent contents, containing typical molluscum bodies which could, by pressure, be squeezed out of the other lesions on the cheek. None of the swellings were apparently painful, but the lesion above the orbit was tender to pressure. There were no lesions elsewhere and no evidence of inoculation could be obtained.



Case of molluscum contagiosum.

Case of Partial Hemiatrophy of the Face and Tongue.

By J. WALTER CARR, M.D.

W. A., MALE, aged 9 years. About a year ago patient's mother first noticed a whitish spot, like a scar, over the lower border of the lower jaw on the right side. Since then a gradual wasting of the lower part of the right side of the face has been observed. No cause can be assigned for the onset of the atrophy; there is no history of illness or of trauma. At present there is marked atrophy of the skin, subcutaneous tissues and muscles over and below the right half of the lower jaw, from the angle to the symphysis. The bone itself also appears to be somewhat wasted and the lower teeth are displaced a little towards the right, as if pushed over by the larger size of the left half of the lower jaw, but an X-ray examination does not show any marked atrophy. The skin shows slight, rather patchy atrophic changes; its sensibility is unaltered, except perhaps for a very slight diminution to touch. The muscles contract normally, those of mastication are not affected. There is marked atrophy also of the right half of the tongue, but no affection of movement, ordinary sensibility, or taste. In all other respects, except for slight psoriasis, the boy seems quite normal, both physically and mentally.

DISCUSSION.

Dr. F. PARKES WEBER alluded to the presence of sclerodermia below the jaw. The association of sclerodermia with hemiatrophy of the face or with hemiatrophy of the whole body was of great interest. Some cases of sclerodermia with hemiatrophy of the body had been brought forward recently.[1] The association could not be a mere matter of chance.

Dr. JEWESBURY asked if there was any eye change in this case. He showed a case of hemiatrophy of the face, not long ago, with very well marked narrowing of the palpebral fissure, and there was also a coloboma of the choroid on the affected side.

Dr. LANGMEAD thought the profession had drifted away from the original description of sclerodermia, which Addison described as being a disease of both skin and muscle. Attention had of late been concentrated on the skin condition, largely to the exclusion of the muscle involvement. He concluded that this case was one of localized sclerodermia, with an uncommonly widespread wasting of muscle as compared to the skin condition.

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[1] *See* P. C. Knapp, *Proceedings*, 1911, iv (Neur. Sect.), p. 28; and Wilfred Harris, *Trans. Med. Soc. Lond.*, 1911, xxxiv, p. 440. The association of sclerodermia with hemiatrophy of only the face is much better known, and the sclerodermia need not be limited to the side of the hemiatrophy.

Dr. CARR replied that the child's eyes were quite normal. He feared he was not able to answer Dr. Parkes Weber's question; in fact, he was anxious to hear the views and opinions of others, as he had not seen a case of the kind before, and therefore had no personal experience to guide him.

Case of Spina Bifida (Meningo-myelocele ulcerated) in a Child treated by Operation.

By LIONEL E. C. NORBURY, F.R.C.S.

C. D., FEMALE, aged 5 weeks, was admitted to the Belgrave Hospital for Children on August 8, 1911, with an ulcerating sacral spina bifida, the size of a large tangerine orange. Very little true skin over the swelling, the covering consisting chiefly of a thin membrane, discharging in several places. General condition of child very unsatisfactory. Anterior fontanelle large and tense; slight degree of hydrocephalus; talipes calcaneus bilateral; no other paralyses. Ulcers healed under treatment with ung. sorbefacin, and then an operation was performed on September 8, under chloroform anæsthesia. Excision of spina bifida by elliptical incisions: Many nerves found attached to the middle of the sac; portion of sac with the attached nerves dissected off and replaced in spinal canal; dura sutured over them; skin edges approximated; lateral incisions made on either side to relieve tension. Rubber tube stitched into rectum to avoid soiling of wound, but the tube did not remain in position very long. Child kept in a slanting position with the head low, until the wounds had healed. Healing of middle incision by first intention, and of lateral incisions by granulation. There was never any leakage of cerebrospinal fluid during convalescence.

The child is now 16 months old. She is intelligent. The anterior fontanelle is almost closed. There is no bulging in the region of the wound. The condition of talipes calcaneus remains, and is being treated at present by massage and passive movement by the mother.

DISCUSSION.

Mr. NORBURY added that the child had an appreciable amount of hydrocephalus, and the usual result from operating for spina bifida was to increase the hydrocephalus. But this child, apparently, now had a normal head, and the fontanelles were almost closed, although it was only 16 months old. Since the operation there had been no leakage of cerebrospinal fluid.

The PRESIDENT (Mr. A. H. Tubby) said he could not give a reason for the disappearance of the hydrocephalus, but he thought that in some cases in which it had disappeared during comparatively early life after the cure of spina bifida it had come on again later, and had ultimately caused the death of the child. He believed the hydrocephalus condition might come on up to the sixth year. He congratulated Mr. Norbury on his excellent result, which was one of the best he had seen.

Mr. DOUGLAS DREW said that he had performed the operation a number of times for spina bifida, and the majority of the cases were followed by hydrocephalus. He knew of a few successful cases, but they were so rare that he felt almost disinclined to operate in cases of this kind.

Case of Ununited Fracture of Neck of Femur.

By H. A. T. FAIRBANK, M.S.

A GIRL, aged 15, giving a history of having been knocked down and run over by a van in October, 1904. The diagnosis is said to have been "comminuted fracture of femur near the neck." The left leg has always been short since the accident, which necessitated her lying in bed many weeks. The shortness is said to be increasing. Pain has been present at times only, but has been worse lately. The pain is severe at night, it wakes her; it is particularly noticeable on rising after sitting for long, but it is not at other times caused by walking, except in wet weather. The left leg is generally wasted, and is held in an everted position. Flexion of the hip is possible to a right angle only; abduction is practically abolished, while internal rotation is markedly limited; extension and adduction are only slightly limited. The trochanter is raised, prominent, and thickened anteriorly. Real shortening, 2 in. A skiagram shows an ununited fracture of neck of femur and coxa vara.

The case is shown in order to elicit opinions as to the best treatment. It is proposed to excise the head of the femur, and retain the limb in hyper-abduction for several weeks.

DISCUSSION.

Mr. FAIRBANK said he hoped to hear advice as to what should be done for the child, and whether the head of the femur was of any use. If one operated and tried to fix the trochanter to the head with a screw, would it be possible to bring about union? Or would any member suggest another form of treatment?

Mr. DREW said his feeling would be not to excise the head of the femur in such a case, except as a *dernier ressort*. Having exposed the trochanter freely, and separated all the muscles from it, he would attempt to draw down the bone. Perhaps it would be necessary to tenotomize the hamstrings, and possibly the tensor vaginæ femoris. He was presuming the shaft had been separated from the neck and the surfaces freshened. It might be then possible to pass long screws through the trochanter into the neck of the bone, and so secure it in good position. He did not regard the case as a hopeful one. Removing the head and neck of the bone he thought would be unfavourable; it would be difficult even to place the top of the trochanter into the acetabulum in the abducted position.

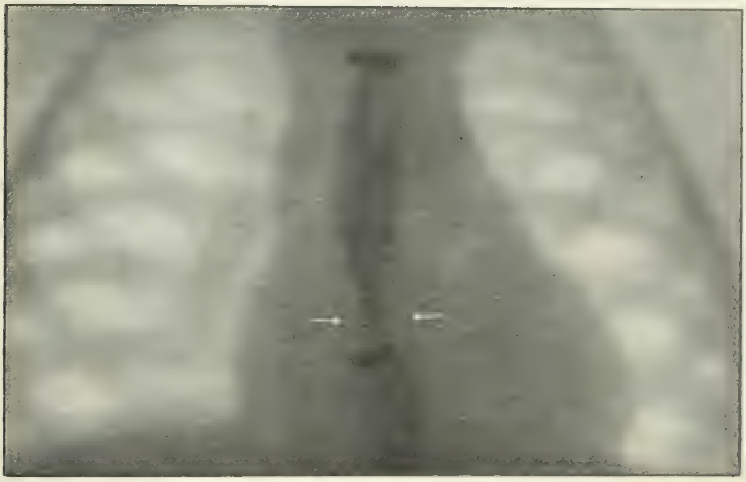
Mr. CLOGG pointed out that there was to be seen in the skiagram some irregularity in the head of the femur, suggestive of osteo-arthritic changes, and he thought these might be the cause of the pain from which the patient suffered. The pain seemed to be very severe, and he assumed that one object in operating would be to give relief from pain. After eight years he doubted whether it would be possible, however much one tenotomized, to pull the trochanter down; nor did he think the head of the femur would hold a screw. If satisfactory union were obtained in this way he did not think pain would be relieved. He thought the best result would be obtained by excising the head of the bone and putting the limb up in abduction.

The PRESIDENT said he had had to deal with this condition on four occasions, but not in so young a subject, nor so long after the accident, the longest time being one year. In those cases he cut down freely on the neck of the femur, and refreshed the surfaces of the bone thoroughly. He had previously had a skiagram taken, and procured in readiness a screw of the proper length—i.e., one which would reach from the outer side of the great trochanter obliquely up through the neck, and finally imbed itself in the head below the cartilage of the articulation. After exposing and refreshing the bony surfaces the leg was pulled upon vigorously, and it could be got down satisfactorily, even in adults, by pulleys. Then he put in the screw. The difficulty in these cases arose if there was much atrophy of bone present. X-ray pictures of Mr. Fairbank's case showed that the upper part of the head of the femur was not atrophied, and the upper two-thirds of the head was of fair density, so naturally one would drive the screw into that. He advised the above procedure in this case. He did not think the pain here was due so much to the osteo-arthritic condition, but to static causes—i.e., the strain on the parts due to the abnormal position, and on parts which were constantly changing in shape. If the leg was drawn down and the operation done under the influence of stovaine the muscles would helpfully relax. If that operation should fail, he felt he would still resort to excision of the head of the femur, and putting the limb in a position of abduction. He asked Mr. Fairbank to show the case again later.

Case of Congenital Œsophageal Stenosis.

By Eric Pritchard, M.D., and Douglas Drew, F.R.C.S.

The patient is a boy just over 2 years of age. He was brought to the Queen's Hospital on September 11, 1912, for constant vomiting. The family history is negative; there are two other children, one older and one younger, but both healthy. The child was healthy at birth, but vomited from the first. He was first breast-fed, then was given barley-water and milk, and then Glaxo, but was equally sick with each variety of food. The vomiting sometimes occurred at once, sometimes after one or two hours' delay. The quantity sometimes vomited was very large. During this time the infant made very slow progress. At about the fifteenth month the mother first tried to give the infant solid food, but this always caused difficulties in swallowing and started vomiting, which continued for some time afterwards, even when only liquid food was given, but the vomiting usually ceased when all the solid food taken had been returned. These symptoms continued from the fifteenth month until the infant was brought to the Queen's Hospital,



Congenital œsophageal stenosis.

although the infant had been subjected to a variety of lines of treatment at different hospitals; the treatment being directed, according to the mother's account, to the stomach.

The present condition shows one of greatly retarded development; the child can sit up, but can neither walk nor stand. The intelligence seems to be distinctly below normal, and it is quite a question whether the child is not mentally defective. The weight of the child on admission was 17 lb. 12 oz., and the weight is now 18 lb. 4 oz.

An X-ray examination after the administration of a few spoonfuls of gruel thickened with bismuth showed considerable dilatation of the œsophagus, between points on the front of the chest which lie ¾ in. below the suprasternal notch above and about 1 in. above the xiphisternum (*see* figure). Below the dilated portion there is a narrow portion of about 1 in. in length. Bismuth emulsion of thin consistence can pass directly into the stomach without delay. When, however, the emulsion is thickened, a thin streak of bismuth is seen to connect the upper dilated portion of the œsophagus with the bismuth shadow of the normal œsophagus below.

The case therefore appears to be one of stricture of the œsophagus at the level above indicated. The history would seem to indicate that the stenosis is congenital in origin.

Cases of congenital stenosis of the œsophagus are exceedingly rare. We can only discover the record of nine previous cases; seven of these are mentioned in Von Ziemssen's "Encyclopædia"; then in the year 1885 F. C. Turner[1] records a case in a child aged 18 months; then comes a case by B. H. Rogers,[2] of Bristol, in 1904; and in 1905 another case reported by Whipham and Fagge.[3] In most of the cases in which a complete history is recorded the symptoms began at the beginning of the second year when solid food was first taken. Two cases recorded in Von Ziemssen's "Encyclopædia" lived to the ages of 74 and 84 respectively; but in all the three recent cases—namely, Turner's, Rogers's, and Whipham and Fagge's—death occurred at an early age, after unsuccessful attempts at dilatation.

[1] *Trans. Path. Soc. Lond.*, 1885, xxxvi, p. 185.

[2] *Brit. Journ. of Child. Dis.*, 1904, iv, p. 390.

[3] *Lancet*, 1905, i, p. 22.

DISCUSSION.

Mr. DREW said he had not attempted to dilate the stricture: he would do so, as there seemed no other course, but it did not look hopeful. It was too high to be treated by abdominal section from below. He would have it examined by the direct method before attempting dilatation.

Dr. LAPAGE said that he had now under his care a child with very similar symptoms. The patient was 2 years old, and quite well developed and bright. Every time she attempted to take solid food there was intractable retching and vomiting, and she would take nothing but bottle food. It was difficult to test such a case by the X-rays because she could swallow fluid food readily, but could not take solid or semisolid food without such retching and disturbance that a screen examination was out of the question.

Dr. WHIPHAM suggested to Dr. Lapage that his case could be tested with X-rays if he passed a soft annealed wire in a catheter. He could then see where it folded on itself, and whether there was any dilatation of the œsophagus above the stricture. He had done that successfully in a case of his own.

Dr. JEWESBURY asked as to the character of the vomiting in this case, and how soon after food it occurred. Also, was there any swelling in the neck noticeable after food to help in the diagnosis?

Dr. PRITCHARD replied that in the early days the infant vomited immediately after it took food, but sometimes it retained an entire meal for one or two hours, and then returned the whole of it. When at the fourteenth month or so it was given solid food it vomited until every particle was returned. Some swelling was apparent in the neck after taking food, and during this time there was cyanosis, and the child was in considerable distress, indicating that the pressure was probably in the pulmonary veins.

Multiple Exostoses with Symmetrical Wasting of the Muscles of both Upper Arms.

By ERIC PRITCHARD, M.D.

BOY, aged 7 years, was brought to Victoria Park Hospital in November, 1912, for cough and debility. On examination the boy was found to have a large number of exostoses on different parts of his body, chiefly symmetrically situated; they occur chiefly on the ribs, upper parts of humerus, left radius, and on both scapulæ. The family history is negative except for the fact that the maternal grandmother is now suffering from "withering palsy" and has the left arm drawn up. Nothing abnormal was noticed in the child until about the age of 3 years, when hard nodules were noticed on the inner sides of the knees, and a little later on the ribs. Since then, up till the present time, the nodules have increased in size and in number, but more especially so during the last few months, during which time the mother states she has noticed a great wasting of both upper arms. Both upper arms appear very thin as compared with the forearms, but the electrical reactions are brisk and there is no reaction of degeneration. The strength of both arms seems good, but the boy easily tires, though not especially in the arms.

The significance of the wasting of the arms is not clear. The X-ray photographs, which show considerable exostoses on the upper third of the humerus, do not appear to show sufficient osseous hyperplasia to account for pressure on the brachial plexus, although such pressure symptoms are not unknown; indeed, pressure on nerves in such cases has been known to produce symptoms comparable to those of syringomyelia.

The ætiology of this condition does not appear to be clear. It certainly runs in families. Teissier and Bénard report three cases in one family in which there was a large exostosis on the internal side of the right knee. The disease is chiefly confined to males, and is hereditary in the direct line. M. Pissarz claims that there is a close relationship between this disease and tuberculosis; in twenty-six cases collected by him there was phthisis in ten of the antecedents, while thirteen of the patients themselves were afflicted with this disease. Piller has pointed out that in one family in which the disease was hereditary the thyroid showed abnormalities in a number of cases. Winkler refers to the absence of pain in these cases and to the common association of symptoms of fatigue on the slightest exertion.

The treatment recommended for these cases consists in the exhibition of thyroid gland to quicken up the processes of ossification, and orchitic extracts to promote nutrition, and radiotherapy to check the proliferation of cartilage cells. The disease also offers a large field for surgical enterprise.

Case of Osteoma of the Forearm causing considerable Deformity.

By SIDNEY BOYD, M.S.

E. E., MALE, aged 9 years. The tumour of the forearm appears to be growing from the ulna, and has caused considerable bowing of the radius. This swelling was first noticed at the age of 5 years. The head of the radius is dislocated outwards. There is imperfect ossification of the lower end of the ulna, and a small tumour at the upper end of the same bone. Patient also has several osteomata on the ribs, one at the upper end of the right humerus, and one on the vertebral border of the left scapula. Father said to have a bony tumour of the tibia. Patient is the ninth of eleven children. An elder brother, now aged 19 years, had a similar but smaller tumour of the forearm; it is now said to be much smaller, and causes no loss of function. Another brother, aged 15 years, has a similar tumour, but it causes him very little inconvenience in his work as typewriter. A sister, aged 6 years, also has multiple osteomata.

Case of Cerebral Non-development.

By G. A. SUTHERLAND, M.D.

F. G., FEMALE, aged 7 weeks. Patient was born at full term and seemed healthy for the first fortnight, taking the breast well. At the end of that time convulsions came on in the form of irregular twitchings of face and limbs. Feeding was changed to Nestlé's milk, and difficulty in swallowing was noticed. The breathing at times was rapid.

Baby fairly well nourished, the skull and other parts of the body appearing to be proportional in size. The child lay in a completely apathetic condition, without smiling or taking any notice when awake. Could be roused to make certain gross movements of the trunk and limbs when disturbed, but seemed insensitive to pin-pricks. There was marked opisthotonos, with a tendency to left-sided pleurothotonos, and the limbs were in a spastic condition, flexion being present at the elbows, and extension at the knees. The hands and feet were in a condition of tetany. Other symptoms: (1) As a rule unable to suck or swallow;

(2) attacks of tachypnœa, the respirations running up to 160 per minute, and being cyclic in character; (3) attacks of tachycardia, the heart-rate running up to 200 per minute; (4) fits of crying, causeless, beginning and ending abruptly; (5) spells of yawning; (6) occasional twitching about the face and hands, but no definite convulsive seizure. Fundi normal. Wassermann test negative. Cerebrospinal fluid scanty but normal; temperature normal.

It is suggested that the underlying condition is one of non-development of the cerebral lobes, and that only the cerebellum, pons, medulla, and basal ganglia are active. The uncontrolled action of these lower centres will explain most of the symptoms present.

DISCUSSION.

Dr. SUTHERLAND suggested that this patient was an example of a somewhat rare condition—namely, absence of the greater part of the cerebral hemispheres from non-development. The following were the clinical symptoms by which the condition might be recognized: (1) The occurrence of twitching about the hands and face within a few weeks of birth, and progressing towards persistent spasm and rigidity of the extremities and trunk. (2) An absence of any signs of consciousness, of emotional expression, and of sensation. (3) Recurring attacks of tachycardia, of tachypnœa, and of pyrexia. (4) Recurring attacks of crying (causeless, expressionless), of coughing, and of yawning. (5) Power of sucking and swallowing absent or much diminished. In the one autopsy on a case of this condition which he had seen the cerebral hemispheres were represented by a small part of the occipital lobe, while the cerebellum, pons and medulla, and basal ganglia appeared to be normal. The walnut-like brain lying on the floor of the skull was surrounded by œdematous pia mater, by clear fluid, and by thickened and hæmorrhagic dura mater. These infants would appear to pass an automaton-like existence influenced entirely by the functional activity of the basal parts of the brain. The lower centres were without any controlling influences (stimulating or inhibitory) from higher centres. They were subject to disturbance (lower level fits of Hughlings Jackson) from the irritation caused by the surrounding excess of fluid (external hydrocephalus) and the pachymeningitis which were present. Hence arise peculiar and irregular disorders of the automatic centres, which were manifested clinically by inability to swallow, by tachycardia, by tachypnœa, and by muscular spasm. The type of affection would appear to be sufficiently well marked to enable one to differentiate the condition from that of chronic tetanus neonatorum, or meningeal hæmorrhage occurring at birth.

Dr. H. D. ROLLESTON referred to a specimen of cerebral aplasia with hydrocephalus from a child aged 4 weeks, with similar clinical symptoms.

shown by Dr. Trevor and himself[1] before this Section during Dr. Sutherland's presidency. During life the symptoms, among which yawning was prominent, rather suggested tetanus neonatorum. In spite of the considerable hydrocephalus, the child's head was not enlarged, measuring 13½ in. round the base.

Dr. F. PARKES WEBER said that recently he was present at the post-mortem examination on an infant whom he had seen several times during life, when it was nearly always in a rigid condition with some retraction of the neck. There had also been auscultatory signs of congenital heart disease. At the necropsy large portions of both cerebral hemispheres were found wanting, the position of the absent portions being represented by cavities filled with cerebrospinal fluid and freely communicating with the lateral ventricles. The first portion of the pulmonary artery was found to be very minute, the blood having been evidently conveyed to the lungs through the aorta and a widely open ductus arteriosus. He hoped the case would be published. In Dr. Sutherland's present case he (Dr. Weber) did not think that the tracings indicated any true Cheyne-Stokes breathing, but only a kind of grouped respirations, without the *crescendo* movement characteristic of true Cheyne-Stokes breathing.

Dr. SUTHERLAND, in reply, said he brought the case forward as a clinical type of condition which could be recognized during life. There seemed to be certain features which pointed to the condition of cerebral aplasia, and Dr. Rolleston's remarks seemed to confirm this. He was not prepared to give a definite opinion as to the ætiology, and possibly different causes might be present in different cases. The brain in the other case he had referred to had been removed for examination, but had been lost. The fontanelle was open in that case—and it usually was—and there was no marked distension of the skull, although there was external hydrocephalus. The bones of the skull got firmly ossified in the ordinary way as the child grew.

[*Addendum.*—The infant died a week later, and a condition of external hydrocephalus, with thickened pia-arachnoid and small brain, was found at the necropsy. A detailed report will be given later on.]

Case of Malformation of the Rectum (Complete Absence of the Post-allantoic Gut and the Proctodæum); Operation; Result.

By H. S. CLOGG, M.S.

BOY, aged 4½ years. On April 7, 1908, when 5 days old, he was admitted to the Evelina Hospital. The abdomen was greatly distended; vomiting was frequent; there was entire absence of development of the proctodæum, without any indication where the bowel terminated. Immediate colostomy in left iliac region performed. He left the hospital on May 5, 1908.

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[1] *Proceedings*, 1911, v, p. 49.

October 10, 1908 (when he was 6 months old): An exploratory operation was performed from the perineal aspect. The bowel ended blindly at the level of the base of the prostate gland, to which it was adherent: it was separated from the prostate and sufficiently mobilized to enable it to be brought to the perineal skin without tension, to which, after opening, it was sutured. To render access easier a portion of the coccyx was removed. In order to mobilize the bowel the peritoneum had to be freely incised, and several resistant peritoneal and connective tissue bands had to be severed. The wound healed satisfactorily without any retraction of the bowel.

December 15, 1908: The colostomy was closed.

At the present time the bowel is seen to be united to the skin of the perineum; there is no stenosis nor prolapse; the motions are passed unconsciously; there is complete absence of sphincteric control.

The case is shown to illustrate (1) the advantages of an immediate primary colostomy over a perineal dissection in cases where the bowel ends at some distance from the perineum, for a deep perineal dissection in an infant, a day or two old, suffering from obstruction must be extremely difficult and probably fatal; (2) that in order to establish a perineal anus the bowel must be freely mobilized and brought to the perineal skin without tension, or very troublesome stenosis will result; and (3) the absence of sphincter control which is to be anticipated, since the sphincters are developed from the proctodæum. In this case there were a few fibres in the position of, and having the direction of, the external sphincter muscle, but as a muscle it could not be said to be developed.

DISCUSSION.

Mr. Drew congratulated Mr. Clogg on his brilliant result. The operation was one of great difficulty, especially in a child so young. He questioned whether the result from the patient's point of view was better than an artificial anus (colostomy), as the child had no control over it.

Dr. Eric Pritchard said he knew of a case of total excision of the rectum with artificial anus situated at the position of the coccyx, in which the patient was far more comfortable as regards his daily motions than he had been before the operation. The patient had been provided with a very well adjusted pneumatic plug, which was re-inserted and blown up every morning after the bowels had acted. The difficulty was, that before the plug had been properly adjusted he could not go into society, because the escape of flatus played disconcerting notes on the instrument. He now had comfort, however. For a hospital patient the expense was a consideration, as the plugs were perishable.

Mr. CLOGG replied that colostomy in an adult, when one could choose one's own time and there was no obstruction, could be performed so as to give a fairly satisfactory functional result, and if such could be obtained in a baby, the child would be quite as well off as with a perineal anus. But in a baby 5 days old, suffering from obstruction and a very distended abdomen and thin abdominal walls, it would be very difficult to perform a satisfactory iliac colostomy. If the health of the child permitted, he thought it was one's duty to do a perineal exploratory operation to ascertain the condition, and if possible to form a perineal anus, in the hope that this would be better than an iliac anus. He would see what could be done in the direction indicated by Dr. Pritchard's remarks.

Case of Cerebellar Ataxia.

By O. K. WILLIAMSON, M.D.

H. R., AGED 10 years, was brought to the hospital in April, 1912, on account of tremor of the head. The illness began three and a half years before this, when it was noticed that his hands trembled and that he had lost strength in them. After this a fine tremor was noticed in the head. Since this time he has been, on the whole, less bright mentally. He gets more easily excited than before, is spiteful, and has fits of bad temper, which is abnormal to him. Since the beginning of the illness his sight has been affected and his gait unsteady, and he frequently falls. He improved for a time, but has been worse again of late.

Family history: Mother's sister suffered from fits, otherwise there is no history of nervous diseases. In-patient in January, 1909, under Dr. Coutts. The notes state that there was then no optic neuritis; a pronounced ataxic gait; he staggers, especially to left side; sight bad; speech rather slurring, indistinct; slight nystagmus on lateral deviation of eyes.

State (April, 1912): Coarse tremor of arms, especially left, also of tongue; gait ataxic; knee-jerks brisk; no ankle clonus; plantar reflex is flexor. Power of hand-grip rather diminished; no marked weakness of legs; speech somewhat indistinct; no nystagmus; cranial nerves otherwise normal. No sensory changes; no headache or vomiting.

November, 1912: Coarse tremor of arms, equal on the two sides, also of tongue; fine tremor of head; gait ataxic and reeling; he tends to fall to the left side; Romberg's sign absent; pupils react well to light and accommodation; hearing normal; doubtful slight lateral nystagmus; knee-jerks normal, no definite plantar response. Signs and symptoms otherwise as in April. Is now in second standard at school.

Dr. WILLIAMSON said that at one time the diagnosis was thought to rest between cerebellar ataxia and juvenile general paralysis. But the condition had gone on so long without progressing markedly in a mental sense that general paralysis could now be ruled out of court.

Pharynx and Larynx from Fatal Case of Hæmorrhage from Throat.

By J. D. ROLLESTON, M.D.

GIRL, aged 6 years, was admitted to hospital on October 22, certified to be suffering from diphtheria. She had had measles five weeks previously, since when her voice had been husky. On October 20 she complained of sore throat, and on October 22 she had a croupy cough and her neck glands became swollen.

Condition on admission: Deposit on both tonsils and uvula, slight nasal discharge, voice husky, stridor, croupy cough and dysphagia. Temperature 100° F. Sixteen thousand units of antitoxin given.

October 23: Deposit clearing away, leaving marked ulceration of tonsils and uvula. No fœtor. No Vincent's organisms in throat smear. Temperature 103·2° to 100° F.

October 25: Sudden and profuse hæmorrhage from throat, cyanosis, and death within five minutes. No diphtheria bacilli, but only cocci, were found in three successive cultures from throat and one from nose.

Specimen shows abscess cavity in each tonsil, ulceration of the uvula, soft palate, epiglottis, frænum epiglottidis, valleculæ, and aryepiglottidean folds. Deep ulceration of laryngeal portion of pharynx exposing muscular tissue. Three small superficial ulcers above right vocal cord. Exact site of bleeding vessel not determined, but no evidence of erosion of carotid or internal jugular, or of any glandular abscess.

Apart from its occurrence in connexion with tracheotomy, death from asphyxia caused by entrance of blood into the air passages is so uncommon in children that the present case is one of unusual interest. A few cases of fatal hæmoptysis in young children from pulmonary tuberculosis have been recorded (Magruder), but there was no evidence of that disease in the present case. Erosion of the neck vessels giving rise to fatal hæmorrhage is a very rare occurrence, and practically occurs in only two conditions, scarlet fever and gangrenous angina. In scarlet fever, erosion of the external carotid has been recorded by Baader, Huber, and Oppikofer, of the internal carotid by Baader, Hynes, and Griffiths and Riddell, of the lingual artery by Ghon and Oppikofer, and of the internal jugular vein by Henoch, and Griffiths and Riddell. Hecker has recently published a case in which, as in my own case, the bleeding vessel was not discovered, but the branches of the superior laryngeal artery or the tributaries of the internal jugular vein were regarded as the probable source of the hæmorrhage. Mr. A. R. Tweedie showed a specimen at the Laryngological Section on November 1, 1912, of ulceration of the internal carotid in a child aged 1 year 8 months, following a sore throat, which may possibly have been of scarlatinal origin, especially as the patient's brother had previously had some swollen submaxillary glands.

In cases of this kind the fatal hæmorrhage may occur spontaneously, as in the present case, or be due to opening a hæmatoma under the mistaken belief that it was a tonsillar or cervical abscess, as in cases reported by Huber, and by Griffiths and Riddell. This mistake is all the more likely to be made as suppurative cervical adenitis may occur at any period of scarlet fever, and in some cases the erosion of the vessel is secondary to a cervical abscess or cellulitis. There was no history or evidence of scarlet fever in my own case, but it is highly probable that the pharynx and larynx were predisposed to infection by the recent attack of measles, especially as the child's voice was still husky when the fatal illness began. Death from pharyngeal hæmorrhage may also be one of the terminations of gangrenous angina which may be either a primary condition or be secondary to other diseases besides scarlet fever. My case, however, did not show the fœtor and prostration characteristic of this disease, nor the clinical and bacteriological features of the closely allied condition—Vincent's angina. It should rather be regarded as an instance of ulcerative sore throat to which the name pseudo-diphtheria is sometimes applied (Welch and Schamberg). This form of sore throat is usually due to streptococci, is often accompanied by laryngeal involvement, especially when it is secondary to measles, and frequently runs a severe course. Thus of thirty-five cases recently reported by Dr. Goodall twenty-five were fatal. In nine of his cases the larynx was involved, though the principal lesions, as in my case, were in the pharynx, and two had recently had measles. In none, however, was death due to sudden hæmorrhage from the throat. The bleeding vessel in the present case was not identified, and as I was not present when the hæmorrhage occurred I am not prepared to say whether it was venous or arterial. It is probable, however, that the laryngeal portion of the pharynx in which the ulceration is deepest was the site of the hæmorrhage.

Before making the post-mortem examination I thought that death might have been due to erosion of the internal or external carotid or internal jugular. These vessels, however, were found to be intact. The internal organs, moreover, were not blanched as in death due to hæmorrhage, but engorged, and the blood in the bronchi and lungs, the acute vesicular emphysema and distended right heart, showed that death had been due to asphyxia. Further, after opening of a large vessel death usually occurs within a minute, whereas in death from asphyxia due to erosion of a smaller vessel the agony is more protracted.

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Dr. E. W. Goodall said his interest in the case lay not so much in the occurrence of the hæmorrhage as in the ætiology. Hæmorrhage was very rare in these deep ulcerations of the throat. Though he had had an extensive experience in connexion with scarlet fever, he could not recall a case fatal from hæmorrhage from the fauces. He had met with cases similar to Dr. Rolleston's (except for the hæmorrhage); he showed one at the Hunterian

Society some years ago. The nature of the condition was an important matter in fever hospitals, for it was a question as to which wards the cases ought to be placed in. Occasionally the disease seemed to be scarlet fever, but in many it was not. One of his patients had diphtheria, followed in two or three weeks by scarlet fever. A few weeks later ulceration of the fauces supervened, and proved fatal. Probably no constant organism was the cause of this affection. Two or three cases had been associated with Vincent's organisms. Taken altogether these cases constituted a distinct clinical group. Usually they were mistaken for diphtheria.

Case of Primary Carcinoma of the Liver in a Boy, aged 6 years.

By C. Paget Lapage, M.D.

Five months before death the boy had an attack of scarlet fever with nephritis. He recovered, but about two months later, when he again came under medical observation, he was suffering from general debility and wasting; he had complained of pain in the region of the right shoulder. The liver was considerably enlarged, with several rounded prominences on its anterior surface. The marked and progressive engorgement of the abdominal veins and the ascites showed that there was obstruction to the venous return in the region of the liver. There was no jaundice. The post-mortem examination showed that the liver was very large, weighing 6 lb., the right lobe being chiefly affected by large masses of tumour which were also growing into and obstructing the hepatic veins and inferior vena cava; there were secondary deposits in the lungs, but nowhere else. Microscopical examination of the tumour shows polygonal and spheroidal cells in masses and columns, with very little stroma; the cell nuclei are oval or rounded and show many mitotic figures, and the cytoplasm is granular; there is much fatty infiltration.

The specimens show the tumour masses in the liver and the secondary deposits in the lungs. Note the bile-stained areas in the tumour masses and in the lung deposits, but not in the liver itself. The slides show the microscopic appearances of the tumour nodules in the liver, lung and veins, and the fatty globules in the cells and cell nuclei. According to Dr. Mair, Pathologist to the Children's Hospital, Pendlebury, the evidence for regarding this tumour as a primary carcinoma of the liver is twofold—viz., (1) the general resemblance of the cells to liver cells in form and arrangement, and (2) the resemblance in physiological function, as shown by the infiltration of fat and secretion of bile in both primary and secondary growths.

DISCUSSION.

Dr. LANGMEAD referred to the case described below which somewhat resembled this. Post mortem the liver showed multiple adenomata.

Dr. H. D. ROLLESTON agreed with Dr. Langmead about the difficulty in distinguishing between adenoma and carcinoma of the liver, especially in the case of multiple adenoma with cirrhosis: but in Dr. Lapage's case, in which there were secondary growths in the lungs, there was no question of this kind. The occurrence of bile in the secondary growths was a notable feature of this case. He had seen it in adult cases, but not in a child.

Dr. F. PARKES WEBER said it was only comparatively recently that this group of primary carcinoma of the liver, in which carcinoma cells were supposed to secrete bile had been recognized. He gave an account of such a case a little time ago.[1] He had since heard, however, that recent observations threw some doubt on the substance secreted by the cancer cells in question being actually bile or bilirubin.[2] But that, he thought, was of secondary interest, because if it were not bile or bilirubin it contained a substance closely allied to bilirubin.

A Case of Congenital Adenoma of the Liver.

By FREDERICK LANGMEAD, M.D.

THE patient was a girl baby, aged 5 weeks, who died in convulsions, associated with diarrhœa and vomiting and pneumonia. Nothing abnormal had been noticed about the baby until the onset of diarrhœa a few days before death. No signs indicated liver disease. At the autopsy the liver was seen to be slightly enlarged and fatty. Eight pearly-white tumours projected slightly from its surface. They were not depressed, and showed no puckering. Each was firm and showed no areas of softening (fig. 1). They were clearly defined from the surrounding liver, but not encapsuled, and were of the same white colour throughout. The largest was the size of a marble, others were nearly as large.

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[1] F. P. Weber, "A Case of Bile producing Primary Malignant Tumour of the Liver." *Proceedings*, 1910, iii (Path. Sect.), p. 147; also *Lancet*, 1910, i, p. 1066.

[2] *See* Beattie and Donaldson, "Primary Carcinoma of the Liver," *Journ. Path. and Bact.*, Camb., 1912, xvii, p. 32.

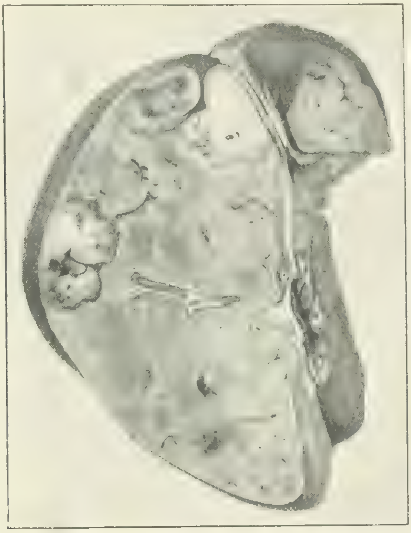

FIG. 1.

Liver showing the sharply defined growths (congenital adenomata).

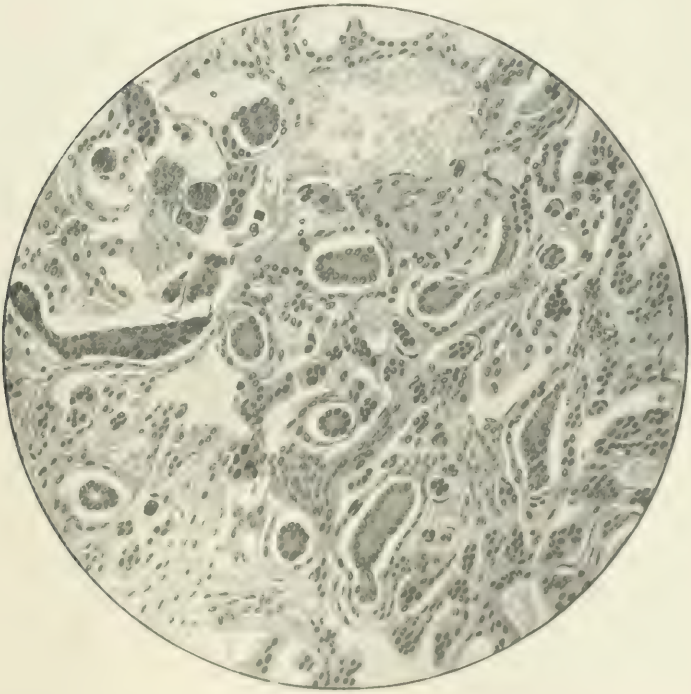

FIG. 2.

Microscopical section of a growth, showing the tubular structure and the blood spaces.

Microscopically they are glandular in structure (fig. 2). Numerous tubules are seen, some in vertical, others in horizontal section. Those seen cut across transversely are lined by columnar cells which surround the lumen. The tubules are supported by connective tissue which is rich in blood-vessels. Blood spaces are noticable also at the margin of the growth. There is no tendency to infiltrate the liver, on the contrary the tumours are sharply outlined. The whole structure resembles somewhat the renal cortex, except for the absence of glomeruli. The condition is clearly one of adenoma arising from the bile canaliculi, in that it is a tubular adenoma of the liver and apparently congenital. It is an example of a condition of extreme rarity. I am indebted to Dr. F. E. Batten for permission to show this specimen.

Epidemic Catarrhal Jaundice.

By LEONARD GUTHRIE, M.D.

SPORADIC cases of catarrhal jaundice in children are not uncommon, especially in the winter months, and they occur more frequently in some seasons than in others. Hence if one sees a few more cases than usual in cold weather, the fact may be regarded merely as a coincidence. The idea that an epidemic of catarrhal jaundice is prevalent, and that the disease may be communicable by one patient to another, does not readily assert itself. More commonly catarrhal jaundice is regarded as an individual ailment, and attributed to "a chill on the liver," to gastro-duodenal catarrh, or to immoderate indulgence at table. When, however, one meets with a number of cases of jaundice in the same district, and when two or more members of the same family are affected at or about the same time, one is bound to presume that some cause of infection common to all is at work; in other words, that one is in the presence of jaundice in an epidemic form.

Had it not been for the presence of the circumstances mentioned, I should hesitate to describe the small number of cases of catarrhal jaundice—only ten in all—which I met with last winter as instances of epidemic catarrhal jaundice. The ten cases of jaundice to which I refer all occurred during the months November, December, 1911, and January 1912. Eight of them were in the contiguous districts of Paddington, Kilburn Park, Harrow Road, and St. John's Wood, and two were in Walthamstow. Seven of the patients were female and three were male; their ages varied between 3 and 11 years. In three instances more than one member of the same family were affected. In one family, two sisters; in another, two sisters and one brother; and in a third a brother and sister became jaundiced within a fortnight to three months of each other. Thus, seven of the patients were related as brothers or sisters. The remaining three cases were sporadic, and unrelated to each other.

The jaundice was deep in all cases, and associated with clay-coloured stools and bilirubinuria. The average duration was three to four weeks, but in one case it only lasted a week. The icterus was ushered in by malaise, languor, and sometimes drowsiness; the temperature was usually slightly raised (99° to 100° F.). Anorexia was the rule, but in one case the appetite was said to be voracious, especially for eggs. Vomiting occurred in two cases; vomiting and diarrhœa in one; vomiting and constipation in another. In one case complaint was made of itching. Bradycardia was not observed in any. The lowest pulse-rate recorded was 66 in one case; as a rule it varied between 72 and 96. A pulse of 120 to 130 was noted in two cases, whilst the temperature was elevated.

The liver was enlarged in six cases, and greatly so in four; in these it extended to within 1 in. or 1½ in. of the umbilicus; it was smooth in all cases, and not tender in any. As soon as bile appeared in the stools, and disappeared from the urine, the liver slowly subsided, and became normal in size in from one to two weeks. The four cases in which no hepatic enlargement was noted were only seen when the patients were convalescent.

The exact interval which elapsed between the onsets of jaundice in different members of the same family could not in all cases be determined. In one family a girl became jaundiced exactly a fortnight after her sister. In another family a girl became jaundiced on December 4, and her brother on December 16, 1911. Their elder sister, aged 11 years, had had jaundice some time in the previous November. In the third family, a boy, aged 6 years, was jaundiced in November, 1911, and his sister, aged 3 years, became so on January 9, 1912. Apparently all cases began with definite malaise, and probably some slight pyrexia, but none of the patients was seriously ill at any time. In this respect there was no difference between the sporadic and the other cases. The hepatic enlargement was as marked in the former as in the latter. In one of the sporadic cases it extended to within 1 in., in another to within 1½ in. of the umbilicus, and in the third it could be felt 1 in.

below the ribs. In two of the familial cases the liver reached to within 1 in. of the umbilicus, and in all the rest it was more or less enlarged. There seems no reason to doubt that the sporadic cases were of the same nature as those which occurred in families. No relapses occurred in any of the cases, and recovery was uneventful.

Epidemics of catarrhal jaundice have been recorded in many parts of the world. For a full bibliography and complete account of such, reference may be made to an important paper, "Catarrhal Jaundice, Sporadic and Epidemic, and its Relation to Acute Yellow Atrophy of the Liver," by E. A. Cockayne.[1] I must express my indebtedness to this paper for many of the facts mentioned in my own. I wish to mention specially epidemics which have occurred in our own country, and draw attention to the fact that they have been particularly prevalent here during the past three years. Dr. Cockayne's earliest reference is to an epidemic in 1852, almost confined to children living in Birmingham or its neighbourhood, and lasting from September to November. Montagu Champneys described one in East Sussex in 1861, which affected eighteen individuals of all classes, whose ages ranged from 8 to 17 years. In 1863 at least 300 people, both children and adults, were affected at Rotherham; and small outbreaks in all parts of the country have been especially numerous in 1894, 1901, 1910, 1911-12.

In the *British Medical Journal* (vol. ii, 1911)[2] reports of small epidemics of catarrhal jaundice are given by C. H. Miles (Stantonbury, Bucks), July to August, 1911; by R. F. Campbell (Wark-on-Tyne), winter, 1909-10; Eva McCall (Cambridge), winter, 1909-10; W. Brown Holderness (Windsor), October, 1911; Whitaker (Evershot, Dorset), November, 1910, and May, 1911. These epidemics are not mentioned by Dr. Cockayne, but he records an attack of jaundice experienced by himself, which he attributes to infection from a servant who waited upon him, and who herself suffered from catarrhal jaundice. Fowler, of Ellon (East Aberdeenshire), witnessed an epidemic in autumn, 1911. Ralph Pognaud[3] met with ten cases in children, aged 6 to 12 years, in Suffolk. Gale, of Sheffield, attended a brother and two twin sisters, who were attacked by jaundice in September and October, 1910. D. H. Vickery, in the *British Medical Journal* for October 5, in this year,[1] gives an account of several cases, which occurred in scattered villages in the neighbourhood of Cheriton Fitzpaine, Devon.

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[1] *Quart. Journ. Med.*, Oxf., 1912, vi, pp. 1-29.

[2] *Brit. Med. Journ.*, 1911, ii, pp. 379, 920, 1533, 1695.

[3] *Ibid.*, 1912, i, p. 72.

In the Stantonbury epidemic, children aged between 4 and 12 years were attacked. The duration of jaundice was short, there were no relapses, and no history of catarrhal gastritis. The obstruction was incomplete for the motions contained bile. Constipation was severe. Pruritus and bradycardia were absent. At Wark-on-Tyne fifteen cases occurred within an area of seven miles. In more than one house two children, and in one house three, were simultaneously affected. The patients were from 4 to 12 years of age, but one was a girl aged 16 years. Her two younger brothers were jaundiced at the same time. At Cambridge seven young children and two lads aged 15 years were attacked. In two instances two members of the same family suffered from jaundice. At Windsor seven cases occurred in a girls' school after the girls had taken a walk on a cold, wet day. Malaise and vomiting followed by jaundice were the chief symptoms. In two cases the liver was enlarged, and in one it was greatly so. At Evershot (Dorset) fourteen cases of jaundice occurred in three villages, between November, 1910, and May, 1911. The patients' ages were from 6 to 30 years. The worst cases were in adults, who suffered from severe epigastric pain and vomiting. The liver was slightly enlarged. The duration of jaundice was from a week to ten days.

EPIDEMIC INFECTIOUS JAUNDICE AND EPIDEMIC CATARRHAL JAUNDICE.

A distinction should be drawn between epidemic infectious jaundice described by Weil in 1886 and epidemic catarrhal jaundice. Weil's disease was also described by Lancereaux in 1882 as "Ictère grave essentiel," by Landouzy in 1883 as "Fièvre bilieuse," or "Typhus hépatique bénin," and by Matthieu in 1886 as "Ictère fébrile à rechutes." Except in degree of severity, the symptoms of Weil's disease do not differ markedly from those of epidemic catarrhal jaundice. The most severe cases of the latter may be very like the milder forms of Weil's disease. Icterus in Weil's disease is associated with urticaria, petechiæ, hæmorrhages from the nose, stomach, and elsewhere, fever, enlargement of the spleen, nausea, vomiting, diarrhœa, and albuminuria. Nervous symptoms, exactly like those of acute yellow atrophy, may end the scene. Relapses are common, and in some cases a remarkable urinary crisis attended by polyuria and excessive excretion of urea takes place on the seventh or ninth day[1] (Kelsch). The mortality varies from 10 to 60 per cent. Post mortem, the changes are those of acute yellow atrophy of the liver, or extreme fatty degeneration with diffuse hepatitis and leucocytic infiltration near the portal spaces. Ætiologically, Weil's disease has been traced to contaminated food or water. In some cases the *Bacillus proteus fluorescens* has been found in the urine and internal organs of patients. Jaeger also found it in ducks which were dying of jaundice on the same water which had been drunk by human sufferers from the same disease. The jaundice is at first acholuric or hæmolytic, like that experimentally produced by toluylenediamine. Later, bile appears in the urine and disappears from the stools.

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[1] *Brit. Med. Journ.*, 1912, ii, p. 907.

In this country *epidemics of catarrhal jaundice* have always been mild in character. No deaths have occurred, but in other countries fatalities have occasionally been recorded: 161 deaths occurred in a total of 22,569 cases amongst the Federal troops of America; Kissel, in the Moscow epidemic, found six fatal cases out of ninety-six cases in children aged between 2 and 4 years. The liver was enlarged in the first four cases, and the spleen in the first two. The liver showed proliferation of the epithelium of the hepatic ducts and proliferation of the bile-ducts themselves at the periphery of the liver lobules. The liver cells in the centre of the lobules were necrosed, and all showed fatty degeneration. In all the fatal cases there were severe nervous symptoms, loss of consciousness, great restlessness, universal twitchings of muscles, and disturbance of heart's action and respiration. In the epidemic in Saxony, nine out of 313 cases died. The patients in seven of the fatal cases were aged from 4 months to 12 years, and two were 60 and 68 years old respectively. In one of the fatal cases the symptoms and state of the liver resembled those of acute yellow atrophy.

From the description given by Cockayne of outbreaks abroad it is obviously very difficult to distinguish between epidemics of infectious jaundice and those of epidemic catarrhal jaundice. The diseases resemble each other in symptoms and morbid anatomy; fatal cases of either are hardly to be distinguished in these respects from acute yellow atrophy or from fatal cases of sporadic jaundice (so-called). Dr. Cockayne cites many instances in which acute yellow atrophy of the liver has affected more than one member of families, and has occurred with such frequency, especially among pregnant women, as to suggest the prevalence of a specific cause. In the case of Weil's disease the infection is probably due to the ingestion of contaminated water and food, but may be due to some biting insect. It is essentially a filth disease, and is chiefly confined to the districts round the Mediterranean Sea, whereas epidemic catarrhal jaundice occurs all over the world. In the case of *epidemic catarrhal jaundice* and sporadic jaundice the cause is unknown, but there is every reason for supposing them to be due to some unknown organisms widely spread, and capable of giving rise to an infective hepatitis. Dr. Cockayne believes the infection to be air-borne, and not, as in Weil's disease, conveyed by food or water. It appears to be most infectious at the time of invasion and not in later stages.

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[1] *Rev. de Méd.*, Par., 1886, vi, 657.

Acute yellow atrophy Cockayne regards as due, in the great majority of instances, to the same unknown organisms acting upon a liver weakened by some temporary strain, such as pregnancy, or having some inherent weakness. This explanation also covers the cases of post-anæsthetic fatalities with jaundice in which the symptoms closely resemble those of acute yellow atrophy, and the pathological changes are of the same nature, although fatty degeneration is more prominent than necrosis of hepatic cells.

The term "catarrhal jaundice," if implying that it is simply due to gastro-duodenal catarrh, the result of chill or over-eating, is, perhaps, misleading. The assumption that catarrhal jaundice is always the result of mechanical obstruction of the ductus communis by a plug of inspissated mucus may be incorrect. Blockage of the main duct, as, for instance, by a gall-stone, does not produce such great enlargement of the liver as was present in some of my own series of cases.

It seems more probable that epidemic jaundice, whether in the severe form known as Weil's disease or in its milder form called catarrhal jaundice, epidemic or sporadic, consists of examples of acute diffuse hepatitis produced by organisms or toxins which may not be the same in all cases. Blockage of the main duct may be secondary to hepatitis, descending and not ascending the duct. C. H. Miles attributed the Stantonbury epidemic, which has been mentioned, either to pneumococcal infection or to invasion by some oral germ. He was able to exclude ptomaine poisoning as the cause, and the milk, water supply, and drains in the neighbourhood were above suspicion. The influenza bacillus has been blamed by some (Meinert, Brünler, and Bonome), and has actually been found by the last named in the blood, spleen, and liver of two children in whom acute yellow atrophy of the liver, together with the *Bacillus coli*, were found after death. But, as Dr. Cockayne remarks, there seem to be no good grounds for regarding catarrhal jaundice as a form of influenza.

Cockayne refers to the possible relationship or analogy between mumps and catarrhal jaundice. The metastases which occur in mumps, and the fact that other glands, such as the testis and pancreas, may be affected, with or without involvement of the parotid, suggest that the infection reaches the parotid or other glands by the blood-stream. In mumps it is possible that the blockage of Stenson's duct may be secondary to inflammation of the parotid itself, and in jaundice the obstruction of the bile-duct may be secondary to hepatitis.

I am indebted to Dr. F. E. Batten for an interesting observation on the association between mumps and catarrhal jaundice in the wards of the Hospital for Sick Children, Great Ormond Street. A nurse in one of the wards developed mumps on December 1, 1911. Between December 7 and December 10 six children in her ward also contracted mumps. On January 2, 1912, another nurse employed in the same ward became feverish, vomited, and developed jaundice on January 8, which lasted till January 17, but she did not contract mumps. A third nurse developed mumps on December 10, 1911, and also suffered from very severe abdominal pain and vomiting, which Dr. Batten attributed to pancreatitis. He is inclined to regard pancreatitis as the connecting link between mumps and jaundice in these cases.

I have already alluded to the Evershot epidemic of catarrhal jaundice in which severe epigastric pain and vomiting were prominent symptoms in adults, and were perhaps due to pancreatitis. Further investigations are needed as to the prevalence of mumps and epidemic catarrhal jaundice at the same time. The connexion does not seem obvious, yet it is possible that mumps, acute pancreatitis and hepatitis (catarrhal jaundice) may sometimes be allotropic examples of one and the same affection.

The occasional association of jaundice with specific fevers, such as intermittent, remittent, relapsing, typhus, enteric fever, diphtheria, and scarlatina, has been observed. Three cases of pancreatitis with jaundice in scarlatina, and one in diphtheria, have been recorded in the *Lancet*, November 9, 1912, by W. L. Goldie,[1] who refers to another case described by Sidney Phillips, of jaundice in a patient suffering from scarlet fever, who also had parotitis and abdominal symptoms suggestive of pancreatitis. The parotitis occurring in specific fevers is, however, probably not due to the specific infection of mumps. Whether jaundice in such cases is toxæmic, or due to a specific hepatitis, or dependent on obstruction to the hepatic duct by swelling of the head of the pancreas, is undecided. The jaundice in infectious fevers is usually described under the heading "Toxæmic," but Roberts ("Practice of Medicine"), and Sidney Phillips both incline to the view that it results from obstruction.

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[1] *Lancet*, 1912, ii, p. 1295.

The chief points to which attention is drawn in this paper are:—

(1) That mild epidemics of so-called catarrhal jaundice have been specially prevalent in this country during the past three years.

(2) That although mild in character hitherto, it is possible that at any time they might become more formidable.

(3) That catarrhal jaundice must be regarded as due to an acute hepatitis resembling mumps in some particulars.

(4) The prognosis in any given case of jaundice in children, whether apparently sporadic or occurring in the course of epidemics, must be guarded; for we have no means of knowing whether it will run a mild and normal course, or terminate as a case of acute yellow atrophy of the liver.

Finally, the question asked by the mother of one of my patients, "Is jaundice catching?" can no longer be answered in the negative, as I think it would have been some years ago.

DISCUSSION.

Dr. H. D. ROLLESTON'S impression was that epidemic catarrhal jaundice was not a specific disease or due to one form only of infection, but that, like bronchitis or a common cold, it might be due to several micro-organisms. In some cases it appeared from the agglutination reactions to be due to *Bacillus typhosus*, and this might occur without any other clinical evidence of typhoid fever—in fact, a local typhoidal infection of the bile-ducts. In some instances the jaundice might be due to epidemic gastro-duodenitis causing catarrhal obstruction in the biliary papilla; in others, again, to epidemic pancreatitis allied to or complicating mumps; and in others to hepatitis. The uncertainty of the prognosis—namely, the usually mild character with occasional death from icterus gravis—was at least compatible with a varied ætiology.

Dr. E. W. GOODALL expressed his regret that this paper came at the end of the meeting, as it raised important questions, not only in regard to ætiology, but in clinical medicine. He did not agree with Dr. Rolleston in regard to the ætiology. He had not had experience of jaundice in epidemic form, but he was acquainted with the literature of the subject, and even this paper did not exhaust the list of the epidemics of jaundice, catarrhal and other. He believed there was a specific fever of which the chief symptom was jaundice, but what relationship that had to Weil's disease was a matter for investigation. He had been interested to hear that Dr. Guthrie's cases occurred in London last winter, for though he had been for years on the look-out for cases of infective jaundice, not till last winter had he met with any. During that season he noticed that there were more cases of jaundice in his wards than he had been accustomed to meet with. Between October and March he had observed fifteen cases, but in only one group was there evidence of infection. A boy admitted with scarlet fever developed jaundice and fever two days after admission. During the next few weeks three other cases occurred in the ward. He had never seen that before. The cases were all slight; sometimes it was necessary to look for the jaundice, and sometimes it was better recognized by the colour of the urine than of the skin. He very much doubted whether there was any connexion between epidemic jaundice and mumps. It was recognized that acute pancreatitis, with or without jaundice, might be one of the complications of mumps; and just as orchitis might be the only manifestation of mumps, so might pancreatitis. A case similar to that of the nurse which had been mentioned by Dr. Guthrie had been recorded by Dr. Gordon Sharp as occurring in the Leeds epidemic of mumps of some three or four years ago.

Dr. JEWESBURY mentioned a case he had heard of lately of a breeder of bull-terriers who had taken a new house and put one of his dogs into the stable, which had been disused for some time. After the dog had been there a short time it developed slight jaundice, but recovered. Subsequently other dogs of the same breed were put there, and they also developed jaundice. It was then discovered that the drainage of the stable was very defective. After the drains had been put right there was no more jaundice among the dogs which were housed there. It seemed that there had been a definite infection causing the jaundice, possibly air-borne, in the way which Dr. Guthrie had suggested.

Section for the Study of Disease in Children.

December 13, 1912.

Mr. A. H. Tubby, President of the Section, in the Chair.

A Discussion on the Treatment of Heart Disease in Children.

THE MODERN METHODS OF TREATMENT OF HEART DISEASE.

By Edmund Cautley, M.D.

Reference to the standard works of the past generation on the diseases of children shows that the recognition of the frequency of heart affections in early life is of comparatively recent date. A few cases of endocarditis in childhood were reported in 1843 by Charles West as a positive addition to medical knowledge. Similar cases had been described shortly before this in the treatise by Rilliet and Barthez.

Our modern methods of treatment are a survival, a modification, and an improvement on those of the past. They depend more particularly on the differential diagnosis of myocarditis and its effects. Former text-books barely mention this important affection of the heart in childhood. Vogel, Professor of Clinical Medicine in the University of Dorpat, Russia (1873), states that myocarditis, or molecular degeneration of cardiac muscle, is found in cases of sudden death in diphtheria. He did not recognize it as a complication of acute rheumatism, although endocarditis and pericarditis were described by him as part of this disease. And so little attention did he give to the treatment of heart disease that there is no reference in his book to the treatment of cardiac dilatation.

Meadows and Tanner (1879) dismiss the treatment of hypertrophy and dilatatio cordis in five lines. The main stress is laid on complete rest. Pain and other unusual sensations are relieved by morphia and aconite; and any dyspnœa by digitalis and antispasmodics. Two lines are devoted to the statement that "much relief is often experienced by the application of belladonna plaster over the cardiac area." I fear we have not the same simple faith in the use of such plasters, in so far as their action is supposed to depend on absorption of belladonna by the skin. The treatment recommended for endocarditis is that of the primary cause—viz., rheumatism, scarlet fever, measles, or continued fever. Opium, alkaline drinks and hot baths are recommended for rheumatism; and linseed poultices, fomentations of poppy or laudanum, opiates, aconite, and hydrocyanic acid for endocarditis. Special value is attached to the use of blisters, if the child is not less than 5 years old. Rest and light, nutritious diet are advised, and stimulants are said to be needed occasionally in liberal quantities. In the treatment of pericarditis it is stated that mercurialization and the use of the lancet have fallen into disuse, the latter being uncertain and often highly dangerous. The measures adopted are those suitable for acute rheumatism, together with blisters, potassium iodide, and stimulants, if necessary. According to these writers carditis is "so extremely rare as scarcely to need any consideration." They refer to a case of pericardial effusion which was successfully treated by paracentesis and the injection of tincture of iodine and potassium iodide.

Charles West (1884), in the seventh edition of his book, remarks that forty years previously morbus cordis was entirely unnoticed in one standard work on children and dismissed in six lines in the other. His own observations were based on 140 cases: endocarditis, eighty-one; pericarditis, fifty; dilatation and hypertrophy without valvular disease, nine. Myocarditis is not mentioned in so many words, but extension of inflammation from the endocardium and pericardium to the cardiac muscle is recognized; and he describes the condition in association with endocarditis of rheumatic origin, apparently regarding it as simple dilatation. Further, he describes a case of dilated heart, probably due to myocarditis, in the course of severe chorea; and fatal cases of dilatation without co-existent valvular disease, the insufficiency of the valves being due to the dilatation. West recognized that the severity of the illness depended on the degree of dilatation. His treatment was directed to reducing the work of the heart by means of rest, the application of four to eight leeches over the heart at the onset, and tinct. aconiti, ½ to 1 minim, every fifteen minutes until the rate of pulse and respiration was reduced. He writes favourably of mercury and opium, prescribes alkalies, and mentions, but had not made a fair trial of, salicylates. For pericarditis he advises cupping, leeches, calomel, Dover's powder, and mercurial inunction; and is in favour of the use of blisters, or blistering fluid, over the cardiac region after the fever has subsided. Paracentesis is mentioned, there being eight cases under 12 years of age on record. In the treatment of chronic heart disease he recommends counter-irritants, digitalis and iron, with belladonna plaster for palpitations, and diuretics and digitalis for œdema.

In the same year, 1884, Dr. Eustace Smith, the senior living authority in this country on the diseases of children, published the first edition of his classical text-book. He recommends sodium salicylate and a mercurial purge for rheumatic fever, blistering for endocarditis, and iron and quinine if the fever has subsided. He prescribes blistering at the onset of pericarditis, and large doses of potassium iodide—e.g., 30 gr. daily at 5 years of age—on the assumption that this drug removes serous effusions. The iodide was not given until the joint pains had subsided. Tartrate of iron was sometimes added. He advises that for chronic valvular disease, giving rise to no trouble, no treatment is needed beyond general care. Active measures are required for palpitations, breathlessness and anæmia. He preferred digitalis in the form of infusion and recommends drachm doses of the infusions of digitalis, senna and calumba, three times daily. Attention is also paid to diet. For dilatation and œdema he prescribes diuretics, and regards the tincture of cantharides, 30 minims daily at 9 years of age, as especially valuable. He mentions caffeine as a diuretic, and recommends jalap as a purgative, and dry cupping as an aid to diuresis. Stimulants are sometimes needed, and Southey's tubes are used for œdema.

Such were the orthodox views on the treatment of cardiac affections in children at the time I began the study of medicine. In comparison with those of the present day the chief improvement, apart from the introduction of a few new drugs, depends on the recognition of the frequency of myocarditis and its effects as the fundamental factor in the majority of cases in children. In this respect our knowledge has advanced, and in consequence we have learnt to appreciate the necessity for prolonged rest. Important points for discussion are the limits which should be placed on the duration of rest and the subsequent use of exercises; the value of blistering, leeches and venesection in acute inflammations and severe dilatation; the use of digitalis and its products; the value of special purgatives and diuretics; and the indications for operative treatment.

To a certain extent the methods of treatment are modified by age, for the child is a very restless animal, its metabolism is active, and the heart is a growing organ. Fortunately the degenerative changes consequent on age, alcohol, syphilis, and renal diseases are very rare. During periods of rapid development, especially at puberty, the cardiac growth may not be proportionate to that of the body and the heart may become functionally incompetent, as indicated by undue frequency of the pulse and shortness of breath. Functional disorders of the heart are comparatively uncommon in children. In many a so-called functional case there is a primary myocardial affection, the result of latent rheumatism or the sequel of some infective disorder. It is apt to follow influenza, is a frequent sequel of diphtheria, and may even ensue on varicella or the common influenza cold. Obviously in such cases successful treatment depends on accurate diagnosis. It is likely to prove unsatisfactory if slight myocardial mischief is regarded as functional nervous disturbance. And it must be remembered that a cardiac murmur does not always mean valvular defect, nor does the absence of a murmur necessarily exclude serious disease.

In applying treatment we must take into consideration the nature of the mischief and the valves affected, if any; the nutritional and functional powers of the cardiac muscle; the idiosyncrasy of the patient and the irresponsibility of childhood; the environment of the child, and the difficulty in securing parental co-operation as soon as the obviously troublesome symptoms have subsided.

Rest is the most important factor in treatment. It is not needed if there is good compensation; then, it is only necessary to guard against overstrain. The duration of rest depends on the degree of dilatation consequent on myocardial incompetence, and on the existence of endocarditis, myocarditis or pericarditis alone or in combination. It may be impossible to ascertain whether a mitral systolic murmur, associated with cardiac dilatation, is due to endocarditis or to incompetence of the valvular orifice because of dilatation and myocarditis. Rest must be continued for one to three months after all acute symptoms have subsided and compensation has been established. It must be most prolonged in myocarditis. Special care is needed during rapid growth and at puberty, periods at which cardiac strain must be carefully avoided, if there is the least suspicion of previous mischief or if there is any functional incompetence. It is a mode of treatment easily carried out in young and placid children, and perhaps extremely difficult in excitable, vigorous children over 10 years of age. A trained nurse is almost invariably essential. In the early stages rest must be absolute, and every cause of excitement, such as visitors, new toys and games, must be prohibited. As compensation is acquired rest is gradually replaced by massage, passive and active movements, and exercises. A warning is necessary against the abuse of exercises for children with even well-compensated cardiac disease. The modern craze of flying to Swedish and other exercises has been, in my experience, responsible for the cardiac breakdown of several children. The growing heart, weakened by disease, is easily overstrained. Exercise must not cause dyspnœa or anginal pain. For three to twelve months it is limited to walking on level ground, and is then increased successively by minor gymnastics, hill-climbing, bicycling on the level, and appropriate games.

Baths, saline and carbonated, such as given at Nauheim and also in this country, and electro-therapeutic methods of treatment, are perhaps unnecessary in childhood. Baths stimulate the cutaneous nerves, dilate the capillaries of the skin and slow the action of the heart, exciting more vigorous action, and diminish the cardiac dullness. They are suitable for moderate cardiac dilatation and can be given on alternate days, with Schott's exercises on the other days.

Diet.—In acute cases, especially if rheumatic in origin, a diet of milk and carbohydrates is advisable. Some physicians allow chicken broth, beef tea, and other meat extracts. Personally, I regard such extracts as unsuitable and prefer to order fruit or vegetable soups, made without meat stock, as a change, and to counteract constipation. In all cardiac affections the food should be nutritious, easily digestible, and given in small meals so as to avoid overloading and flatulent distension of the stomach. The state of the appetite is a valuable indication of progress. One of the first signs of failing compensation in chronic heart disease is anorexia after exercise. It is followed by dyspepsia, impaired nutrition, restlessness at night or insomnia, anæmia, cough, shortness of breath, and swelling of the legs. One of the most reliable signs of improvement is gain in weight, provided there is no œdema. In cases of œdema a salt-free diet is sometimes advantageous, combined with a moderate limitation of fluids. There is no necessity to prescribe a strict diet in chronic cardiac failure, provided that the food is nutritious, given in small meals, and palatable. There is too great a tendency to order milk in quantities larger than the patient can digest efficiently, with the result that the appetite becomes impaired, the tongue furred and the breath offensive, the stomach dilated and the bowels constipated.

Blistering is in my opinion of very doubtful value in endocarditis and myocarditis. That a blister on the skin can affect an inflammation of the cardiac valves requires a more optimistic imagination than I dare own to. Although such treatment is used and highly recommended, it is associated with other remedies which are more probably the cause of the favourable issue. In pericarditis blistering seems undoubtedly beneficial. The best method of application is the use of flying blisters around the inflamed area. Other counter-irritants may be used.

The local application of *ice-bags* in the treatment of cardiac inflammations has been warmly advocated, especially for pericarditis. Sometimes it appears to quiet the excited action of the heart. Possibly this result is merely due to the complete rest so necessary for the accurate adjustment of the treatment. As a rule children do not like cold applications. Poultices and fomentations are more soothing and can do no harm. The addition of opiates or belladonna does not make them more efficacious.

Venesection and *leeches* must be used with caution, for children stand the loss of blood rather badly. If there is much pain, an acute pericarditis, or an acute dilatation of the right ventricle due to myocarditis or cardiac failure, the application of a few leeches over the liver affords great relief. Venesection to the extent of a few ounces may be essential. Bleeding relieves the distension of the right heart for some hours and allows time for other remedies to exert their effect. It must not be adopted lightly, and it ought not to be allowed to fall entirely into disuse. It should be looked upon as a measure for immediate relief of the straining heart and not as a means of reducing inflammation.

Purgatives are essential. It is important to keep the bowels open, and freely so if there is backward pressure and a tendency to œdema. If the liver is engorged there is no pill as valuable as that of mercury, squill and digitalis, given at bedtime and followed by a saline cathartic in the morning. Such a pill may be given twice or three times a day. In cardiac inflammation the hydragogue cathartics are the most valuable purgatives, for they relieve the congestion of the tissues throughout the body and reduce the work required of the heart. If purgatives are required in cases of compensated chronic heart disease, the various preparations of paraffin, senna, aloes, or cascara may be used. A weekly dose of calomel or blue pill is sometimes advantageous.

Diuretics are also of immense value in acute inflammations and in the relief of œdema. Their action can be assisted by dry cupping over the kidneys. Dry cupping over the bases of the lungs is useful in passive congestion of these organs. I have no experience of the tincture of cantharides recommended by Dr. Eustace Smith as a diuretic, nor am I prepared to state which is the best diuretic. Diuretin, caffeine and theocin sodium acetate are all of value, the last mentioned often increasing the efficacy of digitalis.

Digitalis is a diuretic and a cardiac tonic. There is a tendency to limit its use to non-inflammatory cardiac affections, though it may be prescribed tentatively in the course of inflammatory ones, with the exception of endocarditis. It is not always required because a murmur is present. Formerly the infusion was preferred. Herbalists recommend that the leaves should be gathered at night, and there is recent evidence that they contain more of the active principle at this time. Now, most physicians prescribe the tincture as being more stable and regular in composition. It is the simplest and most available preparation. The drug should be given every six hours until a definite reaction is obtained, and repeated if relapse occurs. Try to find the dose which maintains improvement without causing unpleasant symptoms. The patient's own sensations are a good guide. The drug must be omitted, if it decreases or does not increase the secretion of urine; if it causes considerable slowing of the pulse; if it gives rise to anorexia, nausea, and confusion of thought; and if the patient cannot be kept under supervision. Frequently, continuous administration is advisable, less often in children than in adults, and chiefly for cardiac failure due to mitral regurgitation. For such cases, after compensation is fairly well established, a dose of the powdered leaves, $\frac{1}{2}$ to 2 gr. once a week or more often, will maintain the compensation. Or the drug may be given for three to four days at a time every month. I have obtained excellent results both in adults and children by such means, using Nativelle's crystallized digitaline in doses of $\frac{1}{10}$ to $\frac{1}{4}$ mgrm. daily for a week at a time. Digitalone (Parke, Davis and Co.) is physiologically standardized. Ten minims are equivalent to eight of the tincture and to 1 gr. of the leaves. Digalen, a sterile amorphous digitoxin, is said not to be cumulative. This has been disproved recently. One cubic centimetre is equivalent to 0·3 mgrm. digitoxin, folia 2·05 gr., tincture 18 minims, and infusion 5·4 dr. These drugs can be given subcutaneously and by rectum, if necessary. Crystalline digitaline is official in the French Codex. It corresponds to the German digitoxin and is much more powerful than amorphous digitaline. Most of these preparations are recommended because of their constant strength, and it is possible that the claim may be true, and also on the grounds that they are non-cumulative and less apt to upset the stomach. In some cases strophanthus is more suitable—e.g., in severe dilatation and mitral stenosis.

Squill, sparteine sulphate, strychnine and nux vomica are also valuable. Alcohol is necessary in acute dilatation, myocarditis, and cardiac failure. Cardiac stimulants are not needed if recumbency alone is sufficient to reduce the action of the heart to the normal rate and if compensation is established. They are most efficacious when the right side of the heart has been relieved by cupping, leeches, or venesection, and the liver relieved by mercury and a saline aperient. I know nothing about the tincture of cereus mexicana which has been recommended, in doses of 5 to 30 minims for adults, to increase the strength and regularity of the heart without raising the blood-pressure. Vaso-dilators are unnecessary, for blood-pressure is almost invariably low in children. Perhaps iodides are of value in chronic pericarditis.

Salicylates, and their congeners, are generally prescribed in all cardiac inflammations of rheumatic origin. The remarkable effect on the temperature, joint pains and swelling of rheumatic fever is strong evidence of the great value of this drug. Yet, although I have used it systematically for a quarter of a century, I still find it difficult to advance reliable evidence that the drug affects the course of cardiac inflammation. In my experience it is by no means uncommon for the myocarditis to run a prolonged course with mild fever, the temperature running up to about 100° F. at night, in spite of the liberal administration of salicylates or aspirin.

SUMMARY.

In all inflammatory cases in which there is the least suspicion of rheumatism salicylates and alkalies must be prescribed. The patient is kept in bed on a light milk and carbohydrate diet, fruit and vegetable soups being allowed as a change. Complete rest and a trained nurse are unnecessary. Blistering or counter-irritants may be used, but are chiefly efficacious in pericarditis. Vaccines and serums are of problematic value. There is hope that, when the organism of rheumatic fever is isolated, an autogenous vaccine may prove beneficial to the patient. I do not wish to ignore the extremely valuable work done in this connexion, but, though realizing that many observers are agreed that the genuine organism has been already discovered, there seems to be at present a disinclination to accept it as absolutely proved. In infective endocarditis—fortunately infrequent in children—my experience with vaccines and serums has proved unsatisfactory. It is difficult to grow the incriminated organism from the blood, difficult to prove that the organism obtained in cultures is the one causing the disease, and difficult to obtain good results from an autogenous vaccine or from a polyvalent serum.

Recourse is sometimes had to paracentesis for the relief of pericardial effusion. It is rarely essential in rheumatic pericarditis, for even the largest effusions may be absorbed. It is a point for discussion whether the operation would promote more rapid recovery. For purulent effusions the pericardium must be opened and drained. Another and more modern operation, sometimes called cardiolysis, is resection of the rib cartilages over the cardiac area, for the relief of general adhesion of the pericardium. The treatment of cardiac failure and backward pressure is the same at all ages. For extreme œdema of the lower limbs multiple punctures and sterilized dressings are more satisfactory than the insertion of Southey's tubes. Iron and glycerophosphates are given when compensation is established. Compensation is maintained by regulation of the mode of life, diet, sleep, hygiene, and exercise. These patients must not be allowed to become chronic invalids. A more just appreciation of their capabilities is now general, in comparison with the views held by the past generation. If there is merely some mitral regurgitation, the only precautions necessary are against rheumatism and overstrain. Such children ought not to take part in football, races, steeplechases, and like competitions. They may take part in other varieties of exercise during which they can stop as soon as they feel any cardiac discomfort. If compensation is less complete, more care is needed. The diet must be regulated, the bowels kept open, and the general health attended to. A weekly dose of blue pill or calomel, and digitalis once a week or more often over a long period of time, will often maintain and even increase the degree of compensation.

In every case it is essential to recognize the cause and differentiate the type of cardiac disease, and clearly to understand its probable course and prognosis. Our methods of treatment do not differ markedly from those of the past, but I think we may claim that they approximate more accurately to the needs of the particular patient, that we appreciate more clearly the necessity for prolonged rest and great care during convalescence, and that we do not hold such gloomy views as to the prognosis in heart disease.

SOME MANIFESTATIONS OF A HEALTHY HEART IN THE YOUNG FREQUENTLY TAKEN AS INDICATIONS FOR TREATMENT.

By JAMES MACKENZIE, M.D.

WHEN I was asked to take part in the discussion on the treatment of heart disease in the young, I cast my mind back on my experiences to see if I could usefully join in the discussion. On reflection, I was struck with the fact that I had seen a large number of young people who had been subjected to many forms of treatment because of the presence of certain phenomena which my experience had taught me were signs neither of disease nor of impairment. When I reflected further on why it was that members of our profession frequently misapprehended the significance of cardiac phenomena, I could only attribute it to the state of confusion in which cardiac symptomatology has been involved, and the manner in which it is taught in the schools. It seemed to me, therefore, that it might be profitable to place before you the results of some observations which I have made in the endeavour to appreciate the significance of certain cardiac phenomena, inasmuch as the first step towards treatment is a knowledge of what you are going to treat.

THE ESSENTIAL PURPOSE OF A MEDICAL EXAMINATION.

The main object of a physician's examination is to determine the value of certain symptoms of which the patient may complain, or the value of certain signs which the physician may detect. After the examination the essential questions which the physician has to answer before he considers the question of treatment are: What bearing has such a sign or symptom on the efficiency of the circulation? Does their presence indicate heart failure, or do they foreshadow its occurrence at some future period?

In order to show how difficult it may be to answer these questions I would refer to a deficiency in our knowledge which confuses and misleads those who seek an answer.

HOW THIS PURPOSE FAILS.

(*a*) *In the Case of Irregular Action of the Heart.*—Let me illustrate this in a very simple fashion. Let us suppose a doctor detects in his patient or himself an irregular action of the heart, and he wishes to understand its significance; in other words, he wishes to answer the questions which I have just enumerated. Does the irregularity indicate heart failure at the present time, or does it foreshadow its possible onset at some future period? He turns to his text-book for enlightenment, but fails to find the answer to his questions. He may ransack a medical library and even then he will fail to get an answer, or the slightest suggestion as to how to approach the subject. Some large books ignore the subject, others deal with it in so perfunctory a fashion that no clear perception is forthcoming, while in the remainder the subject is dealt with in such a way that it merely bewilders the reader. Thus he will find such statements as that irregular action of the heart is of little or no importance; then a few pages farther on the same writer will state that he rejects candidates for insurance if they show an intermission of the pulse at rare intervals. In the description of grave conditions, a passing allusion to irregular action of the heart may convey the idea that it is one of the signs of a serious condition, but there is not the slightest hint given to help the reader to differentiate the innocent from the serious.

(*b*) *In the Case of Murmurs.*—If it is desired to estimate the significance of a murmur, the same absence of reliable information is found here. This subject, which has received such long and exhaustive study, is invariably dealt with in such a manner that it would be impossible for the reader to glean information which might enable him to answer the questions which I have put, except perhaps in the case of some murmurs associated with advanced disease. Consider, for instance, the origin of the present-day conception of the significance of heart murmurs. Within a few years of the discovery of the stethoscope the murmur was looked on as a sign of disease. This was at a time when the cause of the murmurs was not understood and before any one had watched individual cases long enough to find out what the influence was on the life of the person affected. Doubtless much has been done since then to throw light upon this subject; nevertheless, the early teaching has so affected the profession that the great majority of medical men to-day are labouring under the erroneous doctrines taught in the early days of auscultation, and so look upon murmurs as evidences of disease or impairment of the heart. Upon this imperfect conception of the meaning of murmurs another idea has been based—namely, the back-pressure theory of heart failure. While this theory contains some element of truth, it is so applied as to give a totally false conception of heart failure in the great majority of cases. Thus we find cases with a mitral murmur, systolic in time, diagnosed as mitral disease, and, if there are no signs of heart failure, the individual is said to have "mitral disease, with good compensation." The very term "compensation" conveys the idea of impairment, and that a special effort of the heart has enabled it to combat this weakness. It is in consequence of this misleading conception of heart failure that we find the great majority of heart affections labelled by the murmurs which the heart happens to make, although the cause of the murmurs has nothing to do with the failure of the heart.

(c) *In the matter of Prognosis.*—This great deficiency in our knowledge of cardiac matters comes out with irresistible force when an attempt is made to deal with prognosis. Practically every writer who gives the matter his serious attention is forced to admit that we do not possess the data on which to build a reasonable and sound prognosis. One of our greatest and most experienced clinicians, the late Sir William Gairdner, has accurately described the position of the medical examiner for life insurance in his method of estimating a life value. He says: "There is an incalculable element in most of the cases . . . which we deal with in the most curiously unsatisfactory way, either by rejecting the life altogether, or by putting on a big loading, which, it must be admitted, we assess upon no very sound or clear principles, or, on the other hand, by winking at the defect and passing it over." He points out that there are no reliable data on which to estimate the prognostic significance of murmurs and irregularities. This description of the absence of reliable data on which to base a prognosis holds good to-day not only in life insurance, but wherever a prognosis is based on a physical examination.

I dwell with insistence on this great defect in our knowledge of the significance and meaning of cardiac phenomena, in order that you may better appreciate certain investigations which have been pursued with the purpose of clearing up these matters. I am far from saying that this deficiency has been made good, for that will require the continued labour of many observers; but there are certain lines which must be followed, if this object is ever to be attained, and these I will briefly indicate, and cite some of the results which have been achieved.

METHOD EMPLOYED TO FIND OUT THE MEANING AND SIGNIFICANCE OF SIGNS.

Let us first consider irregular action of the heart. Shortly after entering general practice over thirty years ago I was struck with the frequent occurrence of irregular action of the heart among my patients, but at that time the subject was dealt with in text-books in such a way that no trustworthy estimate could be made of its nature or significance. I began in a very simple way to seek for some basis of classification, and was able to separate some of the irregularities into groups according to the different ways in which the chambers of the heart participated in their production. Later this subject was taken up by many other observers, who studied it both clinically and experimentally, and as a result of these labours irregular action of the heart, which until a few years ago was one of the most obscure subjects in clinical medicine, is now one of the best understood, and our knowledge is based upon sound physiological data. This separation of irregularities into groups according to the mechanism of their production is sometimes looked upon as being merely an academic exercise of little practical importance. As a matter of fact, it was a preliminary but absolutely necessary step to find out the bearing of these irregularities on the patient's present and future state.

HOW THE KNOWLEDGE OF THE SIGNIFICANCE OF IRREGULARITIES WAS ACQUIRED.

To find out the bearing of the different forms of irregularities on the patient's future necessitated a line of observation of much greater difficulty and perplexity. It entailed the watching of many individuals during long periods of years, observing young people growing up into manhood, seeing how the heart behaved during periods of strain, as hard bodily work, pregnancy and labour, illnesses such as typhoid, pneumonia, influenza, and finding out how they reacted to remedies. When heart failure was present it was necessary to find what bearing the cause of irregular action had on the heart failure. In course of time I was able to suspect the cases in which certain irregularities would arise; these I kept in view, examining them from time to time, in order to detect the moment of onset of the irregularity, and also the changes which occasioned it. By this means, for instance, I was able to detect the changes which are coincident with the onset of the irregular heart, which we find in cases of auricular fibrillation. To give you some idea of the labour it has entailed to gain this limited amount of knowledge, I may say that I have taken tens of thousands of tracings, and the great bulk of these have been subjected to long and careful analysis. Such tracings are only incidents in the investigation, being invariably accompanied by a search, often prolonged, into other signs exhibited by the patient.

THE RECOGNITION OF IRREGULAR ACTION AND ITS SIGNIFICANCE.

To illustrate the importance of this work I shall direct your attention to the valuable information regarding some of the irregularities which has been obtained. The great majority of heart irregularities can be divided into four groups, all differing in the mechanism of their production and in their clinical significance.

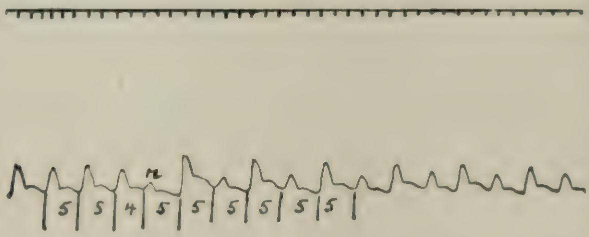


FIG. 1.

Tracing showing the alternate appearance of a large and small pulse-beat the *pulsus alternans*. The figures represent tenths of a second, and the rhythm is regular except for the appearance of a premature beat (*r*).

(*a*) *The Pulsus Alternans.*—The first of these irregularities to which I would draw your attention is a form which is regular in its rhythm, a large beat alternating with a small one (fig. 1). This irregularity, known as the *pulsus alternans*, only occurs when there is great exhaustion of the heart muscle. It may be that the exhaustion is only temporary, as in the tachycardias which arise from an abnormal action of the heart, or it may arise from exhaustion due to such profound alterations in the heart muscle that it indicates in many cases an exhaustion so extreme that death will shortly follow.

(*b*) *The Irregularity of Auricular Fibrillation.*—The second of these irregularities is, as you will see, of a very disorderly kind (fig. 2). It indicates the presence of fibrillation of the auricles, and from the point of view of the future life of the patient it has a very varied significance. I have collected over 1,000 cases of this form of irregularity alone, and it occurs in 70 per cent. to 80 per cent. of all the cases of heart failure with dropsy. Some of my patients have died within a few days of its onset; some have lingered for a few months; others have lived a crippled existence for years, while still others may have been little the worse for its appearance. I have endeavoured, by the study of other phenomena present, to find out the reason for these varied results, and I think we can now formulate with reasonable certainty the facts which guide us in a prognosis and the data on which to base a rational treatment.

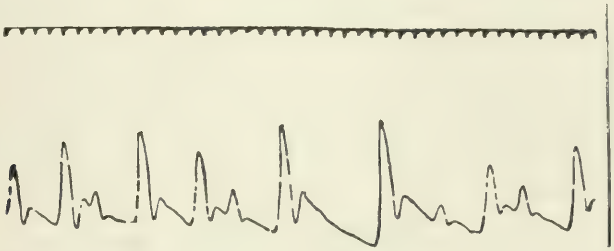


FIG. 2.

Tracing showing the disorderly character of the irregularity in auricular fibrillation.

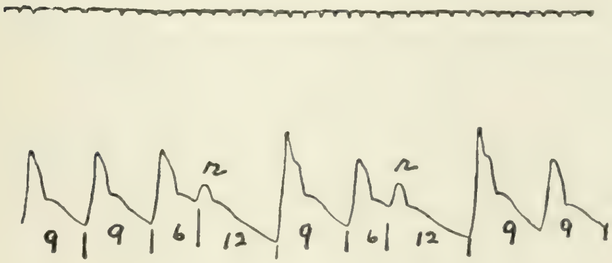


FIG. 3.

Tracing showing the occurrence of two premature beats or extra-systoles (*r*, *r*) in an otherwise regular heart. The figures represent tenths of a second.

(*c*) *The Extra-systole.*—The third form of irregularity is extremely common in adult life and rather infrequent in the young (fig. 3). It is due to a premature contraction followed by a long pause. The vast majority of people who show it are in good health and never suffer from heart failure. It may be present in heart affections and with serious heart failure, but an opinion of gravity should never be based on it alone, but on the presence of other signs of heart failure. When it is the only abnormal sign present it may be ignored, as far as its effect on the functional efficiency of the heart is concerned.

(d) The Youthful Type of Irregularity.—I wish to draw attention particularly to the fourth group—a group which is characterized by a variation in the length of the diastolic period (fig. 4). It is readily recognizable on auscultation by the varying length of the long pauses between the second and first sounds. So long may these be at times that the heart seems to miss a beat. It is frequently respiratory in rhythm, the slow beats occurring during expiration. At other times it may not be so accurately related to the phases of respiration, but it can sometimes be made so by getting the individual to breathe slowly and deeply. On classifying cases showing this irregularity I found the great majority of them were young people. Directing my attention to the occasion of its occurrence, I found that it would appear and disappear in the same individual for no perceptible reason. I found it so common that I have come to the conclusion that every healthy individual probably shows it at one time or another. I found it occurring most frequently and most markedly when the patient lay in bed after a febrile attack, but it is often found in boys and girls in perfect health. I have watched those showing it grow up and pass into adult life, and

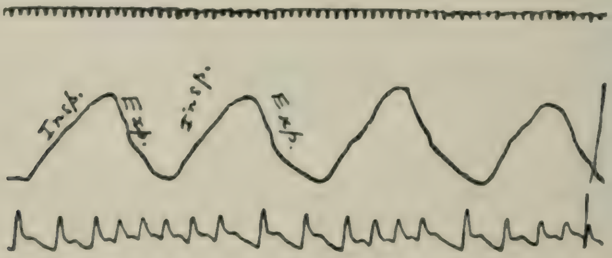


FIG. 4.

Tracing showing the irregularity characteristic of the youthful type. The phases of irregularity correspond in the main with the phases of the respiration (upper tracing), the slow period appearing during expiration.

I have never found a single case where it has been associated with any form of heart failure, so that I now look upon it as being a physiological phenomenon and occurring in perfectly healthy hearts. How very important it is to recognize this view will be apparent later on.

The Significance of Murmurs.

In order to find out the significance of murmurs I followed the same lines as those I had pursued in determining the significance of irregularities. Here I shall not enter into the results of this inquiry into murmurs due to damaged valves, but shall restrict myself to those murmurs which are recognized as functional, and to those where it is doubtful whether they are functional or organic. These are always systolic in time, and their position of maximal intensity may be at the apex, base, or mid-sternum. The result of this inquiry showed that these functional murmurs in themselves were consistent with perfect health, and that their cause never led to the slightest sign of heart failure. Where these murmurs were associated with heart failure there were invariably evidences that the heart failure was the outcome of the myocardial condition and not of the supposed incompetent valves. In any given case with heart failure there was usually present some condition which impaired the functional efficiency of the heart muscle, and induced an exhaustion of it such as occurs in an exhausting illness, or with structural changes, as fibrous or fatty degeneration of the muscle wall. Now these conditions happen so rarely in the young, and when they do happen the evidences of actual disease are so striking, that I am justified in stating that functional murmurs in the young, in the absence of evidence of muscle affection, are signs neither of disease nor of impairment.

Irregularities and Murmurs consistent with Perfect Health.

I have put this view to the test by watching a large number of individuals, and by following the lives of those showing these signs for such long periods that I have no hesitation in saying that murmurs and irregularities may be manifestations of a perfectly healthy heart in the young. The recognition of this fact is of great importance, for if the knowledge that healthy hearts do frequently present murmurs and irregularities was fully grasped, then the doctor would study his patient from a different perspective, and would cease to be obsessed by these and other seeming abnormalities, but would direct his search towards finding out how far the cause of these and other phenomena modified the functional efficiency of the heart.

IMPORTANCE OF MUSCLE IMPAIRMENT.

When the fundamental principles underlying the production of heart failure are thoroughly understood, it will be realized that the heart failure, whatever form it may take, is due to an impairment of the functional efficiency of the heart muscle. So far, then, as heart failure is concerned, the significance of any sign must depend on how far its cause embarrasses the heart muscle in its work. It follows, therefore, that a murmur or an irregularity is only of significance when it is associated with some manifest impairment of the heart muscle, and that an inquiry must be made which will bring out the true meaning of any sign by searching for evidences of muscle impairment.

SIGNS OF MUSCLE IMPAIRMENT.

What are the evidences by which we can recognize affections of the heart muscle? Except for certain irregularities, which in rare cases give very important information, the evidence is somewhat limited.

(*a*) *Increase in the Size of the Heart.*—The first of these is a change in the size of the heart, due either to dilatation or to hypertrophy of its walls. Before we can assume that any increase in size is abnormal, we must first remember that there is no standard size, shape, or position, and that the variations which healthy hearts, especially in the young, can show, are very considerable. Doubtless in certain diseases of the heart the increase is size in so marked and so characteristic that little difficulty is experienced in recognizing it as a morbid condition. With functional dilatation, however, the difficulty of recognizing its significance is so great that evidence based upon increase in size, unless this is extreme, should never by itself be considered as sufficient. If it is associated with persistent increase in the rate of the heart and marked loss of power, then it forms a valuable indication. But an apex beat in the fourth or fifth interspace, 1 in. outside the nipple line, is not in itself an evidence of muscle affection. It is well also to bear in mind that impairment of the heart muscle, dangerous to life, may be present without increase in the size of the heart.

(*b*) *Murmurs not necessarily a Sign of Dilatation.*—A systolic functional murmur must not be considered as due to the dilatation, because these murmurs may not be the outcome of the dilatation. If a large number of healthy hearts be carefully examined it will be found that some show murmurs with the heart of a size usually assumed to be normal. These murmurs may come and go when there is no change in the size of the heart; they may be perceptible only when the individual is lying down or when he is standing up; sometimes they are only present when the heart is excited, at other times they are found when it is beating slowly and quietly. It frequently happens that though there may be marked dilatation, with and without considerable heart failure, no functional murmurs may be present. From such considerations as these we are forced to the conclusion that the mechanism which produces the murmur is something apart from that which provokes dilatation of the heart. The view so widely held that these systolic functional murmurs indicate regurgitation, and that in consequence there is a danger of this regurgitation embarrassing the heart and leading to heart failure, is merely an outcome of the back-pressure theory and has no existence in fact.

(*c*) *The Condition of the Functional Efficiency of the Heart.*—The best and most accurate, and, in fact, the only reliable method of estimating the condition of the heart muscle, is to acquire a knowledge of its efficiency, and, if it be inefficient, the extent of its limitation. This method consists in finding out how the heart responds to effort and recognizing the symptoms which indicate its exhaustion. In the limited time at my command I cannot enter into a description of the physiological principles involved in this method of estimating the state of the heart muscle, nor of the manner in which the symptoms are evoked. I shall only state that the chief symptoms are subjective, the individual himself being conscious of his limitations. This method may seem to you to be so obvious as not to need mentioning, but it is probably because it is so commonplace that its importance has never been appreciated. It may also seem so simple that any one can apply it; on the contrary, the symptoms are at times so subtle and elusive that it requires very careful investigation before they can be appreciated. So many subjective sensations are connected with the circulation that it requires the most careful discrimination to find out those which are ndicative of an exhausted muscle.

There is no doubt that many physicians have learnt from their own experience what I am endeavouring to place before you, but this knowledge is not conveyed with such precision and detail as to make their meaning clear. Thus, until recent methods were employed the different forms of irregularity were never even classified on a basis of the slightest value for practical purposes. The method of estimating the heart's strength by testing the functional efficiency, which is the most essential part in every examination, has been to a great extent neglected. In proof of this, consider the insurance certificate which medical men fill up. All the questions are restricted to the physical examination and not one directed to finding out the functional efficiency of the organ, by which only the facts which are essential to a prognosis of the heart's condition can be ascertained. Notwithstanding, the books which are supposed to guide the medical examiner give no hint that such a method exists. The result is that I have seen individuals accepted at normal rates because a physical examination revealed no abnormality, whereas if an inquiry had been directed to the functional efficiency of the heart, the symptoms of exhaustion which heralded the unexpected death of the individual would have been revealed. On the other hand, I frequently see individuals rejected for life insurance because of the presence of some harmless manifestations, the significance of which the examiner did not understand, and the value of which he could not appreciate, simply because he failed to apply and to appreciate this method of testing the heart's efficiency.

THE METHOD OF TEACHING CARDIAC SYMPTOMATOLOGY AT FAULT.

Probably the whole error arises from the manner in which the student is taught when he passes into the medical wards. Here he is handed over for instruction in the physical signs to a clinical tutor, a teacher whose knowledge is so little in advance of his own that he does not understand the significance of the facts which he teaches. Under the care of this clinical tutor, at a time when he is most suspectible to impressions, he has hammered into him a certain kind of knowledge which remains with him and influences him during the remainder of his life. Thus, he is taught how to recognize the sounds of the heart, the size of the heart and its rate and rhythm, and what is supposed to be the normal in a healthy individual. Then he is shown, as a contrast, diseased conditions, with murmurs and muffled sounds, hearts of varying sizes, rates, and rhythm. Consciously, or unconsciously, he gets the idea that a heart to be healthy must be of a certain size, the sounds clear and free from murmurs, and the rate regular. This idea may be sought to be modified by more experienced teachers later on, but I am certain that every medical man who passes out of his hospital training does so with a false conception of the nature of phenomena which a healthy heart can exhibit. How this works out in after-life I will show you immediately.

How this Imperfect Teaching affects the Practitioner in his Work.

You may think I am dealing at too great length with the part which the medical man plays, but if we wish to appreciate the full consequence of our errors and misconceptions we must know how they have arisen. In order to bring before you more clearly the significance of what I am trying to prove to you I will refer to my own experiences. Some ten or fifteen years ago there arose a strange notion of the dangers of athleticism in schoolboys and college youths. I think this originated in London, but at all events it speedily spread over the face of the earth. Exponents of the idea did not limit their views to the medical Press, but, if I remember aright, warnings were written in the lay Press by some of our learned colleagues. So widespread did this doctrine become that great numbers of boys returned to school with certificates of unfitness to play the usual games, to such an extent that in some schools one-half of the boys were supposed to suffer from impaired hearts. At this time I was pursuing my work in an isolated town in the North, and I wondered what were the symptoms by which these enfeebled hearts were recognized, and what were the evidences of an athlete's heart. My boys played strenuous games and entered into occupations entailing severe bodily labour, but I never saw any of them unfit unless they had manifest disease of the heart. It was not until I came to London five years ago that the real solution dawned upon me. The first consultation which I had in London was concerning a young girl who had fainted. She lived in the country, and a London physician was called in, who recognized some form of heart abnormality, and had her removed to London to undergo a course of what is called Nauheim treatment. She had had two courses and had been under treatment for three months when I was asked if I could suggest any remedy for this intractable complaint. Judge of my surprise when I was told that the only evidence of disease was a persistent irregular action of the heart, which I found to be the youthful type of irregularity—a phenomenon which, so far from being a sign of disease, is, as

I have told you already, presumptive evidence of a perfectly healthy heart. From this time onwards young folks were brought to me in increasing numbers, until I have seen a very large number of youths who were supposed to have athletic hearts, or were supposed to be unfit to play games or to row, because of some impairment of the heart. Except in a few instances of manifest heart disease, the evidence on which the heart's impairment was based was those manifestations of murmurs or irregularity which my experience had shown to be perfectly consistent with a healthy heart. In addition, I have seen a great many youths who have been rejected for life insurance and for the Services, and other occupations which entail a preliminary medical examination, because of the presence of these manifestations of a healthy heart.

In my communications with medical men I have found the notion that a murmur or an irregularity is an evidence of disease so deeply rooted that it is simply impossible to get many of them to look at the evidences which point to the contrary. I have had a great deal of disagreeable correspondence with doctors after I have written to them about youths whose hearts I have pronounced healthy in spite of murmurs or irregular action. I have had replies to the effect that they have had great experience in this subject, and that they know I am wrong. I have asked such correspondents to recite to me the history of one single individual who has ever shown the slightest sign of heart failure, in whom these symptoms were found, and so far I have failed to get the history of a single case. Nay, more, although you will find in the writings of authorities grave warnings of the danger impending on youths who show these phenomena, yet I have failed to find the record of a single case in which these forebodings were realized. Considering how numerous are individuals showing these signs, and considering the strenuous games which are played and the strenuous lives that are lived, had these signs the gravity they are assumed to have, there would be on record many cases of heart failure, yet, as I say, literature is silent on the subject.

Sir JAMES GOODHART said he came to the meeting very much attracted by the heading of Dr. James Mackenzie's paper, intending to say "Ditto," but he had not realized how much confirmation it would involve. He did not think it would be possible to express his own feelings on the subject of the debate better than Dr. Mackenzie had just expressed them. As he had had forty years experience of such cases, he had a right to venture an opinion, though it need not be taken as more than his own. On this one point he was not inclined to agree with Dr. Cautley, though within the limits of heart disease nothing could be better than his paper. He understood Dr. Cautley to say that he did not think functional disease in childhood was common, but he (Sir James) was there to say—and he hoped Dr. Mackenzie's paper would be read far and wide—that if he had estimated heart conditions aright, he saw many more cases which were said to be heart disease in children in whom there was nothing the matter, than he saw of heart cases in which there was actual disease. And the reason was, as Dr. Cautley said, that pronounced disease was recognized. But there was a large number of borderland cases, dealing with supposed displacement of the apex and with murmurs, which were thrown into the limbo of diseased hearts, but in which there was no disease whatever. He could not follow Dr. Mackenzie and give cardiac observations with the cardiogram and muscle tracings, to add strength to this opinion, but he had no doubt of its truth. A few weeks ago he had a long journey into the country in connexion with such a case as that of which he was speaking. There had been great talk of heart disease in a delicate child, when she suddenly fainted after going to church, and following upon some excitement. The child was put to bed for heart failure, with a prospect of having to remain there without being allowed to move for weeks. But there was nothing the matter with her. After recovering from her faint the heart was quite regular. The apex beat might be in a slightly different position from the normal, but in children this is a point that varies much and means little. He was amused at Dr. Mackenzie's remark that he had had a large amount of unpleasant correspondence with various doctors in regard to this condition, and he (the speaker) sympathized with him in that. The consulting physician was placed in a difficult position, for a patient came to him and said "Dr. So and So says there is heart disease," but the consultant thought there was no heart disease. In such a fix somebody was only too likely to be sacrificed, for the public had no mean between the wise man and the fool. The fact was, that general practice did not take time to make its diagnosis: because the patient had fainted, heart disease was jumped at; the patient was put to bed and compelled to remain there without the "wait and see" so requisite in all such cases. He pleaded for more time to be taken over the diagnosis, for more deliberation in committing oneself to an opinion—and the public needed to be taught much as regards functional disturbances of the heart that as yet they knew not. The treatment of all these cases was no doubt largely a matter of rest, but the crux was to decide how much rest was required, for even in cases of definite advanced heart disease there could be too much rest. Absolute rest for long periods in young children was not harmless. The patient in these doubtful cases should be allowed to get up as early as possible, under careful watching. Even in the case of children with heart disease one could order too much rest. He would mention one condition in the adult which bore on child life, and which had always seemed to him extremely interesting in this respect—namely, mitral stenosis. That rarely occurred in children, but on arriving at 16, 18, or 20 years of age it began to be more common. It was essentially a disease of females. He did not think it was ever found as a congenital disease; it was probably a rheumatic endocardial condition, leading slowly to thickening. The worst one could do for such cases was to put them to bed; as long as they could get about and lead a quietly active life they did well and could be kept comparatively well for a long time. He likened that condition to stricture of the urethra. Before that stricture became a serious disability the condition had been in existence maybe for years, but it had been kept more or less open by the passage of the frequent streams of urine. The same happened through the blood passing the valvular apertures in the case of the heart, and moderate exercise, he supposed, favoured the blood-circuit, and so tended to keep the contracting valve dilated.

Dr. John Hay (Liverpool): I believe that this discussion is of twofold value. It not only enables us to hear the considered opinions of authorities such as Dr. Cautley and Dr. Mackenzie, but it is also an opportunity for correctly and adequately emphasizing truths which are perhaps recognized, but which are not appreciated in their true significance. The ground for discussion is a wide one, and in order to limit it somewhat, one must ask in what way the treatment of heart disease in children differs from that in adults. It would be a waste of time not to confine one's attention to those points in which the treatment of the child suffering from heart affection is different from that of the adult.

In the heart of a child affected by rheumatism we are dealing with the first stages of an infective process; in adults, with the results of such process. Whatever views may be held as to the nature of rheumatism, I take it as accepted that rheumatic fever is due to infection by a definite organism. Many facts point to that organism being a streptococcus, but the exact nature of the coccus is more a matter for the pathologists than the clinician. As a result of this infection the heart suffers in practically every case, and there seems good reason to believe that the portal of infection is the tonsils and nasopharynx. The association of tonsillitis and nasopharyngeal disease with rheumatism is now so well confirmed that one cannot refuse the inference of a causal relationship.

Of all lines of treatment in children, prophylaxis is the most important and the most pressing. If rheumatic infection can be prevented, a large majority of children who become the victims of cardiac disease will escape, and this is the aspect which I would like particularly to emphasize. Accepting the view that an unhealthy or diseased nasopharynx predisposes to rheumatic infection—and the recent statistics by Dr. Branson certainly favour this view—we have no option but to attack with vigour all unhealthy conditions of this region. Enlarged tonsils, chronic rhinitis, and adenoids should all receive appropriate treatment. The nasopharynx must be rendered healthy. The medical inspection of school children provides many opportunities for attempting work of this kind. The importance for such treatment should be emphasized in discussion with the parents of the better classes.

I would suggest as another point of practical importance that every child who has manifested at any time signs suggesting a rheumatic infection, such as erythematous rashes, "growing pains," purpura, unexplained anæmia in a child of a quick nervous temperament, suspicious choreic-like movements or fibrous nodules, should be looked upon as a suspect, and even if in apparent health, should be taken as regularly to see his doctor as he is to his dentist, and the parents should be warned of the importance of the occurrence of any of these slighter rheumatic manifestations. In this way valuable time would be saved, and the earlier stages of cardiac mischief recognized before the heart affection has had time to progress to a dangerous degree.

I would like to hear the opinion of the meeting on the question as to what value is to be placed on alcohol as a cardiac therapeutic agent in diseases of children. Professor Kassowitz some years ago gave it as his view that he did not consider that there was any place for alcohol in the pharmacopœia of a children's hospital. Its regular use is probably condemned by all. As a vaso-dilator it is unnecessary in cardiac diseases as found in children. It has been demonstrated that alcohol minimizes the immunizing activities of the body in the presence of any infection. The patients suffering from acute rheumatism are suffering from an infection. Have we any grounds at present for believing that alcohol, when absorbed, acts specifically as a cardiac stimulant? So far as I know, there are none.

Dr. Charles W. Chapman: As the time allowed to each speaker is short I will confine my remarks to the treatment of the more chronic forms of heart disease. I would like to preface my remarks by a plea for a more hopeful view of these cases than that generally adopted. It is true organic disease of the heart cannot be completely recovered from, but restoration to a tolerable state of health is possible in many cases. Then, a hopeless prognosis, untinctured by the possibility of even temporary improvement, is a fearful blow to the parents. Moreover, such merciless pessimism is not justified by experience. Examiners in life insurance and of candidates for the Public Service not infrequently find valvular disease where it was not in the least expected. In exceptional instances valvular disease is compatible with length of days. A male patient, aged 59 years, consulted me on a matter unconnected with his circulation, but routine examination discovered a mitral systolic murmur which the patient stated he had had ever since a rheumatic attack during childhood. Again, I showed a woman, aged 82 years, at the Medical Society who had a typical presystolic murmur with thrill dating from early years. This patient was examined by many visitors at my clinique. A striking illustration is that of an infant aged 16 months, first seen in November, 1897, who had a blowing systolic murmur at the apex and heard at the left scapular angle. The child only weighed 14 lb., was very pale, and became blue on exposure. Alteration in diet brought increase of weight and of resistance to cold. I will pass over the notes until the child was aged 8 years, when the most critical examination failed to find anything abnormal in the heart. Menstruation commenced at the age of 11 years, and the child's health and development are at the present time fully up to the average.

The value of prolonged rest cannot be over-rated in heart failure of all degrees. In my experience this is generally ordered in severe cases of rheumatic fever, but it is less frequently insisted upon in subacute cases where, although the arthritic signs are slight, seeds of future trouble are being sown in the heart. On the other hand, I have seen children kept in bed long after the necessity for it has passed. It is entirely a question of actual dilatation or a tendency to it on exposure to moderate strain.

Success in treatment of heart disease, especially in children, does not consist in the judicious prescription of what are called cardiac remedies, but to a great extent in attention given to what may be called the "side shows" of the malady. The distended stomach must be relieved by carminatives and recurrence prevented by careful dieting and recourse to bismuth mixture, engorgement of the liver by a mercurial, relief to the kidneys by dry cupping and diuretics, pulmonic congestion by leeches or poultices; while in water-logged cases where the patient can scarcely breathe in bed he should be placed in an arm-chair and the legs drained as soon as possible. The contrast is very striking between cyanosis and urgent dyspnœa while the patient is in bed, and the comparative comfort which follows when the diaphragm is no longer pressed upon by the enlarged liver.

Prophylaxis is a pressing matter when convalescence has been established, and here also regard for small details is necessary. Light, though warm clothing, careful dieting, the selection of a suitable place for change of air when the patient can travel safely, the avoidance of late hours and occasions of over-excitement, hurried meals, and, later on, the length of lessons, are among the numerous points demanding the doctor's attention.

These matters, like the choice of suitable drugs, will vary with the individual case and can here only be referred to in general terms. The food should be of a nutritious and easily digestible character and varied as much as possible within those lines. Alcoholic stimulants are, as a rule, harmful, and the most objectionable form is that of the much advertised tonic wines to which such extravagant restorative powers are attributed. All exercises should be within the child's cardiac capability, the first sign of shortness of breath being the index that a safe limit has been reached. A heart cripple should not be allowed to play with strong children without supervision, for the temptation to do as others do may be too strong. Lessons should, as far as compatible with the patient's strength, be continued so as to relieve the weariness of a long illness and to encourage hopefulness.

Careful watch should be kept on the nasopharynx, especially the tonsils. Follicular tonsillitis has in many cases in my experience been the starting-point of rheumatic heart disease. The cheesy material exuding from the tonsillar crypts are, in my opinion, veritable factories of the rheumatic poison. The mouth should be examined in all cases of valvular disease with a view to the removal of all sources of septic infection, if present. I have recently seen two cases of ulcerative endocarditis, one in a child in whom were carious teeth, the other in a woman with pyorrhœa alveolaris.

The rôle of digitalis in the treatment of heart disease is a subject of lasting interest. At a meeting of the Therapeutical Section of this Society during the last session this question was discussed, and I was struck by a remark by Dr. Cushny[1] that digitalis was a heart tonic. When I was a student digitalis was generally looked upon as a cardiac depressant, and those who were privileged to receive teaching from the late Sir Samuel Wilks will remember how he insisted that the drug was a myocardial tonic. Theobromine and theocin sodium acetate are useful diuretics, so is the old digitalis, mercury, and squill pill in suitable doses. When treatment has to be continued over a lengthened period it is a useful as well as a merciful plan to withhold all drugs one day in the week.

Dr. J. Walter Carr said he would limit his remarks to the treatment of acute and subacute rheumatic affections of the heart in children, especially as the treatment of chronic heart disease in early life did not differ materially from that in adults. His experience was drawn mainly from cases in hospital wards, in which functional diseases were not usually met with. Dr. James Mackenzie and Sir James Goodhart saw patients in a different social position, and so they encountered the functional conditions. He had little to add to or criticize in what Dr. Cautley had said. First and foremost there should be absolute rest, its duration proportioned to the degree of carditis present and to the rapidity of the pulse; it should be more prolonged if there were pericarditis in addition to myocarditis and endocarditis, and longest of all when nodules were present, because they pointed to a specially severe form of myocarditis. However, in every case of rheumatic fever in a child the only

[1] *Proceedings*, 1912, v (Therap. Sect.), p. 202.

safe course was to assume that some myocarditis was present. His experience with regard to salicylates was similar to Dr. Cautley's. Repeatedly he had seen, in patients who were taking salicylate of soda, relapses of cardiac rheumatism occur, and even the supervention of pericarditis. A few weeks ago a boy, aged 9 years, was admitted under his care with an acute rheumatic attack; under the administration of salicylate of soda there was rapid improvement, and the temperature fell to normal. The boy was then given a reduced dose of 48 gr. of the drug daily and was kept entirely recumbent; but after the temperature had been normal for a week acute pericarditis developed. He could recall many similar occurrences. It was true he had seldom tried the very large doses which Dr. Lees recommended, but in the one or two cases in which he had done so, cardiac weakness and irregularity developed so markedly that he was thankful to revert to smaller doses. He had many times employed the treatment with which the name of Dr. Caton, of Liverpool, was associated—viz., prolonged rest, with blistering over the præcordia, and moderate but prolonged doses of iodide—but, like Dr. Cautley, he had not obtained any marked benefit from it beyond what he should expect from rest alone. With regard to the ice-bag, he generally used it in acute pericarditis, and it often gave some relief. If the child liked it, he persevered with it so long as any friction could be heard, if not, he gave it up. He did not consider it had any definitely curative effect. As to paracentesis pericardii, he would summarize that briefly by saying that for all practical purposes it was never required in rheumatic pericarditis. During convalescence, although these patients were usually very anæmic and iron seemed to be indicated, yet he had found that it frequently disagreed and seemed to increase the tendency to relapse. He would be glad to know whether the experience of other clinicians coincided with his in that respect. As to the use of digitalis in these cases, he knew of no truer or more valuable aphorism in Dr. Mackenzie's writings than that in which he said that digitalis was useless when the heart was in the grip of a toxin. He (Dr. Carr realized this so much that now he practically never used it in the acute or subacute stages of rheumatic fever; he had never seen it do any good then, or slow a rapidly acting heart; and if it did no good it was likely to be harmful. There was, however, one drug or rather food which he had found to be particularly beneficial in the convalescent stage—namely, cod-liver oil; not greatly diluted with malt or other things, but pure He gave it in doses of half a teaspoonful, increased to one teaspoonful two or three times a day. Children took it very well, and it seldom disagreed. Massage and regulated movements he had tried in some cases, but had not been able to attribute to them any markedly beneficial results.

To summarize, it must be admitted that, to a large extent, until there was available an effective rheumatic vaccine or antitoxin the treatment of rheumatic carditis was merely palliative. In a few cases there was no improvement; the child went steadily from bad to worse, there were successive relapses, increasing dilatation of the heart, and finally death supervened. Fortunately, as a rule, gradual improvement occurred, tempered from time to time by the tendency to relapse. Though a mitral murmur usually persisted, there was often very little dilatation, and the child left the hospital with comparatively slight damage to the heart, and his prospects in life not seriously impaired. In order, however, to achieve such favourable results early treatment was essential. As in childhood the joint pains were usually so slight, only too frequently, especially among the poorer classes, patients were allowed to go about while suffering from active heart disease, and were brought to hospital weeks later with severe dyspnœa and great dilatation of the heart. Treatment then was well-nigh hopeless. Secondly, one had to guard against the tendency to relapse so often seen in these cases. That question, however, had been so fully considered by Dr. Hay that he would not further refer to it.

Dr. G. A. SUTHERLAND: We have had brought before us to-day the two great classes of cardiac troubles in childhood—namely, those due to organic disease of the heart and those due to functional disturbance.

As regards the functional disturbances, Dr. Mackenzie has made a very definite pronouncement and his views are certainly not those which are commonly held or commonly taught. When I say taught, some qualification is perhaps necessary because there is really very little teaching on the subject. The student is taught that the heart ought to have a certain size, certain sounds, and a definite regularity of action, and he naturally infers that anything outside these prescribed limits must be pathological and must require treatment. It has been left to ourselves to find out in the course of practice what exceptions, if any, to an orderly and regular action we are to allow before pronouncing a heart to be functionally and organically sound. These exceptions have been dealt with by Dr. Mackenzie in a most definite manner, and he would apparently extend the application of the term "a healthy heart" in childhood far beyond what we have been accustomed to accept. I shall leave to others the refutation of his views for the reason that I accept his teaching most thoroughly. The irregularities and murmurs he has described have undoubtedly been the cause of much and prolonged and unnecessary treatment in the past, and it is to be hoped that the authoritative statement we have listened to will check this tendency in the future. When Dr. Mackenzie describes these irregularities as "manifestations of a healthy heart," I cannot quite support him from any personal experiences, and he does not appear to me to have shown this so conclusively as he has the main part of his thesis—namely, that these irregularities are not manifestations of a diseased heart.

Still dealing with the question of functional disturbance, we find that there are many subjective symptoms which to the lay mind suggest heart trouble, and which to the puzzled medical mind may also suggest "a weak heart" as the most convenient form of diagnosis. Thus, breathlessness on exertion, palpitation, faintness and fainting, and præcordial distress, are known to be frequently associated with serious cardiac disease, and the tendency sometimes is to accept their presence as evidence of latent cardiac disease and to prescribe a course of cardiac treatment. The frequency of these subjective symptoms in children in the absence of any organic heart disease is very striking, so much so that their presence is strong presumptive evidence that the heart is not the cause of the disturbance. As a general rule such symptoms are associated with a disturbed state of the nervous system, of which other evidence will usually be found. If they are cardiac in origin, one may confidently expect to find definite signs of heart disease on physical examination.

The treatment of organic heart disease has been discussed very fully by Dr. Cautley, and I shall only refer to one type of case. Dr. Mackenzie has elsewhere laid stress on the importance of basing our prognosis and treatment on the symptoms produced by heart failure rather than on the conditions found on physical examination of the heart itself. This applies very generally, I believe, in the case of adults, but in the case of children it requires some qualification. Cardiac disease in childhood, with few exceptions, is due to rheumatic infection, but the vast majority of cases, after recovery from an acute or subacute attack, present few or no symptoms. The mischief has been done, the valvulitis, or the myocarditis, has reached a quiescent stage, and the functional power of the heart is unimpaired. In such a case, however, I should not form a prognosis on the symptoms, or rather on the absence of symptoms, but consider that a physical examination of the heart may reveal definite and serious changes of a permanent character. The results may not appear during childhood or adolescence, but will tend to come on during adult life. In a certain proportion of the cases, under suitable treatment, there will be no sequelæ even in adult life, and a functionally healthy heart is secured. There is no call at this stage for any cardiac treatment. What is required is to protect the child as far as possible from fresh rheumatic attacks, to treat these most carefully if they do occur, and to guard the child against any severe muscular strain. If a child is left alone to pursue its own forms of exercise it will naturally tend to limit them in such a way as to avoid cardiac strain. Just as Dr. Mackenzie has protested against making invalids of children because of some functional disturbance, so I should like to protest against making invalids of children with organic heart disease, but without any symptoms of cardiac disability.

Dr. Alexander Morison assumed that when Dr. Cautley said functional cardiac disorders in children were unimportant he referred to rhythmical disorder. The first of the irregularities shown on Dr. Mackenzie's diagram would probably occur when the child was in a parlous condition, and there could be no question as to its serious significance. The second and third were negligible, because the former was very rare and the latter harmless. The fourth he did not know was regarded as novel, because sixteen years previously he had drawn attention to that condition as a respiratory irregularity, and was under the impression that it had always been regarded as such. In regard to the acute condition, Dr. Cautley had emphasized the point which the late Dr. Sturges commented on very fully. Dr. Cautley had spoken of myocarditis as the key to the situation, and very interestingly mentioned the fact that one was dealing with the growing heart. Sturges insisted that in the young one was dealing not so much with endocarditis, or myocarditis, or pericarditis, but frequently with a generalized carditis; and no doubt the character of the tissues promoted this spread. Digitalis it had long been known, when used in any febrile condition, failed to have influence in checking the rapidity of the heart's action. But, no doubt because of the traditional dread of the consequences of the use of the drug, opium was not, in his opinion, pushed in children as it should be. Owing to the constitutional excitement, one found in a child a kind of pathological belladonnaism—a quick pulse, dilated capillaries, a dilated pupil, and thus a standing physiological antidote to over-dosage. As a rule too little opium was given in the treatment of acute heart disease, properly so-called in children, and the dread of it was greater than was justified.

With regard to the chronic conditions, much depended on whether the heart was tethered to its surroundings. In the condition which he was in the habit of terming cardiac infantilism, one found a patient 15 or 16 years of age with the development of a child of 10 or 11 years. This condition is usually met with in children with large *adherent* hearts due to disease acquired early in life. The child was very anæmic and undergrown, puberty was delayed, and after death he had found the thyroid gland smaller than normal. For that reason it had been his custom to give these children thyroid extract, in the expectation that the enlarged and labouring heart would thereby have its activity supported and its power conserved. In exophthalmic goitre with excited heart one sometimes saw the heart lessen in activity and the thyroid gland undergo shrinkage and change. In the cases he had referred to, commencing early in childhood and showing cardiac infantilism, there was apparently an inadequacy of that gland in consequence of the excessive work the heart had had to perform.

The only other point, for lack of time, which he could refer to was that of cardiolysis, which was practised for the relief of these adherent cases. His experience of that operation had not been very happy. His most successful case was that of a young man who had been under his care since his thirteenth year, and had been much relieved by a removal of ribs which afforded more space for the heart. He did well for four years, and then had an opportunity of going to a convalescent home apparently in his normal health; but six weeks later appeared to have acquired infective endocarditis and he came back to hospital with manifestations of that condition. He died in about a month. His pericardium was found post mortem to be totally adherent to his præcordial skin, which in no way impeded the heart's action. So long as there was no drag on it, the fact of the pericardium adhering to the præcordial skin appeared to be innocuous. Another case was that of a boy who was operated upon for costo-pericardial and visceral adhesions. He lived for three months and was apparently relieved by the operation. He then had pericarditis with effusion and died. Another child, the subject of cardiac infantilism, had an enlarged and adherent heart, survived operation and seemed comfortable, but died with a clot in the pulmonary artery. Probably the principle of the operation was right, and its practice in carefully selected cases justifiable.

Dr. F. W. Price: In my view the prevalence of heart disease in children is much greater than is commonly supposed, the reason being that it is very easy to overlook a rheumatic infection of the heart in childhood unless we are especially on the look-out for it, and even when we are on the look-out it is often very difficult to come to a definite conclusion as to whether the heart is affected or not. It seems to me that even yet it is not sufficiently widely realized that rheumatism as it appears in childhood presents many important points of difference from the disease as it appears in maturer life, and the non-recognition of this fact is the reason why damage to the heart by rheumatism is so frequently overlooked. Acute carditis should not be looked upon as a complication of rheumatism; it is just as much part of the disease as is the affection of the joints. It follows, therefore, that the points which conduce to successful treatment are prophylaxis and early diagnosis. When there is no doubt that the heart is implicated by rheumatism, absolute and *prolonged* rest in bed is essential. In my view salicylates are of value, and I believe that the method of administering sodium salicylate introduced by Dr. David Lees is more efficacious than the usual method. I have been disappointed with the results of Dr. Caton's plan of treatment.

Passing to chronic affections of the heart, Dr. Mackenzie has dealt very fully with the possibility of mistaking certain cardiac phenomena for manifestations of an impaired or diseased heart and as indications for treatment. It seems to me that that danger is much more relevant to the primary purpose of this discussion than might at first sight appear; for it is certainly of vital importance that when the practitioner on examination discovers what he thinks are abnormal cardiac phenomena he should be able to determine without any possibility of doubt whether those phenomena are evidences of an impaired or diseased organ before deciding whether any or what treatment is necessary or advisable. The subject of irregularity of the heart was gone into in some detail. I venture to suggest that even yet it is not nearly sufficiently generally understood that the problem of cardiac irregularity, which formerly was notoriously a problem which baffled clinicians, has now been mainly solved. There is just one point I should like to add in regard to cardiac irregularity—namely, that in the case of intermission of the pulse it is important to determine whether the intermission is due to extra-systoles or to partial heart-block; since in the case of extra-systoles there is not sufficient evidence to lead to the view that, taken by themselves, they are indicative of an impaired or diseased organ, whereas partial heart-block may be the only sign of myocardial damage.

Now, given a case of chronic heart disease, it is of the utmost importance, firstly, that the fact of damage should be recognized: and. secondly, that its degree should be approximately estimated. In regard to both of these points I quite agree with the second speaker that while the results of physical examination (such as the amount of enlargement) are important, the question of the efficiency of the working power of the heart is of supreme importance, and that in all cases it is our duty to try and find out if this is impaired, and if so, in what degree.

We may divide treatment into two parts: (1) that which consists in an endeavour to delay the onset of cardiac failure as long as possible: and (2) that which is concerned with the management of cardiac failure when it does arise. In regard to the first, one of the most unfortunate of things is the fact that such a large number of patients with damaged hearts endeavour to live beyond the limits of the heart's power—that is, as though their hearts were normal. It is of supreme importance that this should be avoided. The medical attendant will have done the greatest service if he succeeds in getting the parents to try and educate the child to acquire the habit of always living within the limits of the heart's strength. It is impossible to give hard-and-fast rules. But a cardinal principle is this: that any exertion which the child indulges in should not produce symptoms of cardiac distress, whereas any exertion which falls short of producing these is usually not harmful. In my opinion, what is called the Nauheim plan of treatment is not advisable in the case of children. I think Swedish exercises in strict moderation are sometimes of value. When, in spite of a carefully regulated life, there are indications of progressive exhaustion of the heart's strength, the amount of effort should be proportionately reduced, and rest in bed, it may be for a long period, is often necessary. In my experience prolonged rest in the recumbent posture is of considerably greater value in cases without auricular fibrillation than in cases with it; and for this reason in cases without auricular fibrillation we should be careful to avoid crediting drugs with results which are in reality due to other causes.

In regard to the employment of the digitalis series of drugs, what I have to say is largely based upon investigations in which I have been associated with Dr. Mackenzie and Professor Cushny at the Mount Vernon Hospital. In the great majority of cases there is a remarkable difference in the response to these drugs between cases with auricular fibrillation and cases without it. In cases with auricular fibrillation brilliant results often attend the administration of these drugs. It is best to commence with large doses, and continue the administration until some definite reaction is obtained. A considerable proportion of cases of auricular fibrillation relapse after the drug has been stopped. If this occurs, we should then try to find out what dose will best maintain the improvement without producing any toxic symptoms. In no case in which digitalis failed did any of its allies succeed. Therefore, digitalis appears to be the most efficacious. In no case in which the tincture of digitalis failed did any of the other preparations of that drug succeed. We found the different samples of tincture of digitalis supplied by good firms of chemists wonderfully constant in strength. In regard to the so-called alleged active principles of digitalis, it cannot be too strongly insisted that there is no pharmacological or clinical evidence that they are either pure substances or that different samples of them are uniform in potency; and therefore there is no reason to believe that they are more reliable than, and possibly they are not so reliable as, different samples of the tincture. I have conducted some investigations on the action of digitalis on the blood-pressure in man, and found that, judged by the methods in use for observing the blood-pressure clinically, the administration of the drug is not attended by a rise in pressure. As a rule, considerably large doses of strophanthus are required, but this drug shows great variation in regard to dosage. As a rule, squill has little effect upon the heart, and larger doses are required than digitalis. Strophanthus and squill appear to have a greater tendency to produce diarrhœa than digitalis. I have conducted some exhaustive investigations on the action of tincture of aconite on the pulse-rate, and found no evidence to lead to the belief that it has any effect whatsoever. When pyrexia is present digitalis appears to be ineffective, whether auricular fibrillation is present or not. In the most acute cases of cardiac failure, when it may be desirable to elicit a more rapid effect than is possible by the administration of tincture of digitalis by the mouth, intravenous injections of strophanthin may be employed. Sometimes they are followed by marked improvement in from four to eight hours.

Lastly, in my experience digitalis is in the vast majority of cases of heart failure the best diuretic; but I have sometimes been agreeably surprised with the results following the administration of theocin sodium acetate.

Dr. Bezly Thorne said that he held strongly to the view that irregularities of pulse frequency and functional murmurs are signs of impairment of the heart muscle and of the arterial tunica media; and that, even so, they are expressions of a syndrome of which they are not the fundamental constituents. They occur associated with a dicrotic pulse marked by absence or feeble development of the predicrotic and post-dicrotic waves, the tracing of which shows that the arteries, not having received their normal fill of blood, are empty during an undue proportion of the cardiac cycle. Such phenomena are associated with two important conditions: a deficiency of arterial recoil and consequently of conductivity, and an actual defect of myocardial energy, the importance of which is relatively aggravated by the fact of the central organ being called upon to perform a considerable part of the work which should be carried out by the arterial heart. Under some circumstances the impoverished tunica media seems to pass temporarily out of action (*vide* figures), and it is easy to conceive how great may be the danger of heart-strain if a strenuous effort be persevered in while that condition lasts. Another symptom of the condition under consideration is a measure of postural tachycardia. Whether the pulse be abnormally frequent during repose or not, it will be unduly accelerated by the act of rising from the sitting or recumbent postures, and in some cases remains so for an indefinite period, unless a posture of repose be resumed. The murmurs also are so largely influenced by posture that in many cases they disappear on the patient standing up, reappear on his sitting down, and are loudest while he is recumbent; that is, as Dr. Foxwell suggested some years ago, while a distended right auricular appendix is leaning its weight on the root of the pulmonary artery. It is difficult to determine whether the systolic apex murmur, when it is present, should be attributed to papillary weakness or to asthenic dilatation of the auriculo-ventricular ring. Irregularity of the pulse is liable to be induced or aggravated by any additional demand on myocardial energy, and by vaso-dilatation however induced, but in some of the less severe cases the balance may be redressed by the stimulus of moderate exercise. It should be added that there is generally some dilatation of the ill-nourished and incompetent myocardium.

As regards the ætiology of this affection, careful investigation, especially when it can be carried back to the early stage of life, lights on some form of autotoxis, and in the greater number of cases on that of gastro-intestinal origin. In others a rheumatic or gouty taint

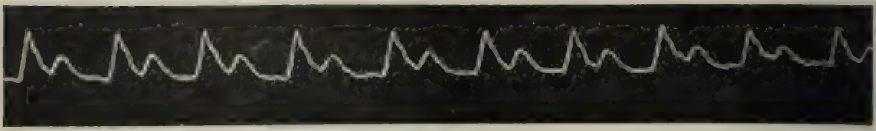


Fig. 1.

Male, aged 18 years. "Functional" bruit at apex from the age of 4 to 10 years. Broke down after a game of rackets at public school, summer of 1911. Typical pulse of cardio-vascular atony.

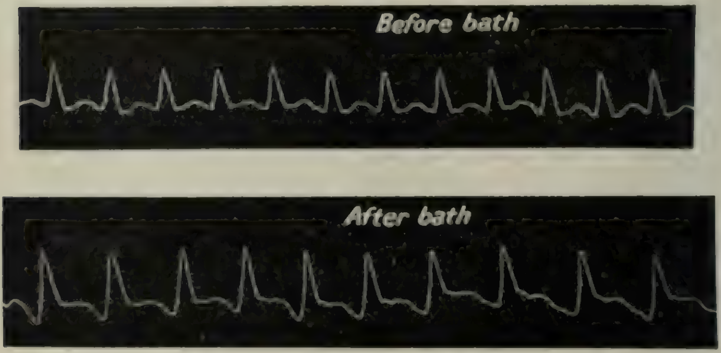


Fig. 2.

First day of treatment.

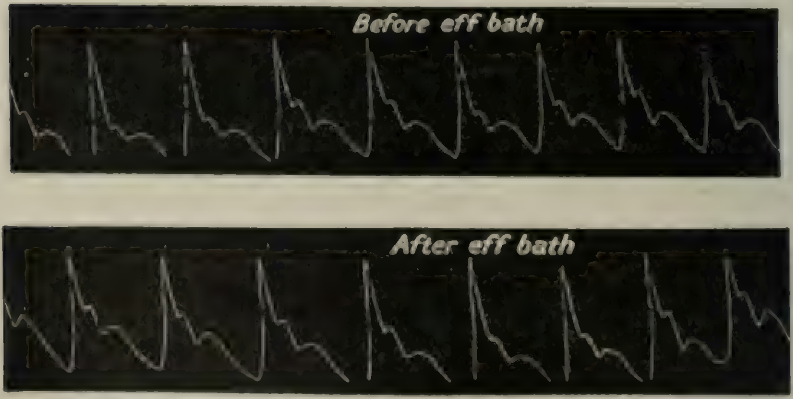


Fig. 3.

Last day of treatment.

Figs. 2 and 3.—Civil engineer, aged 30 years. Seen in adolescence with symptoms under consideration while studying for examination on account of "slackness" and loss of power of mental concentration. Broke down in India. Treatment, February and March, 1912. Nauheim baths, adrenalin, gastro-intestinal correction.

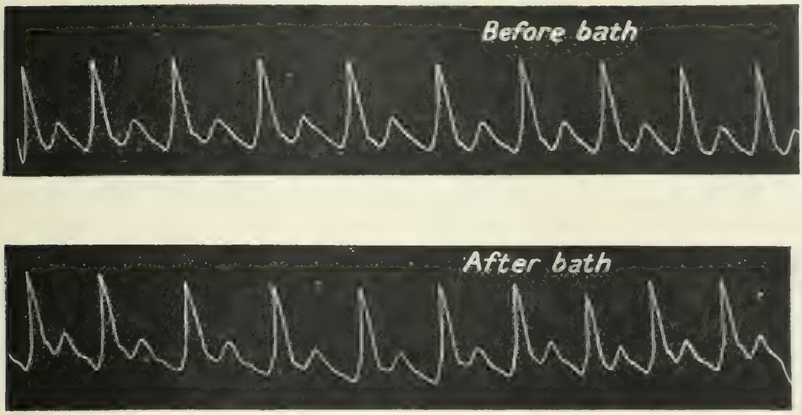


FIG. 4.

First day of treatment.

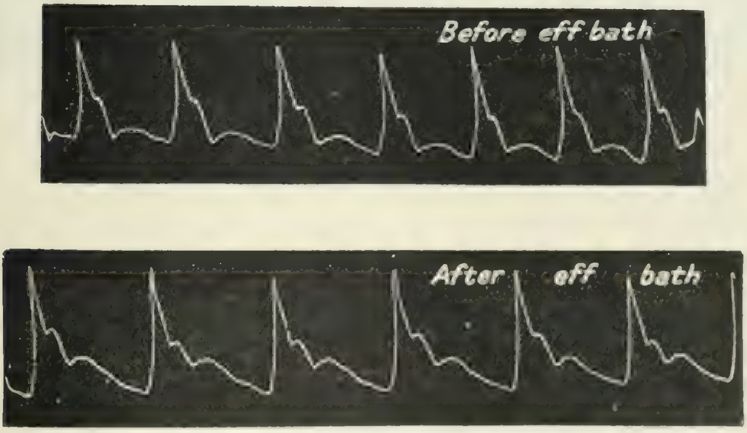


FIG. 5.

Last day of treatment.

Figs. 4 and 5.—Army officer, aged 39 years. Nutritional and digestive troubles from childhood. Always "slack." Resigned commission on account of inability to stand manœuvres or spend a day in the saddle. Treatment: Nauheim baths (August and September, 1911), adrenalin, gastro-intestinal correctives.

seems to bear a causative relation to the syndrome. Not a few of its subjects bear the rachitic stamp, in the form of beaded ribs, retraction of the lower part of the sternum, or a tendency to pigeon breast. But whatever the cause, the result is impairment of power and efficiency and defective ability to bear strain, whether mental or physical. Such children and adolescents are liable to become the subjects of the strained heart usually attributed to athleticism. But sound and healthy lads do not strain their hearts in the games and sports of school and university life; while the subjects under consideration, short of wind and lacking in staying power, may meet misfortune in a football match, a paper-chase, a boat race, or a game of rackets.

TRUNCATED APICES. TUNICA MEDIA OUT OF ACTION

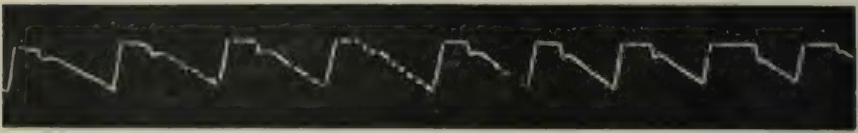


FIG. 6.

Woman, aged 30 years. Menstruation. Bases of waves vary from 12 mm. to 15 mm. in horizontal measurement (taken in bed).

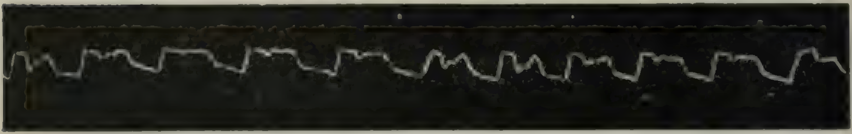


FIG. 7.

Girl, aged 17 years. After three-quarters of an hour's walk on level ground. Bases of waves vary 10 mm. to 12 mm. in horizontal measurement (taken in sitting position).

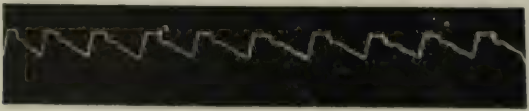


FIG. 8.

Woman, aged 50 years. After journey from Bournemouth and drive across London (taken in bed).

If they escape acute trouble, some live on with less than normal energy, able to meet with the requirements of a methodical life; others, unequal to the strain of an active life at home or abroad, more especially in sub-tropical and humid climates, pass through periods of invalidism. Some such have been lulled into security by the assurance that there is nothing organically wrong with them. As they approach or pass beyond middle life, the atrophied tunica media and the weakened myocardium, no longer able to do the double work of filling the aorta and driving the blood through the peripheral vessels, entail the troubles and disabilities characteristic of what is generally referred to as arterio-sclerosis, but as it might, perhaps, be better called, atrophic dilatation, as opposed to the hypertrophy of the media to which the late Dr. Savill gave the name of "hypermyotrophy."

The treatment of such cases must be primarily based on correcting, and guarding against, autotoxis of any and every kind. A careful application of the Schott method of the administration of baths or exercises stands next in importance. Among drugs, the solution of adrenalin, given by the mouth in doses of from 5 to 10 minims, three times a day, is pre-eminent as a means of recalling what remains of the tunica media to functional activity, and of so relieving the work of the heart. Thus administered it never raises the blood-pressure; nor, for that matter, does digitalis.

Dr. Bezly Thorne added that, for the reasons which he had indicated, it would be a misfortune were the thesis to be accepted that functional murmurs, and irregularities of pulse-frequency, ought to be regarded with complacent acquiescence as consistent with a state of perfect health.

Dr. CAUTLEY, in reply, said that in regard to the remarks he made in the paper as to the infrequency of heart disease of a functional nature in children, the views taken by Dr. Mackenzie and Sir James Goodhart in opposition to his own seemed to be based upon heart affections not exactly in children, but in young persons about puberty and adolescence. In his experience, functional heart disease—not mere irregularities due to indigestion or alterations in respiration—was distinctly uncommon. Paroxysmal tachycardia and the neurotic heart were comparatively rare. Many of the cases of so-called functional heart disease in children had a myocardial origin in some infective disorder.

and must be treated by means of prolonged rest. He did not mean that irregularities of the heart and functional murmurs were not met with in children. Sir James Goodhart's remarks seemed to imply—though he knew he did not hold such views—that faintness was a sign of heart disease. It was neither that nor even a sign of cardiac disorder, but was a vasomotor phenomenon depending on the nervous system. No one with a sound knowledge of medicine would say that a girl, because she fainted, had organic heart disease.

Dr. JAMES MACKENZIE, in reply, said that his wish was that his remarks might be pondered over, and the point of view carefully considered by those who had the opportunity of observing individual cases for long periods.

Section for the Study of Disease in Children.

January 24, 1913.

Mr. A. H. TUBBY, President of the Section, in the Chair.

Case of (?) Fibro-lipoma.

By DUNCAN C. L. FITZWILLIAMS, F.R.C.S.

A BOY, aged 2 years 8 months, has been under the care of a foster-mother for two years. When the foster-mother first took care of him she noticed small lumps on the inner back part of the right arm. This gave rise to no trouble. There was no swelling on the chest at that time, but in December last she noticed a swelling on the right side of the chest; latterly this has become a little larger, and she therefore brought the child up for treatment.

The child never complained of pain in the lumps nor do they seem tender. On examination there were four small swellings, distinct from one another, on the back of the arm, and in the axilla they feel fibrous, some being larger than others. The axilla is filled with a mass about as large as an orange, which bulges forward the anterior axillary wall and appears in front under the pectoral muscles. It is firm, with well-defined edges, and slight lobulation. It is movable on the structures beneath and under the superfacialis. There is no impulse on coughing or crying.

The child is very backward and undersized, with marked signs of rickets. The fontanelle is still open.

Case of Lymphangioma resembling Hernia of the Lung.

By DUNCAN C. L. FITZWILLIAMS, F.R.C.S.

A BOY, aged 6 years. The mother has noticed that something was wrong with the right side of his neck when he cried. She states that she noticed this vaguely when she was washing the child, and she can recall it as far back as twelve months ago, but she took little notice of it until it began to get larger during the last three months. In November the child got whooping-cough and this lasted until the end of December. During this time his mother noticed that the lump in his neck got very large during the spasms of coughing.

On examination, there is a fullness of the root of the neck above the right clavicle and behind the sterno-mastoid muscle. It is ill defined and quite soft, and small nodules can be felt in it. It seems to disappear under the fingers towards the region of the subclavian artery. On coughing or straining the root of the neck fills up and bulges in a prominent manner, and on palpation the tumour again subsides. Percussion is unsatisfactory as there is so little resistance, and though breathing sounds are heard they are probably transmitted.

The condition at first sight is extremely like a hernia of the apex of the right lung, but there is no indrawing on deep inspiration, nor can any ring be felt in the deep part of the neck, and the diagnosis is that the condition is an extensive lymphangioma with large loculi, the large portion of the tumour being situated in the chest.

Dr. PORTER PARKINSON thought that the physical signs were not clearly those of lymphangioma, as similar signs could have been found in association with hernia of the lung, and the latter were more common in the upper than in the lower part of the neck. There was also a very loud breath sound, giving the impression that one was nearer to the lung than when listening over the chest. The resonance with one finger on the clavicle was very good, but with the second finger higher up there was not a resonant note. The fact that the lump entirely disappeared with a deep inspiration favoured the diagnosis of hernia of the lung.

Two Similar but somewhat Unusual Heart Conditions in Sisters.

By R. C. JEWESBURY, M.D.

M. W., AGED 8 years. The mother was told by her doctor that this child's "heart was affected," when she was 3 weeks old. The child was not cyanosed at birth and has never been noticed to get blue. She has suffered from dyspnœa on exertion for the last three to four years and has complained of growing pains during the last two years. No history of acute rheumatism. She has had measles twice, but no other illness. Heart enlarged. Apex beat in sixth space in nipple line; systolic murmur at apex conducted out, second sound accentuated. In the second and third left space a loud, rumbling murmur, partly presystolic and partly systolic, is heard, and a well-marked thrill is felt in this position. At the pulmonary area a systolic murmur is present, conducted upwards to the neck. Aortic sounds: First sound soft, otherwise normal.

E. W., aged 10 years. No heart disease was ever suspected until she was brought to hospital a few days ago. She had always been a healthy child. No history of rheumatism; heart not enlarged. In second and third spaces a well-marked thrill (systolic), and over this area a prolonged, continuous, booming murmur is present, very similar to that heard in her sister's case.

DISCUSSION.

Dr. JEWESBURY said he felt no doubt that the murmur was due to congenital heart disease, though it was difficult to say what the exact lesion was. The diagnosis lay between imperfect ventricular septum, with, perhaps, some pulmonary stenosis, though the entire absence of cyanosis made the latter improbable, or else patent ductus arteriosus. The interesting fact was that the same congenital defect was present in two sisters of the same family. The younger of the two girls had some acquired heart disease in addition. Three other children in this family had normal hearts, and both parents were healthy.

Dr. LANGMEAD suspected that these were examples of patent ductus arteriosus. The murmur, which was of a to-and-fro character at the base, was suggestive of that, as well as the way in which it was conducted upwards towards the clavicle, and the ease with which it was heard at the back. There was also an unusual kind of shadow at the cardiac base in the skiagram. These points, taken together, seemed fairly strong evidence of patent ductus arteriosus. The absence of an increase of dullness to the right could not be relied upon very much, because it was likely that one or other ventricle was particularly small, and the cardiac borders might not be far from the normal. With regard to the incidence of congenital heart disease in children of the same family, last week he saw two children of the same family, both of whom had a congenital systolic murmur in somewhat the same position, but he did not think the diagnosis was patent ductus arteriosus in those cases. The murmur was a simple systolic one in both cases, not to and fro.

Three Cases of an Unusual Form of Disease of the Hip-joint—"Calvé's Pseudo-coxalgie."

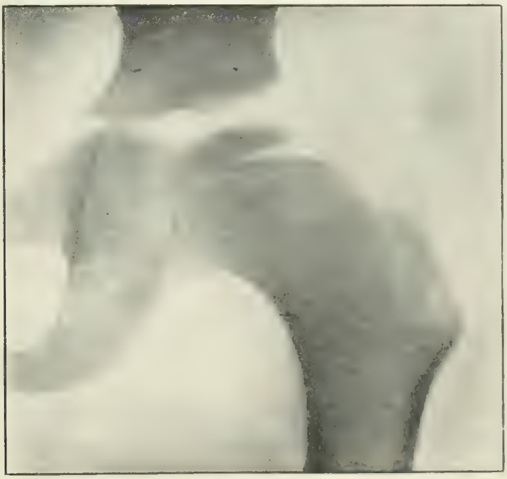
By R. C. ELMSLIE, M.S.

Case I.—A. H., male, aged 10 years. In November, 1909, he fell and twisted his right foot: attended an out-patient department for a week and recovered. About three weeks later he was kicked on the right hip, which was noticed by the mother to be much bruised; from that time he limped and had occasional pain in the knee, especially after lying down. He had no pain at night. In the following March he came to the hospital on the medical side, was noticed to be limping, and referred to the surgical side. At that time (March, 1910) the hip movements were free except full hyperextension; the limb was ⅜ in. shorter than the left, the shortening being above the great trochanter; there was little wasting of the thigh. A skiagram showed that the acetabulum was clear and smooth, but that the epiphysis of the head of the femur was a little flattened, its surface uneven, and its upper edge proportionately thicker than it should be; the neck of the femur appeared shorter and thicker than the normal. He was kept in bed for three weeks and the condition did not alter. He was then allowed up. A fresh skiagram taken in May showed no alteration. After this he was kept under occasional observation until February, 1912, during which time the condition did not appreciably alter. Subsequent skiagrams showed no important change, although the flattening of the epiphysis and the thickening of its upper border became rather more evident.

Case II.—C. S., male, aged 8 years. The parents in this case had not noticed anything wrong, but were informed after a school medical inspection that the boy limped. He was brought to the hospital in January, 1912. At that time there was little limitation of the abduction of the right hip-joint, most marked in the flexed position; other movements were free. The shortening was very slight, not more than ¼ in., and there was a little wasting in the thigh. A skiagram was taken, and the appearances were found practically identical with those in the last case. The parents were then informed that no treatment would be necessary.

These two cases agree as to clinical signs and skiagraphic appearances with Calvé's description, but neither was observed in the acute stage described by that author. The third case presents, however, some differences.

Case III.—D. H., female, aged $7\frac{1}{2}$ years. At the beginning of 1912 this child had an attack of St. Vitus's dance and rheumatism in the right leg. In the summer she was an in-patient at a hospital for St. Vitus's dance; the hospital notes at that time do not mention any abnormality of the hip. She was transferred to a fever hospital for diphtheria in August. Whilst convalescent of the attack of diphtheria she was noticed for the first time to limp, but she had at this time no pain or disability. When she returned from a convalescent home the mother noticed the limp, and brought her to St. Bartholomew's Hospital in December. No injury to the hip could be remembered. The right hip is kept flexed, further flexion is free, extension is limited, abduction abolished, and rotation in both directions limited. The limb is $\frac{3}{8}$ in. shorter than the left, this shortening being above the great trochanter. A skiagram shows the head of the femur to be deformed in much the same way as in the previous cases, but in addition the upper part of the acetabulum is eroded, and the head of the femur displaced a little upward; the femoral neck is short and thick.



Calvé's pseudo-coxalgie. Case II, C. S.

Cases I and II certainly, and Case III probably, belong to the class described by Calvé as "une forme particulière de pseudo-coxalgie."[1]

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[1] *Rév. de Chir.*, Par., 1910, xxx, pp. 54-84.

The absence of serious symptoms and the presence of deformity of the head and neck of the femur from the first are characteristic, and the skiagraphic appearances are exactly like those described by Calvé. Such cases are not rare. That the condition is not tuberculous is indicated by the occurrence of deformity before the onset of any notable symptoms, by the slightness of the symptoms and their rapid disappearance, and by the fact that none of the cases that have been described have suppurated. There is no evidence to support the theory of any other specific form of infection. Calvé expressed the view that there is a disturbance and retardation of ossification of the epiphysis, due to rickets, the subacute arthritis from which the patients suffer being an added infection of an avirulent nature of a *locus minoris resistentiæ*. There is very little evidence for the theory of a rickety origin, but that the lesion includes a retardation and disturbance of ossification must be agreed. The suggestion may be made that this disturbance is similar to that described in the tubercle of the tibia by Osgood and Schlatter, in the os calcis and in other exposed epiphyses, and that it is possibly traumatic in origin.

Acetabular changes are clearly shown in the last case, and it may be thought that this case should be placed in a different group, but on careful examination it will be seen that the acetabulum is a little enlarged in the other cases and it also appears to be so enlarged in some of Calvé's, although the reproductions of the skiagraphs in the latter's paper are scarcely good enough to enable one to be certain upon this point. Probably this case is one of the same group, in which, however, a rather more acute and destructive inflammatory stage has occurred, with enlargement upward of the acetabulum and subluxation of the femoral head.

The most important point in the treatment of these cases is that they should be diagnosed from tubercle and should not be treated by immobilization. They require temporary rest whilst the hip is painful, beyond that they are best left alone. The third case will be treated by a rack splint to secure an increased range of movement in the direction of extension and abduction, and then by a calliper splint.

DISCUSSION.

The PRESIDENT (Mr. A. H. Tubby) said the Section was much indebted to Mr. Elmslie for the opportunity of seeing these cases; he believed they were the first of the kind which had been shown in this country. He read Calvé's article three or four months ago, and found a very clear, clinical picture of the cases. He pointed out that they were a well-defined class of hip-joint lesion. Calvé said the striking points were, first, the association of these conditions with other deformities, such as coxa vara; secondly, that there were often other rickety stigmata present, and great stress was laid on the alteration of the shape of the epiphyses at the head of the femur, for whereas the normal epiphysis was always hemispherical, in this condition it became flattened, spread out, and disk-like. One particular point dwelt upon was that the growing points of the epiphysis were scattered in the way one of Mr. Elmslie's skiagrams showed. Calvé also said there was a curious encroachment of the distal part of the epiphysis of the neck upon the head of the femur—i.e., it seemed as if the upper third of the femur was made up more of neck than it should be and was dwarfed in proportion. Calvé observed that the head of the femur appeared to be much too prominent in Scarpa's triangle; he did not believe the cases were tuberculous or syphilitic, but regarded them as more in the nature of rickets. With regard to treatment, as Mr. Elmslie said, the condition was transient and passed off in a few days. The important point was to recognize the condition. In three or four cases he (Mr. Tubby) had noticed this flattened shape at the head of the femur in the ordinary routine of hospital work. One was associated with coxa valga and one or two with coxa vara. Now that Mr. Elmslie had brought forward cases, doubtless many more would be shown. He would like to know what the exhibitor thought was the nature of the infection in the third case, which allowed the head of the femur to shift upwards and become a pathological dislocation.

Mr. ELMSLIE replied that he did not think such cases were rare, for he had seen two cases in his ordinary out-patient work at the Metropolitan Hospital, which was not a large one, and he had seen several more in other out-patient departments, all in the course of two years. Further, the symptoms were so slight and transient that probably many cases never came to hospital. He would probably not have recognized the condition but for his routine practice of examining every case of possible hip disease by means of a skiagram. In answer to Mr. Drew, he denied that there was any erosion of bone in the cases, but there was an insufficient formation of bone in the epiphysis at the head of the femur. The cartilage surface, he believed, was intact. He was not referring to the third case, as he had put that down as doubtful. Calvé said the absence of erosion of the joint surfaces was the remarkable feature of the condition. With regard to the third case, the infection of the hip-joint might have been the same as that which caused the chorea; it might be a Calvé case with added infection, in which, as Calvé said, there was considerable destruction with but slight symptoms, because a damaged part was being affected, and organisms of low virulence could produce it. The association with rickets was Calvé's suggestion. He (Mr. Elmslie) did not think there was anything in it. He was himself accustomed to class as osteogenesis imperfecta a group of children in whom there were extensive deformities of the skeleton, due to very intensive malformation or lack of proper formation of bones, the children being stillborn or dying in the first year or two of life. He believed that in the condition under discussion the lesions were traumatic. The only case in which he would suggest operation was the doubtful one, and even there not an open operation. He did not consider it would be justifiable to do an open operation in any of these cases.

Case of Congenital Syphilis with Enlargement of the Liver and Spleen.

By J. Walter Carr, M.D.

A girl, aged 12 years. No definite family history of syphilis can be obtained, but patient is the third child, and her mother had two miscarriages between the births of the second and third children. The girl was born at full term, and there is no evidence that she had any symptoms suggestive of syphilis during infancy. She was in the Victoria Hospital for Children in March, 1911, for syphilitic disease of the soft palate, with pain and difficulty in swallowing. At present the uvula is destroyed and the remains of the soft palate are adherent to the posterior wall of the pharynx; there is a perforation on the left side of the palate, communicating with the left nostril. The liver is much enlarged, reaching nearly to the umbilicus; it is hard, smooth, and not tender. The spleen is also enlarged and hard, extending about 3 in. below the costal margin. There is no albumin in the urine. There is a slight thickening of the middle third of the right tibia. The eyes are healthy and there are no characteristic appearances in the teeth. The report on the blood, by Dr. Henderson Smith, of the Lister Institute, is as follows: Total reds, 3,750,000; total whites, 6,500; polymorphonuclear neutrophiles, 61·8 per cent.; polymorphonuclear eosinophiles, none; mononuclears—large 18·7 per cent., small 19·4 per cent. Two nucleated reds were seen in counting 350 white cells. Hæmoglobin, 100 per cent. Wassermann reaction strongly positive. Lately the child has got thinner and has complained of loss of appetite and diarrhœa, probably only as a result of a temporary gastro-enteritis.

DISCUSSION.

Dr. Walter Carr asked the opinion of members of the Section as to the probable nature of the enlargement of the liver and spleen in cases of this kind. They were not uncommon, but he had never had the opportunity of seeing one in the post-mortem room. The hardness of the organs was suggestive of lardaceous disease, but examination of the urine did not give the slightest evidence of lardaceous disease of the kidneys: if gummata were present the enlargement would probably be irregular. Most likely there was a diffuse gummatous infiltration, culminating in extensive fibrosis. In view of the fact that the Wassermann reaction was so strongly positive, he asked whether it was desirable to give salvarsan, or was it sufficient to administer mercury?

Dr. PORTER PARKINSON said that the liver and spleen were almost invariably smooth, which was like a diffuse infiltration. In many cases he believed there was albuminuria. He had at present under his care a child with many congenital syphilitic lesions, and with a spleen two or three times as large as in the child now shown, but there was much albumin in the urine, and he thought it might be albuminoid disease. It was comparatively rare to find roughness on the surface, anything like a gumma, so he supposed it must be amyloid disease or a diffuse infiltration of gummatous or fibrous material. In his experience the administration of mercury did not produce much shrinkage of the organs.

Dr. LANGMEAD said that he remembered a girl, aged 12 years, with enlargement of the spleen and liver, which was due to syphilis, the Wassermann reaction being positive. There was also slight jaundice. Salvarsan was given, and produced a toxæmia (cholæmia), from which she died in about twelve hours, with intense jaundice. He understood it was now generally recognized that a local reaction occurred, and if, owing to extensive fibrosis, there was but little liver which still functioned, such a reaction would be liable to put that small portion out of action by congestion and would cause death. Probably that was what happened in the case he cited. Both the liver and spleen showed extensive fibrosis.

Dr. F. PARKES WEBER said that in congenital syphilitic children with enlarged liver and spleen he believed that the pathological change in the affected viscera was generally a kind of fibrosis: he was not aware that amyloid disease had been found much in such cases. There should be extreme caution in these cases not only in regard to treatment with salvarsan, but also in regard to treatment with mercury.

Case of Hirschsprung's Disease.

By J. WALTER CARR, M.D.

A BOY, aged 9 years. He has probably suffered from gradually increasing constipation from birth. When aged 3 years he was in the West London Hospital for about ten days for severe constipation with visible peristalsis of the large intestine, and at that time it appears to have been considered that there was, perhaps, some obstruction due to chronic tuberculous peritonitis, but this now seems improbable.

Since then he has always been more or less constipated, requiring a great deal of purgative medicine. For six weeks before his admission to the Victoria Hospital for Children on January 10, his bowels had only been opened by injections. Some very hard scybalous masses were felt in the hypogastrium, and the possibility of a new growth had even been considered, but they have mostly been removed by enemata. The boy has been taking a mixture containing two teaspoonfuls of petroleum three times a day, and a pill of aloes, nux vomica and belladonna, and recently he has had several spontaneous actions of the bowels. He looks fairly healthy. The abdomen and the lower part of the chest are greatly enlarged, the increase in length between the ensiform cartilage and the umbilicus being particularly marked. The whole abdomen is very tympanitic, and there is very marked visible peristalsis, but it is very difficult to follow its direction. The liver, spleen and heart are displaced upwards, the heart's apex beat being in the third left space. The rectum appears to be normal.

Dr. Stanley Melville has made an X-ray examination of the abdomen after a bismuth meal. He reports that there is apparently no delay in the passage of the meal as far as the ascending colon, but from the hepatic flexure onwards the bismuth is seen in irregularly shaped masses in what appears to be an enormously dilated and probably thickened colon. At the end of seventy-two hours the chief part of the bismuth is seen in the rectum, but the outline of the dilated colon can still be made out. From time to time during the examination vigorous peristaltic movements were seen along the colon.

Opinions are invited as to the desirability of operative treatment in the case.

DISCUSSION.

Dr. THURSFIELD referred to a case of Hirschsprung's disease shown last year by Dr. Hector Mackenzie and Mr. Battle.[1] In the discussion which followed the question which was raised was not whether an operation should be performed, but what operation should be chosen. The general opinion seemed to be in favour of appendicostomy and washing out of the colon, at least as a preliminary measure. Sir William Osler at that meeting was strongly of opinion that medical measures were of no ultimate use, and that every case came at last to the surgeon. But he (the speaker) had seen certainly one case, possibly two, which recovered without surgical aid.

The PRESIDENT related the following experience. A child was passed over to his care by Dr. Hebb, who had tried medical measures for the condition

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[1] See *Proceedings*, 1912, v (Clin. Sect.), p. 146.

The child was aged 11 years, and the parents were averse to operation. At first he could only obtain consent for exploration, and he found a huge cylindrical mass of colon 6 to 7 in. in diameter, more or less loaded with fæces. In accordance with his promise not to do more, he closed the abdomen. But the child was detained, and permission ultimately obtained to deal with it radically. When he opened the abdomen a second time he found abdominal tubercle, as a result of which he made inquiry into the hospital milk. From the exploratory operation it was inferred that the difficulty would be with the pelvic portion of the distended colon. The distension began from about the middle of the ascending colon, and went down almost to the anus. He was, however, able to accomplish an anastomosis and take out all the distended colon. The child recovered from both the operation and from the tuberculosis, and so it was a successful case.

Mr. SIDNEY BOYD said that in X-ray examination of these cases a better idea was obtained by injecting bismuth *per rectum*. In one such case in which he had watched the bismuth pass up the bowel, the colon was shown to be quite abnormally arranged. In the present case he suspected that the place thought to be the hepatic flexure was not that at all. In the case he watched, the colon turned up from the left iliac fossa to the gall-bladder, and then passed down to the rectum on the right side of the abdomen. In regard to treatment, he had operated upon one case after two years' unsuccessful medical treatment, by excising the dilated portion of the bowel. He had some difficulty in getting at the lower portion, and he could not have made a complete removal except by the abdomino-perineal method, which he regarded as too severe. Therefore, he contented himself with performing anastomosis as low as possible. It was nearly a year ago, and the result had been perfectly successful, the bowels acting every day without medicine. With regard to appendicostomy, he thought it might be usefully employed as a means of clearing the colon previous to colectomy, if ordinary measures were not successful in doing so.

Arterio-sclerosis with absence of both Radial Pulses in a Girl, aged 15 Years.

By CHARLES W. CHAPMAN, M.D.

F. G., SEEN as an out-patient by Dr. Halls Dally on November 10, 1912, and admitted under my care on November 20. She complained of cough and orthopnœa on exertion or exposure to cold. There was rheumatism on the mother's side.

History: No special illness until four years ago, when she suffered from loss of power in the legs with loss of speech, the attack lasting fifteen minutes. There were several similar attacks during the following six months. Bronchitis two years ago. She is said to have had chalky lumps on her abdomen last summer. When first seen the area of cardiac dullness was increased in both directions, that on the left side being 9 cm. beyond the mid-sternal line; the apex beat was in the sixth space at the extreme margin of the dullness. The finger-tips were slightly congested. Catamenia not commenced. Teeth very bad.

On admission the cardiac dullness was as stated; the subclavians were visibly pulsating, and aortic pulsation could be felt by the finger in the aortic notch, the brachial and radial arteries were hard and devoid of pulsation, the iliac arteries were visibly pulsating, but only feeble pulsation could be felt at the left post-tibial and not at all on the right; the apex beat was in the sixth space and the action cantering. Urine: Specific gravity 1022, albumin a trace, sugar absent, no casts or red blood cells. Wassermann reaction negative.

Since admission the child has gained weight and has generally improved; the cardiac dullness is diminished and she can lie down in comfort. There is still a cloud of albumin. The skin on the abdomen is dusky, but no cyanosis or blanching is present.

DISCUSSION.

Dr. CHARLES W. CHAPMAN added that there was very great enlargement of the liver, and he compared the case with one he showed in 1908 at the parent society of the Section,[1] on which there was a post-mortem. Both were girls, aged 15 years; there was no history of acute illness, nor were there murmurs or thrills, and both had arterio-sclerosis. In the early case there was evidence of congenital syphilis, with occlusion of arteries. Albuminuria was common to both, but casts had not been found in the present case. In this case there was a slow process of occlusion of arteries. There was cardiac hypertrophy in both cases, and in the former case the post-mortem revealed granular kidneys. Yet in the present child there was no coldness of extremities, but there was poor development, owing probably to a deficient circulation extending over a long period. The skiagram showed the cardiac hypertrophy, while the electrocardiogram and polygraphic curves showed the absence of valvular or other gross lesions. Dr. Branson had published a paper entitled "Obliterative Arteritis,"[2] in which were many pathological observations bearing upon the subject of arterio-sclerosis in children.

Dr. HALLS DALLY regarded the case as interesting from several points of view, especially in comparison with the previous case two years ago. But in the former case there was a definite history of syphilis, whereas in the present case no causal factor could be ascertained for the condition; there was a negative Wassermann reaction, and no ascertainable history of nephritis. When the child first came to the out-patients' department she looked fairly well nourished, but the extremities were somewhat cold. He thought there might be another explanation—i.e., instead of there being a retrograde condition, possibly the arteries had never developed, and there was a lack of canalization of some of them. The feel of the radials and posterior tibials was much like that of a cord; they rolled under the finger. If one invoked an organized clot, occluding the vessels, there must be some cause for it, and here a collateral circulation must have been established. One could not distinguish any aberrant vessels.

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[1] *Rep. Soc. Study Dis. Child.*, 1908, viii, p. 264.

[2] *Trans. Path. Soc. Lond.*, 1905, lvi, pp. 212-23.

Dr. F. PARKES WEBER said that the child, who was too small for her age, belonged to the class in which there was evidence of organic (not merely functional) renal abnormality and retarded growth or imperfect development of the body.[1] The records of some such cases showed absence of any history or any evidence of syphilis; a positive Wassermann's reaction for syphilis or other evidence of congenital syphilis was apparently more often present in the children showing a greater or lesser degree of infantilism accompanied by polyuria (diabetes insipidus) without signs of organic renal disease.[2] The renal condition in the present case might be one of congenitally imperfect development with functional insufficiency, and with or without superadded acquired disease of the kidneys. The arterial condition might be partly due to associated insufficient development of the cardiovascular system, and might also be partly a result of the imperfect renal system.

Dr. CHAPMAN replied that he considered the thickness of the vessels was an argument against lack of development of them; and he reminded members that the former case was syphilitic.

Case of Interstitial Keratitis and Osteo-periostitis of the Tibiæ treated with Neo-salvarsan.

By SYDNEY STEPHENSON, C.M.

L. B., AGED 6 years.

Family history: Has three sisters, aged respectively 9, 8, and 2 years, all apparently healthy. The mother gives no history of infection, &c. Ten years ago she was affected with some nasal trouble, for which she was treated as an out-patient at the Hospital for Diseases of the Throat, Golden Square. One child, born prematurely at the seventh month, died when aged 7 weeks from some cause unknown to the mother. No miscarriages or stillbirths. The father, who is stated to have "knocked about a lot," is at present under treatment for stricture of the urethra.

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[1] See cases of Glover Lyon (*Lancet*, 1901, i, p. 102); H. Morley Fletcher (*Proc. Roy. Soc. Med.*, 1911, iv (Child. Sect.), p. 95); Leonard G. Parsons (*Brit. Med. Journ.*, 1911, ii, p. 481); R. Miller (*Proc. Roy. Soc. Med.*, 1912, v (Child. Sect.), p. 38); A. E. Naish (*Brit. Journ. Child. Dis.*, Lond., 1912, ix, p. 337); and F. Langmead (in the discussion on Miller's case, loc. cit.).

[2] See F. P. Weber, "Diabetes Insipidus in a Boy, with Positive Wassermann's Reaction," *Brit. Journ. Child. Dis.*, Lond., 1912, ix, p. 211.

History of illness: Nine or ten months ago the patient is said to have suffered from "rheumatic pains" in backs of hands, shoulders, both knees, and both legs. Although medically treated he did not improve much. After a visit to Australia, he returned to England two months ago. He first attended the Queen's Hospital for Children some three weeks ago. Just before last Christmas the father noticed a film over the child's right eye, and then found that he could only tell day from night with the affected eye. The patient was referred to the Eye Department on January 9 last, and was admitted.

State upon admission: The lad shows no signs of syphilis as regards his face, cranium, or teeth. The inguinal and cervical glands are slightly enlarged and "shotty" to the touch. The skin is in a state of xeroderma. Nodes are present on the middle third of each tibia, being more marked on the right side. The overlying skin is not discoloured, and the swellings themselves are not tender. There appears to be the remains of a similar node about the middle of the right ulna. Chest and heart normal. The Wassermann reaction was strongly positive.

Right eye: Trifling ciliary redness and photophobia. The pupillary area of the cornea shows a disk-like interstitial opacity, while the peripheral parts of the cornea are almost clear. The anterior epithelium covering the opacity looks as if it had been breathed upon. The pupil, although the eye is being treated with atropine (2 gr. to the ounce, three times a day), is no larger than 4 mm. It is probably fastened down by posterior synechiæ. Tension normal. Vision, perception of light.

January 20, 1913: Neo-salvarsan, 0·6 gr., injected intravenously.

Mr. Sydney Stephenson added that in his experience, as probably also in that of others, the association of interstitial keratitis and osteo-periostitis was not altogether uncommon. The interstitial keratitis here was atypical, and he wished to draw attention to the fact that the condition of the eye, though not of the shins, had improved markedly after one injection of neo-salvarsan four days ago. That was contrary to his general experience of both salvarsan and neo-salvarsan.

Fibrosis of Right Lung, with Heart completely on Right Side, in a Female Child, aged 17 Months.

By SHEFFIELD NEAVE.

HISTORY by the mother is as follows: L. W., aged 17 months. Measles ten weeks ago, and since then bad cough and vomit. Bowels irregular, some diarrhœa. Breast-fed till admission. Four other children in good health; two dead from broncho-pneumonia and whooping-cough. Maternal grandfather died of phthisis. Wassermann negative; von Pirquet negative; sputum negative, smear-stained for tubercle. Since admission to hospital has only a slight cough with much sputum, which is swallowed, but appears not to be offensive. Temperature normal. Right lung has the signs of an advanced fibroid state, probably with bronchiectasis. Heart completely on right side; this confirmed by X-ray photograph. Spleen palpable. Fingers not clubbed.

This case appears to be interesting in the following particulars: (1) the advanced fibroid state in so young a child; (2) the question as to ætiology.

Out of the most recent books I have, two say that tubercle of the lung is a common cause in children of fibroid lung, one that it is practically never so caused, and one that it is very seldom so caused. On p. 427 in his book on diseases of children, Cautley begins his article on this subject by saying: "Chronic inflammation is usually tubercular. It occasionally follows pneumonia," &c. On p. 184 of his lectures on diseases of children, Hutchison says: "The fibroid form of tuberculosis is not uncommon even in young children." Still says, p. 369 of his book: "Tuberculosis is so rare a cause of fibroid lung in children that it would almost be enough to disprove the presence of tubercle." Miller's book, p. 333, says: "Chronic pulmonary tuberculosis plays a small part in the matter, and although it may be difficult to exclude it, yet most cases of the disease are the result of a non-tubercular pneumonia."

Other than tubercle, the ordinary causes we have to select from are: broncho-pneumonia, lobar pneumonia, pleurisy, syphilis, congenital atelectasis, foreign body, or some other form of pressure. Choosing from these—for my part in the absence of adequate history of previous ailments—I think that the greatest probability lies with broncho-pneumonia in this case. There is, I think, a possibility that congenital dextrocardia may have existed in the first instance, but this must be unlikely, more especially as no other organ seems to be transposed.

I hope by showing this case to obtain from members of this Section opinions as to the ætiology of it, more especially in reference to tuberculosis, and further as to whether this is not a very early age for so complete a case, if they will kindly give the benefit of their experience.

DISCUSSION.

Dr. WHIPHAM suggested that, as there was no history of pneumonia, it was not a case of acquired fibrosis of lung, but one of persistent atelectasis of the lung, with consequent displacement of the heart. In his experience, fibrosis of the lung in childhood was nearly always the result of an antecedent pneumonia.

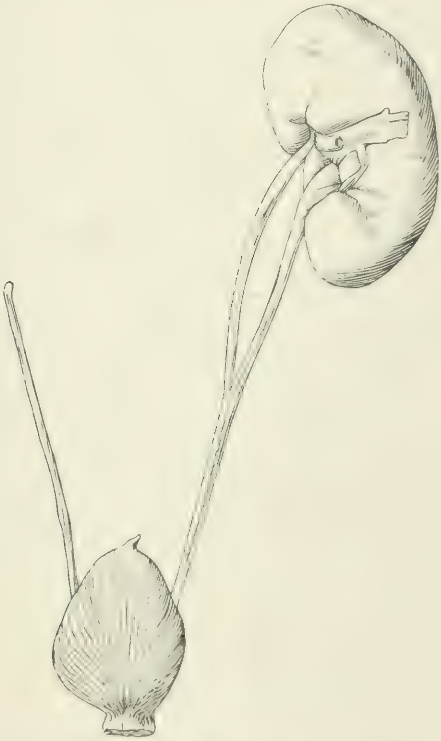
Dr. THURSFIELD agreed with Dr. Whipham that this was probably an acute condition. He could not on any other hypothesis reconcile the position of the heart, supposing it to have been dragged over, with the lack of deformity of the chest.

Congenital Renal and Ureteral Anomaly.

By J. D. ROLLESTON, M.D.

Boy, aged 12 years. Death from generalized paralysis, following very severe faucial and nasal diphtheria, on forty-seventh day of disease. Violent attacks of abdominal pain during last twenty-four hours of life. Albumin present in the urine from the third to thirty-eighth day. Amount of urine passed during first eighteen days of disease ranged from 21 oz. to 37 oz. in the twenty-four hours. Specimens show: (1) Single or asymmetrical left kidney, 4¾ in. long, 2 in. broad, 1½ in. thick, 6 oz. in weight; (2) duplication of ureter, the upper branch, 4 in. long, supplying the upper third, and the lower branch, 3½ in. long, supplying the lower two-thirds of the kidney; (3) union of the two branches of the left ureter 3 in. from the bladder; (4) the right and left ureteral orifices in the bladder; and (5) a right ureter which is patent from the bladder to its blind upper extremity. The right kidney, renal artery and vein, and right suprarenal were absent. No abnormalities of genital, circulatory, or other systems.

It is a well-known fact that there are no symptoms peculiar to single kidney, which in my case, as in most of those on record, was a necropsy surprise. The abundant and persistent albuminuria was such as is usually found in malignant diphtheria. The amount of urine was measured during the first three weeks of the disease merely to serve as a guide to prognosis, a marked diminution in the urine during the acute stage of diphtheria being of bad omen. There was no oliguria in the present case, the single kidney being perfectly able to do the work of two. Whether the violent attacks of abdominal pain were associated with the abnormal condition of the kidney or ureters I am unable to say. There was no evidence, however, of renal calculus, to which single kidney seems to be unusually predisposed (Morris [16]).



Congenital renal and ureteral anomaly.

It is interesting to note that the blood-pressure which, during the first three weeks of the disease, had fallen from 100 mm. on the third day of the disease to 80 mm. on the ninth, rose with the onset of paralysis to 110 mm. on the thirty-sixth, and on the day before death to 120 and 130 mm. This rise of blood-pressure was probably not connected with the renal condition, but was such as often occurs in late diphtheritic paralysis, and was possibly due, as I have suggested elsewhere [20], to an irritative condition of the vasomotor centres in the medulla, in which the other nerves undergo a varying degree of paralysis.

The case is one of unusual interest from the anatomical, medical, and surgical standpoints. As far as I have been able to ascertain from a study of the literature, no other cases have been recorded of asymmetrical kidney, with partial duplication of its ureter co-existing with a patent ureter on the side on which the kidney is absent.

The condition of single or asymmetrical kidney is by no means common, being found, according to Morris [16], in only one out of 2,400 autopsies: 213 were collected from literature by Ballowitz [2] in 1895, 286 by Anders [1] in 1910, and 300 by Dorland [6] in 1911. Probably these figures are too large, some of the cases, especially those recorded by the earlier writers, being examples of fused and not of single kidney. The congenital absence of the kidney is due to failure of the Wolffian duct to throw off the corresponding renal bud after the duct has reached the cloaca.

Duplication of the ureter, which is usually incomplete, as in my case, is not so very uncommon, being found in 1 to 2 per cent. of all corpses (Lessing [13]). The association of solitary kidney with complete or partial duplication of the ureter, though uncommon, is not unique, cases having been recorded by Morgagni [15], Blaise [3], Rufz [21], Laroche [12], and Gérard [7]. The last authority regards the association of double ureter with solitary kidney as a pure coincidence.

The presence of a ureter on the side on which the kidney is absent is very exceptional. Out of 286 cases of single kidney collected by Anders [1] there were only twenty-four in which a more or less rudimentary ureter was present on the side on which the kidney was absent. In the majority of these the ureter was impervious, and I have been able to find only seven cases in which, as in my own case, the ureter was patent throughout its entire length—Busk [4], Haberer [8], Hallopeau [10], Horand [9], Nelson [17], Paulicki [18], Winter [22]. The medical interest of single kidney lies in the frequency with which the organ is liable to disease. According to Anders [1], seventy-nine out of a total of 170 cases, or 46·5 per cent. in which renal changes were recorded showed morbid changes, and 42·3 per cent. some form of chronic nephritis. The outlook in these cases is less hopeful than when both kidneys are present. The surgical importance of single kidney is impossible to over-estimate. The present case shows how important it is, before operating on the kidney, to ascertain not only whether there are two ureteral orifices, but also whether urine escapes from both; in other words, not only is cystoscopy necessary, but also catheterization of the ureters. The partial duplication of the ureter adds a further difficulty. In catheterization of the kidney, collection of urine from the upper or lower branch respectively would give an erroneous idea as to the functional value of the organ. At present no instrument has been invented to detect this anomaly (Jeanney). In Mauclaire and Séjournet's case [14] the upper two-thirds was served by one ureter and the lower one-third by the other, while in Jeanney's [11] case, as in my own, the upper one-third of the kidney was supplied by the upper branch and the lower two-thirds by the lower branch of the double ureter.

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Specimen of Non-development of Cerebrum.[1]

By G. A. Sutherland, M.D., and H. W. Perkins, F.R.C.S.

F. G., female, aged 9 weeks. The head resembled that of a normally developed infant of this age, the circumference being 13½ in. The anterior fontanelle was widely open, measuring 2 in. in the sagittal direction and 1 in. in the coronal. The sutures were not united. On opening the skull and dura mater a quantity of blood-stained fluid escaped, amounting to 9 oz. On centrifuging this the blood cells were thrown down, leaving a clear supernatant fluid of a straw colour.

The dura mater appeared to be normal and the falx cerebri was fully developed and present throughout its whole length. The hæmorrhagic fluid present was apparently in the cavity of the arachnoid. This cavity was partially divided in the occipital region by a few fine, cobweb-like adhesions, present chiefly on the left side. The brain, lying on the base of the skull, was very small and covered with thickened, œdematous-looking pia arachnoid, which concealed its outline. On the removal of this membrane it was seen that the brain was extremely ill developed as regards the cerebral lobes. The brain weighed 80 grm. Each cerebral hemisphere measured 9 cm. in length, 6 cm. in width, and 3·7 cm. in depth, taking the maximum measurements. The two halves of the dwarfed cerebrum on each side of the falx cerebri were roughly symmetrical, except that posteriorly into the occipital lobe on the left side a large hæmorrhage had taken place, the clot being recent and unorganized.

The middle third of the cerebral cortex on each side presented numerous convolutions, which although small and narrow were separated by well-defined sulci. The anterior parts of the cortex were extremely thinned and so intimately associated with the pia arachnoid that it was difficult to recognize any well-marked convolutions or sulci. The appearance here was that of numerous small cavities, like a honeycomb, bounded externally by the thinned cortex and pia arachnoid. Definite convolutions and sulci were present at the posterior pole on the right side, while on the left side the posterior pole was the seat of the extensive hæmorrhage referred to above. This hæmorrhage formed a roughly spherical clot about 25 mm. in diameter, limited externally by the thinned-out cortex and pia mater, and suggesting from its appearance the possibility of its having arisen from bleeding into a thin-walled cyst. The basal surface of the cerebral lobes presented a crinkled appearance, well-defined convolutions and sulci being absent. The cerebellum, medulla, pons, and peduncles presented a normal appearance. All the cranial nerves could be identified, and the blood supply and blood-vessels were normal so far as could be determined.

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[1] From a patient shown at the November meeting, see *Proceedings*, p. 37.

A transverse section through the right cerebral lobe at the level of the corpus callosum showed that the lateral ventricle was slightly distended, and that the ependyma and choroid plexus were normal. The brain substance beneath the ependyma had a spongy, reticulated appearance, and in the floor of the ventricle, beneath the lining membrane, was a small cavity about the size of a pea, filled with blood-clot. The left ventricle was but slightly enlarged. The foramen of Monro was patent and normal. The third and fourth ventricles were not enlarged, and the patency of the "iter" was established. The cortex of the mesial aspect of the cerebrum, when cut into, showed numerous small spaces about the size of a hemp seed.

Dr. SUTHERLAND reminded members that he showed the child alive at the previous meeting, and the specimen showed very much what was expected from the symptoms present during life.

Sub-aortic Stenosis.

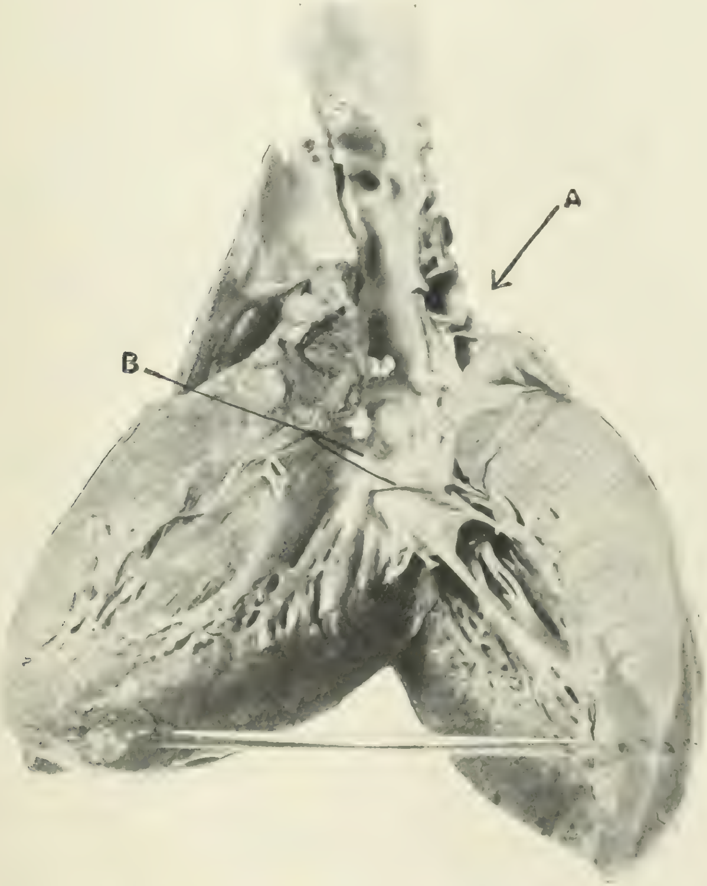
By HUGH THURSFIELD, M.D., and H. W. SCOTT.

THE specimen of sub-aortic stenosis described was taken from a boy, aged 14 years, who was brought dead to St. Bartholomew's Hospital in May, 1912. He was seated at work when he suddenly fell from his seat; when picked up he was able to swallow some water, but though brought directly to the hospital—a short distance—he was dead on arrival.

On inquiry it was found that he had been an in-patient seven years previously, suffering from a mild attack of faucial diphtheria. Reference to the notes taken at that time showed that he then had a loud systolic murmur heard over the whole præcordia and the back: the point of maximum intensity was the aortic area in the second right intercostal space. No mention is made of a thrill nor of the character of the impulse. The diagnosis suggested was "patency of the septum ventriculorum." Apart from this illness he had enjoyed fair health, but was known to have a "weak heart," and from time to time had attended the Casualty Department for a cough. He was the fourth of five living children, all of whom enjoyed good health; besides these children his mother had four miscarriages.

The body was that of a tall, muscular, well-developed boy; there was no clubbing of the fingers, and no external malformation or injury. On opening the thorax the heart was seen to occupy a larger area than normal, the greater part of this increase being due to hypertrophy of the left ventricle. The right side of the heart was moderately dilated; the auricle showed some thickening of its walls, and the ventricle was, in comparison with the left, small and thin-walled, and empty of blood; the tricuspid and pulmonary valves were normal. The left auricle, on the other hand, was greatly hypertrophied, the thickness of the wall being at least ⅛ in.; the dilatation was slight. The mitral orifice and cusps, except for the band to be mentioned, were normal. The ventricle was enormously hypertrophied and considerably dilated. The aortic orifice was narrowed by a fibrous ring situated on the septum ventriculorum immediately below the undefended space and extending over the posterior aspect of the mitral aortic cusp. The diameter of this ring at its widest was less than ¼ in.; its edges were smooth and free from vegetations. The aortic semilunar cusps were thickened and the two coronary cusps fused into one, so that the orifice was much narrowed. Immediately above the non-coronary cusp was a thin line of fibrous thickening in the wall of the sinus of Valsalva; and at the level of the ductus arteriosus, which was closed, was a transverse line of thickening, which appeared to have produced a slight narrowing of the lumen of the aorta. Otherwise the intima of the aorta was normal, and no abnormality was noticed in any other vessel. The heart weighed 14½ oz.; the pericardial membrane was normal.

Examples of sub-aortic stenosis have been seldom recorded. Dr. Norman Moore, in 1882, described a specimen, which is still in the Museum of St. Bartholomew's Hospital. It was obtained from a lad, aged 18 years, who was admitted to the wards in May, 1882, with dyspnœa and swelling of the feet and ankles, under the care of Sir William Church. The clinical notes state that he was markedly anæmic and generally œdematous. His pulse is described as "feeble and thready," 120 a minute. The heart's impulse was felt in the nipple line 1 in. below the nipple; the cardiac dullness extended to the middle of the sternum. A loud double murmur was audible over the whole cardiac region, in the axilla, and all over the back; its maximum intensity was at the apex and down the sternum. No mention is made of a thrill, nor of the character of the impulse. The patient was four weeks in hospital, with an evening temperature usually of 101° F., albuminuria, hæmaturia, and casts. He was said to have been always weak and subject to fainting fits.



Sub-aortic stenosis. **A**, aortic semilunar valve; **B**, sub-aortic fibrous ring.

At the post-mortem examination the pericardium occupied almost the whole of the exposed area, "almost the size of a man's head." The heart was freely movable in the fluid which distended the sac; this fluid was slightly turbid, but there were no flakes of lymph. "At the base of the heart was a hard mass, about 2½ in. by 2 in., exactly in the situation of the lower part of the thymus in a child."

There was some, but no great, hypertrophy of the heart. Just below the aortic valve was a thickened ring of fibrous tissue attached to the septum on one side, and to "the outer wall" (*sic*) of the heart above the mitral valve on the other. Above this ring the aortic cusps were competent and in no way abnormal. The aorta was free from atheroma. At the origin of the innominate artery there was an opening, almost circular, 1 in. in diameter, the mouth of an aneurysmal cavity partly filled with organized clot—the mass referred to above. The liver and spleen were very large, and the kidneys weighed 28 oz., and were pale, swollen, with capsules which split easily.

Professor Keith, in the Hunterian Lectures of 1909, had seen only four hearts which showed this condition; one was the specimen just mentioned, one in the Museum of St. George's Hospital, one in Guy's, and one which was lost in the fire at Toronto University.

Another example was recorded by Fletcher and Beattie from the Royal Infirmary, Edinburgh, in 1904; it was obtained from a girl, aged 17 years, who had enjoyed good health to the age of 15 years. This patient was dyspnœic but not œdematous; the pulse-rate was 120 a minute, the beat very feeble, and almost obliterated on raising the arm. The apex beat was feeble, somewhat far out and down. On auscultation a loud rough and prolonged diastolic murmur was heard all over the heart and at the root of the neck; this was most intense at the inner end of the third left intercostal space and down the sternum; at the apex there was a short systolic murmur. The right border of the heart was not beyond the right sternal border. At the autopsy there was a mass of dense fibrous tissue between the pericardium and the epicardium. The pulmonary artery was narrow at its orifice and the valve segments smaller than normal, but they showed no degenerative changes. The aortic valve segments were much thickened and the left anterior cusp was ulcerated and perforated, the perforation surrounded by recent vegetations. Immediately above the posterior cusp was a mass of vegetations on the wall of the aorta, and beneath these a small aneurysm; 5 mm. below the aortic cusps was a thickened calcareous ring 6 mm. in diameter. The aorta was healthy. No mention is made of clubbing of the fingers or of a præcordial thrill.

Shennan, in 1897, described a specimen in a male, aged 19 years. This boy was poorly developed, with marked clubbing of the fingers, but there was no œdema. There was marked episternal pulsation, a marked systolic thrill to the right of the middle line in the first and second intercostal spaces, and a blowing systolic murmur so loud in the aortic area as to be audible 5 in. from the chest wall. The heart showed hypertrophy of the left ventricle, and the same sub-aortic fibrous ring as has been already described. There were recent vegetations on the ring, extending upwards to the aortic cusps.

This rare malformation has been explained by Professor Keith as follows: "The part of the fœtal heart from which the infundibulum is derived—the bulbis cordis—normally disappears completely from the left side of the heart, but a trace of it may persist, giving rise to the condition known as sub-aortic stenosis: . . . thus while the abnormality on the right side of the heart depends on an arrest of growth, that on the left depends on an arrest of atrophy."

The specimen which we have recorded differs from the others in the fact that it presents further evidences of malformation in the fibrous thickenings present in the wall of the aorta. That situated at the level of the ductus arteriosus is especially important, since it represents the constriction which is normally present in the fœtus at this spot, the persistence of which leads to the abnormality known as coarctation of the aorta.

It is obvious from the descriptions given above that the condition is not incompatible with good general health, at any rate, when it is present only in a slight degree, and so long as there is no infection of the abnormal structure or of the valve; and we would suggest that it affords a probable explanation of no inconsiderable number of those cases of congenital heart disease in which, without cyanosis or clubbing of the fingers and with good general development, the children have a loud rough systolic murmur heard at its maximum down the sternum. Such cases are not very uncommon among London school children, and are detected in the course of routine examination, not by reason of any failure of health.

The physical signs which we should expect to find in such a case are: (1) Evidence of a hypertrophy of the left ventricle; (2) a loud systolic murmur with its maximum intensity in the second and third intercostal spaces on the right side, and conducted down the sternum; and (3) a systolic thrill, most marked in that area. It is, however, remarkable that the thrill is only recorded in one of the four cases noted.

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A Case of (?) Sub-aortic Stenosis.

By A. E. Gow, M.D.

The patient, T. S., a well-nourished boy, aged 6 years, has been under observation in St. Bartholomew's Hospital under Dr. Herringham, and at the Queen's Hospital for Children for the past fifteen months. There are six other children in the family, all healthy. At the age of 2 years the patient contracted scarlet fever, and his parents were told at that time that he had heart disease. He has had no other serious illness. There is no rheumatic history. His mother states that he is irritable and bad-tempered. If annoyed he gets very excited and jumps about, screaming, frequently becoming blue in the face. He is easily exhausted. There have been several attacks of diarrhœa during the past year, two of which have been associated with the passage of blood by the rectum. His weight is 43 lb.—a gain of 6 lb. during the year. There is no clubbing of the fingers, and no polycythaemia. The pulse-rate is about 100, regular, and small in volume.

The physical signs, which have undergone no appreciable change since he was first seen by me, are as follows: The cardiac impulse is diffuse and visible on the left side in the fourth and fifth spaces, the point of maximum impulse being in the fifth space, 2½ in. from the middle line. A slight thrill, systolic in time, is present near the sternum in the upper two spaces on the right side, and feebly above both clavicles. The area of cardiac dullness extends ¾ in. to right of sternum in the third and fourth spaces; ½ in. to left of sternum in second space; 3¼ in. from the middle line in fifth left space. A harsh systolic murmur is to be heard all over the front of the chest, the point of maximum intensity being just below and external to the right sterno-clavicular joint. It is audible over the back of the chest, especially on the right side, and can be traced into the carotids, but not to the brachial or femoral arteries. The second sound at both bases is natural in quality, but is intensified.

A Note on Mongolism.

By F. G. Crookshank, M.D.

Though Mongolism is undoubtedly a state of infantilism that tends to undergo modification at puberty, its significance is apparently phylogenetic rather than ontogenetic. Whilst, however, those cases in which mental defect is least accentuated tend, morphologically, to resemble children of undoubtedly Mongolian descent, there are, in many Mongoloid imbeciles, certain characteristics that are not, at first sight, easily recognized in true Mongols. Such characteristics appear to be simian, and are, moreover, amongst the existing anthropoid apes, either most marked in or peculiar to the orang. It has long been recognized that the orangs are the Mongols amongst apes; and both the anthropogenetic system of Haeckel and the more recent scheme of Klaatsch are sufficiently familiar to all. But of course, neither Haeckel nor Klaatsch suppose that any extant variety of mankind is directly descended from any extant variety of orang. Amongst the many points to which I would draw attention are, the shape of the skull, the primitive state of the air cells, the slight brow ridges, and the facial bones; the eyes, the ears, the lips, tongue, hair, skin and mucous membranes; the larynx, heart, intestines, and external genitals; the hands, feet, and vertebral column. The vocal peculiarities, the habitual adoption of the Buddha position, the auditory rather than visual mind and brain, the relative proportions of cerebrum, cerebellum, pons, and medulla, and the functional values of the bulbar nuclei, are all interesting and important.

A longer account of some of the homologies is given in a paper published in the *Universal Medical Record* for January, 1913; but since writing that note I have gathered much additional information which strengthens the position therein assumed, although one or two of the statements should, perhaps, be slightly modified. The temporofrontal junction, for instance, does apparently occur sometimes in orangs and in Mongols, although Hartmann insists that it is less frequent in them than in Aryans, on the one hand, and gorillas and chimpanzees on the other.

I wish also particularly to draw attention to one or two points of historical interest. Dr. Shuttleworth, it is well known, has long since suggested that Mongoloid imbeciles are really "unfinished children." Now Dr. R. Langdon Down, to whom, as well as to Dr. Shuttleworth, I am much indebted, some years ago, in a paper to which I listened with great advantage, used these words: "It would appear, however, that the characters which at first sight strikingly suggest Mongolian features and build are accidental and superficial, being constantly associated, as they are, with other features which are in no way characteristic of that race; and if this is a case of reversion, it must be a reversion to a type even further back than the Mongol stock.

from which some ethnologists believe all the various races of men have sprung."[1]

Mdlle. Lutrovnik, in a most interesting thesis (Paris, 1908), after rejecting the notion that Mongolism is a true reversion to, or a reproduction of, the Mongolian racial type, hints at the possibility that it is a manifestation of reversion to simian type. She does not, however, formulate any definite proposition, and leaves the question open for investigation. Yet it is clear from internal evidence that her hesitancy in the matter is due to the fact that she was, at the moment of writing her thesis, familiar with the morphology of the gorilla and the chimpanzee, and not with that of the orang.

Dr. Douglas Hunter, of the Clifton Asylum, Yorkshire, some ten years or so ago commenced to collect material for a study of Mongolism, and, though at first inclined not to lay stress on either the Mongolian or the simian homologies of Mongoloids, later wrote thus: "In the light of some further study of the subject, however, the writer is still inclined to think that if the Mongolian idiot suggests a simian ancestor at all, he suggests one approximating more to the type of the orang than to any other of the higher apes." Dr. Hunter, unfortunately, did not prosecute his researches, and never published his notes. I first became aware of his interest in the subject after the publication of my own notes, and he has now most generously sent me, not only his MS., but some valuable photographs. He has drawn my attention to some points in the morphology of the teeth that had escaped me, and I find that, while our observations do not appear in any way to conflict, they are, in respect of many details, quite curiously complementary. I feel, therefore, justified in saying that the views expressed by Dr. Shuttleworth, Dr. R. Langdon Down, Mdlle. Lutrovnik, and Dr. Hunter, are all really perfectly consistent with those that I have endeavoured to express. It is only necessary to insist further that, if the anthropological notions with which the purely clinical and pathological observations are to be correlated become accepted, true homology should obtain, not between the Mongoloid and the orang, but between the Mongoloid and a propithecanthropoid ancestor (probably more erect and with smaller jaws than the orang), that should occupy a station in the line of descent somewhere between the ancestral gibbons and the orangs of to-day.

[1] *Journ. Ment. Sci.*, 1906, lii, pp. 188, 189.

Section for the Study of Disease in Children.

February 28, 1913.

Mr. A. H. TUBBY, President of the Section, in the Chair.

A Slight Congenital Deformity of the Hands in a Child.

By F. PARKES WEBER, M.D.

THE deformity consists in partial flexion of the fingers at the metacarpo-phalangeal joints. The child can use his hand fairly well to grasp with, but cannot completely extend any of the fingers except the left index-finger. Both the little fingers can, however, be *passively* completely extended. In some spontaneous positions of the hands slight ulnar deflexion of the fingers is likewise noticeable. The patient, W. H., is a boy, aged 17 months, who was born at full term, easily, without instrumental help. He is the only child of healthy parents, who are both young. The mother had had one miscarriage before the child was born. There is no history of any similar deformity in the family, nor is there any special family tendency to arthritic affections. The child has a little convergent squint and possibly slight hydrocephalus. He likewise presents some rachitic "beading" of the ribs, and is a little bodily and mentally backward for his age. He cannot yet walk.

The position of the hands at first reminds one somewhat of the carpopedal contractions in tetany, but the child has no facial irritability and nothing else which would in the slightest degree suggest tetany. The feet and toes are quite normal in shape and position. Since the child was 3 months old the mother has practised a kind of massage with passive movements for his hands, and thinks that he can use his fingers better as a result of her treatment. Skiagrams of the hands show nothing special. There seems to be no disease in the joints. Dr. Weber has not heard of any exactly similar case. There is a history of "maternal impression." The mother, whilst she was pregnant, frequently had visits from a girl, one of whose hands was deformed, probably as a result of an abscess or injury. The position of the fingers in that girl's deformed hand somewhat resembled that of the patient's fingers.

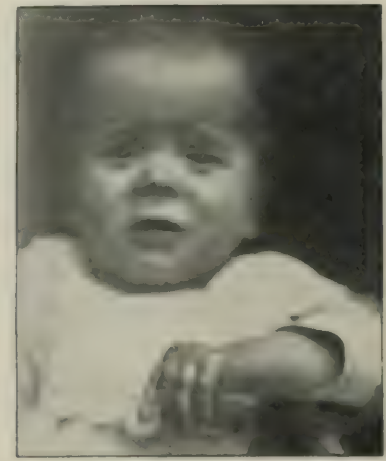

FIG. 1.

To show attitude of the left hand.

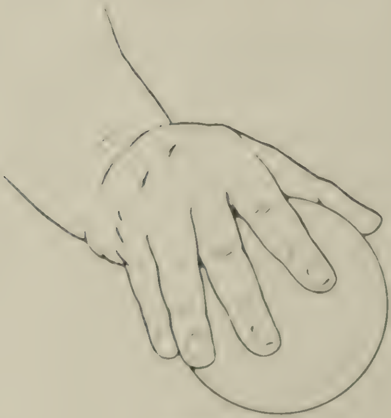

FIG. 2.

Drawing of the child's right hand, showing the stiff attitude resulting from inability to extend the fingers at the metacarpo-phalangeal joints.

DISCUSSION.

Mr. LAMING EVANS said he considered the case allied to conditions of congenital dislocations of the knee. His advice was that the parts which were contracted should be, under an anaesthetic, stretched and kept so. He thought that the principal lesions were situated in the ligaments which were shortened.

The PRESIDENT (Mr. A. H. Tubby) said the case seemed parallel to one which he had figured in his book on "Deformities," where there was congenital contraction of all the fingers and the thumbs of both hands. In that case he was able to verify the nature of the contraction. It was due to congenital shortening of the palmar fascia and its digital prolongations. If one examined the present case, one could still completely extend and flex the wrist without in any way interfering with the contraction of the fingers. Therefore, although he thought that Mr. Laming Evans's explanation was very reasonable, he was still disposed to regard it as a congenital contraction of the palmar fascia. The case was an exception of the usual rule, that when one met with a congenital contraction affecting the fingers, it was almost invariably the fifth fingers. Another striking point was that contraction in these digits was often associated with contraction of the little toe. The treatment which was being pursued in this case he regarded as the right one—namely, massage, passive movements, and extension, and, as Mr. Evans suggested, putting on a malleable iron splint. There would probably be no need for operation.

Mitral Dwarfism.

By F. PARKES WEBER, M.D.

THE patient, B. L., is a girl, aged 15 years, but her size is that of a child of 10 years. She is 50 in. (127 cm.) in height and weighs 54 lb. (24½ kilo). Ordinary examination and examination by Röntgen rays show that the heart is considerably enlarged to the left; the apex beat is in the sixth left intercostal space in the anterior axillary line; a presystolic and diastolic thrill can be felt, and a loud double mitral murmur can be heard, at the apex, showing that there is mitral obstruction accompanied by mitral incompetence, the mitral incompetence doubtless accounting for the hypertrophy of the left ventricle. There is a little crepitation at the base of the left lung behind. Röntgen-ray examination of the lungs with the fluorescent screen shows nothing special. Nothing abnormal can be felt by abdominal palpation. The teeth are fairly well formed. The nose is somewhat "saddle-shaped," but there is a history of past injury by a fall. The thyroid gland appears to be rather small.

There is no pubic or axillary hair. Menstruation has not yet commenced. The urine is free from albumin and sugar. The pulse (after examination) is 100 to the minute, of moderate pressure. Blood examination: Hæmoglobin, 80 per cent.; red cells, 4,400,000, and white cells, 6,600 to the cubic millimetre of blood. The blood serum gives a negative Wassermann's reaction for syphilis. The cuti-reaction for tuberculosis (after von Pirquet) is negative. Mentally the child seems normal and is employed at some light work by which she is able to earn a little money.

The history is that the child was born naturally, without instrumental aid, at full term. She learned to walk at the age of 13 months, and seemed like other children till the age of 5 years. There was no rickets. At 5 years of age she suffered from measles and congestion of the lung, and after that time was backward in her growth. Three years ago she had an attack of hæmoptysis, and her heart was said to be diseased. Three months ago she again occasionally spat some blood. She is said never to have had rheumatism. There is no other history of dwarfism or infantilism in the family, notably in regard to the patient's sisters. The mother, who is a "rather small" woman, aged 45 years, has had nine children (no miscarriages or abortions), of which the patient is the eighth. One (the fifth) of the nine children was born dead, and four died early. The eldest child (a married woman, aged 25 years), the sixth child (a girl, aged 19 years), and the ninth child (a girl, aged 9 years), are all living and healthy. The father, aged 44 years, is said to be healthy. Neither the father nor the mother have ever suffered from rheumatism or any severe infectious disease.

In the present case, as in most cases of mitral dwarfism, there is no history of past rheumatism. In some cases the mitral stenosis has been supposed to be of congenital origin, and to have been associated with hypoplasia of the large arteries, the imperfect and limited blood supply leading to defective growth of the whole body—a kind of anangioplastic dwarfism.[1] According to that theory the mitral disease is primary and leads to diminished flow of blood through the aorta and large arteries, which consequently remain small, the dwarfism of the whole body being regarded as a conservative adaptation to the defective cardiac and arterial conditions. A. Gilbert[2] thought that in certain cases at least (for instance, in the rare cases of familial mitral stenosis[1]) the mitral stenosis and the dwarfism were both to be regarded as manifestations of an inborn tendency to faulty development. Marcel Labbé is inclined to attribute the mitral stenosis and the general dystrophy in such cases to a tuberculous or syphilitic taint.[2]

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[1] Infantilism of Lorain's type was, I think, regarded by E. Brissaud as an "infantilisme anangioplasique" resulting from the action of a chronic congenital or acquired disease during the period of bodily growth and development.

[2] A. Gilbert, *Gaz. méd. de Par.*, 1884, lv, p. 198. See also A. Gilbert and F. Rathery, "Le Nanisme mitral," *Presse méd.*, Par., 1900, viii, pp. 225, 231.

In the present case there is absolutely no evidence of the presence of syphilis or tuberculosis, and I have hardly a doubt that the cardiac disease is of rheumatic origin, although there is no history of the child ever having had rheumatism. But rheumatism in young children is not rarely overlooked or mistaken. The dwarfism in this case I regard, indeed, as a sign, not of the congenital origin of the mitral disease, nor of the presence of a tuberculous or inherited syphilitic taint, but as a sign that the mitral disease commenced at a relatively early period of extra-uterine life, long before the period of maximum normal development. Of the mitral obstruction and the mitral incompetence I consider the former to be the more important factor in the ætiology of the dwarfism. This dwarfism, I think, may be best explained as the expression of a conservative adaptation or conservative hypoplasia of the whole body—Nature's attempt to limit the growth of the patient in accordance with the limited arterial blood supply. Such a *conservative hypoplasia* is to be contrasted with the better-known conservative vital process, known as *compensatory hyperplasia*, which is illustrated in the present case by the *hypertrophic enlargement* of the left ventricle to make up for the regurgitation of blood through the leaky mitral valve.

In conclusion, it should be mentioned that "mitral dwarfism" (the "nanisme mitral" of French authors) is only one variety (probably the best known) of a group of "cardiac dwarfisms." The dwarfism in all these cardiac cases is a partial infantilism. Therefore the terms "mitral infantilism" and "cardiac infantilism" may be employed if preferred.

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[1] Cochez, "Le Rétrécissement mitral pur, congénital, familial, et héréditaire," *Bull. méd.*, Par., 1898, xii, p. 493.

[2] Marcel Labbé, "Rétrécissement mitral pur et nanisme," *Presse méd.*, Par., 1908, xvi, p. 497. See also Labbé, Rosenthal, and Marcorelles, "Rétrécissement mitral pur avec nanisme," *Bull. et mém. de la Soc. méd. des hôp. de Par.*, 3 sér., 1908, xxv, p. 636.

DISCUSSION.

Dr. ALEXANDER MORISON said he had been in the habit of describing this condition as cardiac infantalism, though perhaps not with perfect accuracy. He thought Dr. Weber's explanation attributed almost too much ingenuity to Nature. If this child had mitral constriction very early in life it was probably some congenital defect, because he believed it was agreed that a constricted mitral valve in quite early life was very rare. But a mitral orifice otherwise diseased might gradually shrink. He had seen stunted growth associated with heart disease in which the condition to be dealt with was not mitral constriction, but mitral regurgitation and an enlarged heart with adherent pericardium. He believed the stunting of growth in these children was on much more general lines than that of a limitation of the aortic irrigation of the body generally. He hesitated to speak of hypo-thyroidism in connexion with these cases, but in a marked case of enlarged heart with adherent pericardium in a stunted child he found the thyroid very small: and he gave such children small doses of thyroid on the supposition that the condition was not unusually associated with cardiac infantilism.

Dr. MORLEY FLETCHER regarded the case as one of mitral disease probably associated with adherent pericardium. There appeared to be a greater degree of hypertrophy and dilatation than could be accounted for by the presence of mitral disease alone. He considered Dr. Weber's explanation of the retarded bodily growth in such cases as probably correct, that it was due to a poorly developed aorta. Many years ago the late Dr. Gee suggested this and used the term "hypoplasia of the aorta" in connexion with this form of infantilism.

Dr. JEWESBURY said he thought the enlargement of the left ventricle was probably due to mitral regurgitation, rather than to aortic disease. There was a well-marked systolic murmur to be heard at the apex, as well as a rough mitral pre-systolic, which was very loud. The systolic murmur was well conducted out into the axilla. He thought double mitral disease was present.

Dr. PARKES WEBER replied that his notes said the enlargement of the left ventricle was obvious both from the ordinary examination and that by X-rays. He attributed it to mitral incompetence, which was certainly present in addition to the mitral stenosis, and was sufficient to account for the ventricular hypertrophy. Possibly there might be adherent pericardium in addition, as Dr. Morley Fletcher suggested, but there was no proof. In one or two of the French cases of mitral dwarfism Dr. Weber thought that mention had been made of insufficiency of the thyroid gland, and thyroid treatment might be tried, as suggested by Dr. Morison. When mitral stenosis developed very early in life, Nature, though she could not enable the body to get rid of the obstruction at the mitral valve and make the supply of arterial blood normal again, nevertheless did the best thing that could be done under the circumstances—namely, she limited the growth of the body so as far as possible to adapt the body to the limited supply of arterial blood. When grave defects in the cardiac mechanism originated later in life, after the body had finished growing, this kind of "conservative adaptation" was of course no longer possible, but Nature did what she could do, by inducing compensatory hypertrophy of the muscular walls of the cardiac chambers so as to increase their propelling power, where that was possible.

Case of Mongolism.

By F. G. Crookshank, M.D.

M., aged 5 years, is the younger of two boys born to European parents, now aged 36 and 28 years respectively. He has dark, wiry hair and a yellow skin. The mother says that when born he was "almost black," but that he is now getting fairer. His eyes are not "oblique," but the folds are of a characteristic form. The tongue is not fissured, and the head is not short, as in the Kalmucks and other Altaic Mongols, but long, as is the case, according to Siegert and others, in a certain proportion of "Mongoloids." The lips are very plastic, and the mouth is characteristically funnel- or trumpet-shaped. The characters of the canine and incisor teeth are simian, as in the Javanese and Malays. The pelvis is "high," and the lines of the "groin-fork" include an acute angle. The radio-humeral index is also high. The boy is in no sense an idiot, or imbecile, but is peculiar. He bites and snaps, and is, amongst his comrades, a famous hooter and contortionist.

Case of Mongolian Idiocy.

By Edmund Cautley, M.D.

The child was born on November 13. The mother was aged 38 years and the father 35 years. Patient is an only child, and since birth has had much difficulty in suckling, and on crying went blue. She was admitted with considerable pulmonary catarrh. The head is unduly small, measuring only 14⅛ in., and somewhat brachycephalic; and the fontanelle is very large. The child is unable to hold up its head, except momentarily. The palpebral apertures are somewhat slanting, with epicanthic folds and swollen lids. The tongue is unduly large, and the child sucked it to some extent. There is general hypotonia. The little finger is unduly short, so, too, the thumb. Dr. Cautley did not doubt that it was a true Mongolian idiot. One might talk of Mongolian characteristics, but he thought the term should be used only in connexion with such a case as he showed. This child had attacks, while in the acute stage, of pulmonary catarrh, periodic group breathing, such as is sometimes seen in association with tuberculous meningitis; rapid respiratory movements, of equal depth, followed by a pause. When seen in tuberculous meningitis it is sometimes associated with dilatation of the pupils, such dilatation subsiding during the pause. But that did not happen in this case. Recently he saw another Mongolian child who had remarkably rapid breathing, the ordinary rate being 60 to 80 per minute. When first examined there was nothing to be found except a little postnasal catarrh, not sufficient to account for this rapid breathing. It went on for a month, and then the child got infected with a cold which was about the house, and developed bronchial catarrh and broncho-pneumonia, with occasional Cheyne-Stokes breathing. That subsided, and the temperature came down. There was, however, a relapse, with more fever, and the child died. The temperature had kept at 103° to 104° F. for five weeks; an unusual duration, for Mongolian idiots stand illness badly. In the case he now showed he would not like to say the grouped breathing was definitely connected with the Mongolism, or that it was due entirely to the pulmonary condition. There was also a definite congenital systolic murmur over the base of the heart, perhaps due to a patent ventricular septum. Congenital morbus cordis is frequent in Mongolian idiots.

DISCUSSION.

Dr. SHUTTLEWORTH asked if there was a cardiac affection in Dr. Cautley's case. Dr. CAUTLEY: No. He had supposed the peculiar breathing might be connected with the condition of the heart. He agreed that the case bore on it the impress of Mongolism, which he, perhaps from a narrow point of view, had been accustomed to look at somewhat exclusively in connexion with children of defective intelligence. With regard to the first case which had been shown by Dr. Crookshank, he supposed it might be due to his (the speaker's) narrow point of view, but he failed to recognized any of the characteristics in it which one was accustomed to associate with Mongolism in defective children, except, possibly, the presence of epicanthus and the condition of the palate, which latter, however, was, as Dr. Crookshank had said, found in cases not suspected of Mongolism. Of course, from the ethnological point of view, it was interesting to trace in children of European parentage traits showing some connexion with Mongolian ancestry. And no doubt, as had been shown in a paper by Dr. Crookshank in the *Universal Medical Record*, there had been a very wide diffusion of the Mongolian element throughout Europe; and possibly one might see cases of reversion to a remote ethnical condition of ancestry in cases of contemporary European parentage. But he thought it was rather stretching the term to brand as Mongoloid a child who had none of the definite characteristics, either physical or mental, which one had been accustomed to associate with that designation. For instance, obliquity of the palpebral fissures was not at all marked in the boy shown by Dr. Crookshank, the tongue was a very ordinary one, such as one might see in 90 per cent. of the children in any school, there was not the characteristic "Mongol" formation of the hands, nor, he supposed, of his feet either. It was true that the hair was somewhat wiry and black, and he was told that the section of it would be circular; but that also existed among other children than those of Mongolian type. Dr. Crookshank stated, and probably with truth, that it had been said that the early condition of many European children was more or less allied to that of the Mongolian race, and that the resemblance passed off as age progressed. But in any English community, taken haphazard, it would not be difficult to pick out certain features which might remind one of the Mongolian race, though one might receive the assurance that such person had no Mongolian blood in his veins. Care should therefore be taken (in this Section at least) not to include in the term more than the term connoted to the medical mind, whatever it might impart to ethnologists. There were, no doubt, cases with certain mental as well as physical characteristics which had but a "dash" of Mongolism in them; one saw this among comparatively high-grade children in special schools. He thought he had seen it in schools where certain backward children did not display the whole of the characteristics found in the typical Mongol, but there was an approach to some of them. And he felt that the dullness which accompanied that modified physical condition was probably due, at any rate in a certain degree, to Mongolism. But to talk of a child, aged 5 years, who was fairly bright (though odd in some of his ways) and had no definite physical or mental conditions indicating Mongoloid abnormality, as "Mongoloid" would, he believed, lead to regrettable confusion. He might be wrong, but he did not agree with the extension (for medical purposes) of the designation so far as Dr. Crookshank favoured.

Dr. CROOKSHANK, in reply, said that he had shown the case to illustrate the point that quite a number of European children, not idiots or imbeciles, exhibited Mongoloid characteristics—a fact quite explicable on Klaatsch's hypothesis of the origin of human stocks. He was quite willing not to call the case one of Mongolism, but it did seem to him a little illogical to deny the term Mongoloid, or Mongolian, to children who presented Mongoloid resemblances, and to restrict it to those cases, of a lower grade, in which features like the fissured tongue (simian rather than Mongolian) were present, or such evidences of cerebral aplasia, as peculiarities in respiration, that might equally be observed in negro idiots. Evidences of lack of bulbar development might occur in any race without the coincidence of any Mongolian features. He suggested that Mongolian characteristics only occurred in such cases of arrested or delayed cerebral development as those in which, on Klaatsch's hypotheses, there was a phylogenetic explanation for their occurrence. He quite agreed that Mongolian resemblances were quite common amongst London children. Such was indeed his point. But it was in only a certain number of these cases that arrest of development in the lower stages occurred and that there was presented to us the real "Mongol idiot," or imbecile. The points with regard to the dental characteristics could be well studied at the Natural History Museum at South Kensington. Dr. Crookshank had hoped to have shown another case in which the primitive long and flattened palate was well marked. The boy was, however, ill and in his stead another boy, with negroid features and a very primitive palate and set of teeth, had been shown to illustrate the point, sufficiently well known perhaps, that the *vertical* "epicanthic" fold was not the true characteristic of the Mongolian eye, and may be well marked without any suspicion of "Mongolism."

Congenital Coalescence of Radius and Ulna.

By J. E. Adams, F.R.C.S.

Two children in the same family with deformity noticed from birth. Both forearms are held in a position of pronation and although there is movement at the inferior radio-ulnar articulation, supination is almost absent. There is a forward and outward curve in the shaft of the radius. Radiography has shown that the radius is fused with the ulnar a short distance below its head, and the deformity is the same in both forearms and in both children. By the kindness of Mr. Shattock I am enabled to show a specimen from the Museum of St. Thomas's Hospital which illustrates precisely the same deformity. A similar case has been described by Dawson in the *British Medical Journal*, October 5, 1912. Suggestions are invited as to treatment.

DISCUSSION.

Mr. Adams added that he showed the cases on account of the rarity of the condition, and he would be glad of advice as to the correct lines of treatment. He exhibited the specimen, and invited members to compare it with the X-ray picture. Should operation be undertaken to enable supination to be carried out? Or should it be left alone? Mr. Dawson dealt with his case when the lady was aged 33 years, and he first removed a bridge of bone, so that the radius was separated from the ulna, and he interposed a piece of muscle between two bones. Finding that inadequate, Mr. Dawson freed the lower end of the radius and ulna from one another. Thirdly, Mr. Dawson divided the interosseous membrane, and, fourthly, he removed the remaining portion of the radius above the insertion of the biceps tendon. Lastly, he did an osteotomy of the lower part of the shaft of the radius, and put the hand in the position of supination. The ultimate result was said to have pleased the patient. But Mr. Dawson did not say that he had operated, or proposed to operate, upon the other arm, although the deformity was bilateral. It was very difficult to deal with the condition operatively, but if operation were undertaken, Mr. Adams thought one must remove a considerable amount of radius above the position of insertion of the biceps; probably that should be followed by osteotomy of the radius, and the hand put in the position of supination.

Mr. LAMING EVANS said he had seen three such cases, two of them under his own care, which he had left alone. The degree of functional disability did not appear sufficient to justify surgical intervention. The other was the case mentioned in connexion with Mr. Dawson, which he had seen with Mr. Dawson after the fourth operation. He saw the patient with Mr. Dawson, because the patient wanted yet another operation done—namely, osteotomy of the lower end of the radius, so as to obtain a still further supinated position of the hand. The result was, however, not commensurate with the surgery which had been practised. She was a neurotic woman, who seemed rather to desire operation; and Mr. Dawson was pleased to have another opinion against further operative procedures. Of the several operations that had been performed, he thought that the osteotomy of the lower end of the radius had been most beneficial. The actual movement at the new radio-ulnar joint was very small; the apparent pronation and supination was performed almost entirely by rotation at the shoulder-joint. The cases under his own care were interesting, in that the deformity was present in both mother and child in exactly the same form.

The PRESIDENT said he had been glad to hear that Mr. Laming Evans had seen the case, because he (Mr. Tubby) had also seen the case three times. Certainly the result was small compared with the amount of surgical work which had been carried out.

Mr. ADAMS replied that he saw these children nine months ago for the first time. He had not done anything to them yet, and he was no more disposed to now that he had heard the remarks of members.

Lymphangioma of the Axilla.

By DOUGLAS DREW, F.R.C.S.

BABY, aged 9 months, was admitted to the Queen's Hospital with a large, soft, lobulated tumour of the axilla. The tumour occupied the whole axillary space extending up to the clavicle, and required an extensive dissection for its removal, as it surrounded the vessels and nerves; in order to facilitate this the pectoralis minor was removed with the tumour. The specimen is a good example of this variety of tumour.

Cicatricial Contraction of the Thumb following a Burn about Eighteen Months previously.

By Douglas Drew, F.R.C.S.

The X-ray photographs show the thumb drawn back and bound down to the wrist by dense fibrous tissue; after dividing the contracted tissue and liberating the thumb, the raw surface was covered by a large flap cut from the back of the hand.

Separated Epiphysis of the Femur treated by Plating.

By Douglas Drew, F.R.C.S.

The operation was performed on December 9, 1909. The child is shown, as the plate was not removed as the child was lost sight of. The X-ray photograph shows that, in the process of growth, the screw in the epiphysis has been gradually drawn out; there is about ½ in. of shortening of the limb and the movements are free. As the plate causes no inconvenience and is not hindering the growth, it does not appear necessary to remove it.

DISCUSSION.

Mr. Drew said he showed the case because it had been generally stated that where a plate had been used for separating the epiphysis, it was advisable to remove the plate owing to the possibility of arrest of growth of the limb. He believed the impairment of growth in this child was not due to the plate, which was placed at the outer aspect. Otherwise, he thought there would have been very marked knock-knee, whereas it was extremely slight. It was not to be wondered at that there was some shortening, considering the severity of the injury at the time. Numerous attempts were made to reduce the separated epiphysis, and he did not see the child until the ninth day after the injury; the skiagram then showed the epiphysis displaced on to the front of the shaft of the bone, and he had some difficulty in reducing it. When the pressure was released the epiphysis shot forward again out of position. Therefore it had to be held in position while the plate was being fixed. He had a skiagram taken a month ago, and it showed how, in the process of growth, the screw had gradually been drawn out upwards, so that it lay opposite the epiphyseal line.

The President said this was a most excellent case, and he congratulated Mr. Drew on it. He once had a similar case in a boy, aged 13 years, in whom the epiphysis had been out of place, and lying on the front of the shaft of the femur, for six weeks. He had great difficulty in getting it back into place, and had to separate nearly all the soft attachments, so that he feared necrosis of the epiphysis. He was able, however, to lever it into place, and then inserted a long screw, $3\frac{1}{2}$ in. in length, on the under cartilaginous surface of the epiphysis, and through it and so into the shaft of the femur. He figured and described the case in the *Lancet*. The boy had a perfect knee-joint, and was still (nine years afterwards) going about with the screw in position.

Costo-transversectomy ; Spinal Caries and Mediastinal Abscess.

By EDRED M. CORNER, F.R.C.S.

PATIENT, a male, aged 6 years 5 months, was admitted on December 6, 1909, to the Hospital for Sick Children. On admission there was some kyphosis in the lower dorsal vertebræ with indications of abscess formation on the left side at the level of the eighth dorsal vertebra.

X-ray (November 30, 1909) showed the existence of a large mediastinal abscess in front of lower dorsal vertebra.

Operation (December 14, 1909): Costo-transversectomy; abscess opened and drained on left of spinal column at the level of the eighth dorsal vertebra.

X-ray (December 29, 1909) shows abscess cavity and some destruction of the ninth and tenth dorsal vertebræ, especially in the transverse processes of the left side and in the heads of the corresponding ribs.

Operations: On February 18 and March 16, 1910, the abscess cavity was scraped. On May 17 a little more of the ninth and tenth ribs on the left side was excised.

X-ray (July 8) shows no indication of abscess cavity remaining; there is formation of new bone in the damaged ribs.

Operation (November 22, 1910): Sinus scraped.

On February 28, 1911, patient was sent to a convalescent home in a Phelps's box; he still had a small sinus.

October 1, 1912: Readmitted.

October 3, 1912: An emulsion of bismuth and iodoform was injected into the sinus. X-ray shows small cavity containing bismuth and considerable destruction in lower dorsal vertebræ, especially the eighth, ninth, and tenth. The sinus gradually closed after the injection.

Present condition: Patient has a marked but not extreme degree of kyphosis in the lower dorsal region. On the left of the spine at the level of the ninth dorsal vertebra there is a depression marking the position of the old sinus, which is now healed.

X-ray (February 10) shows no abscess formation, but there is destruction of the bodies of the lower dorsal vertebræ, especially the ninth and tenth.

Mr. CORNER added that when the abscess was opened the anterior wall of the abscess was found to be the posterior wall of the heart, so that it was impossible to curette the abscess cavity properly, with the result that, although it was sewn up at the time, it broke down, and gradually a sinus formed. This abscess took not less than two years to heal. With such knowledge before one it would be wise, in dealing with a case of the kind in future, to content oneself with making an incision in the skin, and then aspirating the abscess. The other point which the case taught him was, that before the abscess was opened the skiagram showed a shadow which could be distinguished from that of the heart. But after the abscess had been opened it was impossible to distinguish its shadow from that of the heart. Hence later skiagrams were not of so much clinical value.

Case of Congenital Absence of Fibula, Deformity of Tarsus, and Absence of Toe.

By E. M. CORNER, F.R.C.S.

THE patient is a baby and presents a perfectly typical appearance. The skiagram shows the absence of the fibula. The tibia is bowed sharply and, perhaps partly in consequence, seems shorter than the other. The summit of the convexity of the tibia is sharp and presents a groove or sharp dimple over it. The movements of the ankle-joint are naturally somewhat restricted. The fifth toe is apparently absent. The doctor who first saw the patient diagnosed an intra-uterine fracture which had united. The absence of the toe led to the correct diagnosis, confirmed by the skiagram, of "congenital deformity of leg." In addition, the skiagram demonstrated the characters of the deformity: absence of fibula, one centre of ossification representing both the astragalus and calcaneum, absence of the fifth toe and its metacarpal.

The centre of ossification of the astragalus is present at the end of the seventh month of fœtal life and that of the calcaneum during the sixth month. Both show in a skiagram of this child's foot.

The PRESIDENT said he must have seen between thirty and forty such cases during the last twenty years, and in that time he had tried various forms of treatment. His opinion was that the only real method of value was to fix the foot absolutely at the ankle-joint. It was of no use attempting to get a movable ankle-joint and a suitable useful one at the same time. When the patient was over 8 years of age, one should do an arthrodesis, and, if necessary, reinforce the union of the surfaces by plates on either side, so as to make it as strong as possible. The other alternative was amputation. Many of these cases were so awkwardly placed, and the joint surfaces were so irregular, that they were very difficult to deal with. He remembered one where there was a projection on the front of the tibia, and the epiphyseal line was so wavy that he did not know what he could do with it. Probably the case would come to amputation.

Chronic Albuminuria with Hepatic Enlargement.

By EDMUND CAUTLEY, M.D.

THIS girl, aged 8 years 2 months, is the daughter of healthy parents, but a grandparent and great grandparent are said to have died of phthisis. She has had measles, varicella, mumps, and pertussis. During 1910 she was an in-patient at another hospital for four months for albuminuria. Later on, a gland was excised from the left side of the neck, and tonsils and adenoids were removed. From June 20 to July 8, 1911, she was admitted to the Belgrave Hospital for bronchitis, albuminuria, and right otorrhœa. Two months before this she had had pneumonia, bronchitis, and measles. The attack of bronchitis subsided quickly. Her urine contained much albumin but no casts. A year later she was again admitted for bronchitis and the urine was found to contain a fairly dense cloud of albumin. In November last she was admitted for prolapsus recti. At present she has been an in-patient since January 28 for an attack of bronchitis and anasarca. The face and legs had been moderately swollen for four days. The liver extended 2 in. below the ribs and the spleen was palpable; the abdomen being considerably distended, though there was no dullness in the flanks. Urine, 0·2 to 0·7 per cent. albumin. A centrifugalized specimen on February 12 showed many white cells, several granular and hyaline and a few epithelial casts. The total daily amount of urine varies greatly—e.g., 12, 22, 30 and 13 oz. on successive days. Von Pirquet's reaction is negative and there is no reason to suspect syphilis.

The case is shown with a view of eliciting opinions as to the nature of the albuminuria, the causation of the hepatic enlargement, and the probable prognosis. I should like, also, to elicit the experience of members on the value of experimental tests of the permeability of the kidneys, the excretion of chlorides and urea, and on the presence of urea in the blood serum.

The liver has decreased in size while under treatment by rest and diet.

Summer Diarrhœa and Summer Heat.

By H. Charles Cameron, M.D.

I feel that perhaps I ought to apologize for bringing to your notice so well worn a subject. I should not have done so had it not been that the Epidemiological Section has recently held very frequent debates upon this very topic. The result has been, I think, that the subject of late years has been dealt with too exclusively from a single point of view—that of the epidemiologist. I believe that the carefully argued papers contributed to the Section of Epidemiology by Niven [4], Dudfield [1], and others have had a very great effect in influencing current opinion on the subject, and that as a result all factors in the ætiology of summer diarrhœa other than that of microbial contamination have been too much neglected. All of these papers start with the assumption that the symptom diarrhœa may be taken as constituting a specific disorder capable of being subjected to a mathematical analysis. Dr. Dudfield, for example, opens his paper, read on February 23 of last year, with the following words: "I do not propose to take up any time in discussing what is meant by the term 'infantile diarrhœa,' as I assume that we are all thinking of the same clinical picture." It is because I believe that in dealing with the statistics of infants dying of diarrhœa it is quite impossible to make any such assumption that I have brought the subject before the notice of this Section. Into the total of deaths from diarrhœa among infants in the third quarter of any year a vast variety of disorders is crowded. Many of the cases have been seen under conditions which render an accurate diagnosis unlikely; not a few have received no medical attention at all. Because in infancy one function alone is highly developed—that of absorbing food—disease of whatever sort is apt to show itself with symptoms of alimentary disturbance, so that diarrhœa becomes the common accompaniment of disorders of the most varied nature. It is not my purpose to deny that acute infective enteritis undergoes exacerbation during hot weather or that increased decomposition of milk plays an important part in the production of alimentary disorders. I do contend, however, that even if it were possible to secure a faultless supply of milk we should still witness an enormous rise in the frequency of the symptom of diarrhœa during hot weather.

That the clinical diagnosis between infective and non-infective diarrhœa in the majority of cases is one of great difficulty is generally admitted. Certainly severe and even fatal symptoms, usually regarded as characteristic of so-called summer diarrhœa, may supervene upon an attack of dyspepsia. The products of fermentation of food in the intestine which serve to provoke excessive peristalsis have also a deleterious effect upon the epithelial lining of the alimentary canal, so that their long-continued action or extreme concentration may ultimately lead to the most profound intoxication. With the loss of selective control over absorption by the cells lining the alimentary canal and with the subsequent degeneration of the liver cells the usual symptoms of intoxication, characteristic of summer diarrhœa, appear—high pyrexia, partial coma, immobility of the limbs, unnatural posture, albuminuria, alimentary glycosuria, and irregular, disordered breathing. I believe that it is possible to recognize all stages between a simple dyspepsia and a fatal food intoxication, and that we should protest against the tendency to regard all fatal cases of diarrhœa in the summer as infective in origin.

It may assist in making my meaning clear if I tabulate the possible relations which may exist between the height of the thermometer and the amount of diarrhœa prevalent in infants and endeavour to assess the part played by each.

(1) The increase in diarrhœa may be due to the *direct* effect of heat upon the infant.

(2) The increase may be due to the *indirect* effect of heat by lowering the tolerance of food, and by increasing the danger of overfeeding in an infant rendered thirsty by the loss of fluid which diarrhœa, vomiting, and excessive perspiration entail.

(3) Heat may exert an *indirect* effect by lowering the immunity of the child to infections of all kinds, both of the alimentary tract and elsewhere, and it may indirectly swell the total mortality by

aggravating the course of all alimentary and infectious disorder of whatever nature.

(4) Heat may exert an *indirect* effect by favouring the growth and multiplication of micro-organisms outside the body, or by promoting a favourable means for the transport of these organisms, by the multiplication of flies or by the spread of dust.

It is because I think that recent writings have dealt too exclusively with this last supposition that I have ventured to ask that some greater emphasis should be laid upon the others.

(1) The view that the increase of diarrhœa is in great part the direct result of heat is supported by a variety of considerations. It is well known that the power of the infant, and especially of the weakly infant, to maintain a constant temperature is ill developed. Although the necessity of preventing an excessive loss of heat is insisted upon by all writers upon infant feeding, less stress is laid upon the importance of avoiding the overheating of the child. Yet summer diarrhœa in its most severe forms is almost confined to those of very tender years. Among the occupants of crowded courts and tenements again and again only one member of the community is affected—the infant under 1 year of age. With the attainment of a higher degree of adaptation between heat-loss and heat-formation, the older children tend more and more to escape. In estimating the direct effect of heat upon the sensitive organism of the child, it is to be remembered that we have to take into account not the temperature of the outer air, much less the temperature of the 4-ft. earth thermometer, but the temperature of the stifling room in which the infant is confined. Where rooms are small and ill-ventilated, where, from lack of windows or in back-to-back houses, there is no through draught brought about by the difference of temperature between the back and front of the house owing to the position of the sun, where the percentage of moisture in the air is high, either from overcrowding or from cooking or washing of clothes, observation shows that diarrhœa is unusually common [2]. Again, many records have shown that in times of great heat the temperature of even healthy infants is raised, while their average gain in weight is reduced. Both of these symptoms occur with considerable constancy during hot weather even in cases which are still quite free from symptoms of diarrhœa and vomiting, and they may be regarded as prodromal symptoms sometimes preceding an attack of diarrhœa by a long interval and culminating in the attack. For example, Schloss [7], who has lately studied the question in Berlin, recorded the average daily gain in

weight of infants who were free from diarrhœa in two of his wards, with the following result :—

	May and June before the heat	July and August during the heat	Sept. and Oct. after the heat
First ward	17·5	10·0	14·0
Second ward	16·0	10·0	18·0

Lastly, the close correspondence between the temperature curve and the curve of the total deaths from diarrhœa is itself an argument in favour of the view that the effect of heat is immediate and not mediate. Wherever slum buildings and overcrowding exist the two curves keep pace alike in rise and fall. In this connexion the observations of Finkelstein, Willim, Liefmann and Lindemann, and others upon the correspondence of the daily temperature with the daily total of cases are interesting, for the relation then becomes even more marked than in the usual figures in which the unit of time is a week. If each case arises by infection and is propagated by infection, we should expect that the epidemic when once established under favourable circumstances would sometimes of its own momentum break beyond the bounds set to it by the temperature curve, and that its decline, if occasioned by exhaustion of infective material, would not always coincide with the fall in the thermometer.

(2) We must consider that in times of great heat and of great humidity, on the ordinary ratio of food, the production of heat may be so great that pathological effects are produced. In adults the consumption of food under such circumstances is instinctively lowered. While less is eaten, more is drunk to make good the loss of fluid from increased perspiration. In infants the difficulty is greater because the infant's food and drink are bound up in one and the same fluid. No doubt the infant fed at the breast attains the same object though in a different way. The provisions against overfeeding in the breast-fed infant are very effective. When the child ails, the diminished suction soon interferes with the free flow of milk, and it requires some days of vigorous convalescence before it is again established. Moreover, when the breast is insufficiently emptied the last milk is not obtained, so that the percentage of solids, and especially of fats, falls. No doubt, too, the alteration in the mother's diet, the greater intake of fluid and the decrease in the amount of solid matter reacts to some extent upon the milk and produces a fluid of less concentration. In the bottle-fed none of these safeguards are present, and the thirsty child, if left to itself, is more apt to suck an excess from a bottle, which offers no

fine adjustment of amount and composition from day to day in accordance with the needs of the child.

Salle [6] has studied the question experimentally in puppies. Prolonged exposure to heat produced a rapid loss of weight, diarrhœa, vomiting, and a rise of temperature. When a diverticulum of the stomach was made in the manner suggested by Pawlow the gastric juice was diminished both in amount and in digestive power while the hydrochloric acid secretion decreased. Such a failure of digestive power must give rise to increased fermentative changes in the carbohydrate constituents of the food with consequent stasis and diminution of gastric peristalsis. Dyspepsia so produced, while common enough during a spell of hot weather even among healthy infants, becomes almost the rule among the neglected, artificially fed children of the poor, living in hot, close, stagnant atmosphere of the slums of cities.

(3) Heat may exert an indirect effect by lowering immunity to infections of all kinds. I know of few statistics which deal with the relative frequency in summer heat and winter cold of infections other than those of the alimentary tract. It must be common experience how frequently bronchitis and broncho-pneumonia are associated with severe attacks of diarrhœa in the summer. Furunculosis and subcutaneous abscesses appear to be much more common in hot weather. Among the infants in his clinique, Meyer [3] reports a rise from 15 per cent. of total cases to 41 per cent. in the former and from 6 per cent. to 30 per cent. in the latter. The effect of exposure to fresh air, wind and cold in increasing immunity and of the reverse conditions in lowering it is too wide a subject to be considered in a few words. "Evidence," says Pembrey [5], in this connexion, "steadily accumulates to show that the bad effects of overcrowded rooms are due, not to diminshed oxygen, not to an increase of carbon dioxide, not to any toxic discharge from the human body, but to the absence of free ventilation, to the warm and humid atmosphere, which reduces the respiratory exchange and metabolism of the inmates, and renders them less resistant to the attacks of micro-organisms." No doubt alimentary infections share in the common increase, but among the cases which go to swell the total deaths from diarrhœa during the summer there are many in which the diarrhœa and vomiting are symptomatic of other disorders.

(4) There remains the supposition that heat is prejudicial because of the increased bacterial content and increased decomposition of milk. If I have laid emphasis on the part played by other facters than the

contamination and decomposition of milk, it is not because I wish to underrate the importance of a pure supply of milk. I believe, however, for the reasons I have given, that even if we could ensure a perfect supply of milk, we should still witness during hot weather a rise in the mortality from diarrhœa little less than that to which we are now accustomed. If the cause of summer diarrhœa is to be found in the multiplication of bacteria in the milk, then some explanation must be found for the frequency with which infants fed at the breast are attacked, and for the high mortality among infants fed upon condensed milks and patent foods, in which the bacterial content is relatively low. Moreover, it is to be remembered that the organisms which occur in milk are usually of a harmless character, and that butter-milk has even proved itself of some value in infant feeding. No doubt the organisms in milk sometimes include others less innocent in character, yet a vast amount of bacteriological work has not succeeded in establishing a causal connexion between any one organism and so-called epidemic diarrhœa.

In conclusion, I may put forward for acceptance or criticism the following propositions:—

That neither direct overheating of the child, nor disturbances of digestion, nor intercurrent infective disorders, nor direct infection of the alimentary tract, is sufficient of itself to explain the rise in the mortality from diarrhœa in the summer, but that in that rise each plays a part; that of all the deaths registered as due to diarrhœa in the summer a small fraction only is due to bacterial infection of the alimentary tract; and that the progress of knowledge with regard to the prevention and treatment of diarrhœa has been not a little hindered by regarding the problem from a standpoint too purely epidemiological.

If so much is granted it follows that greater emphasis should be laid upon such prophylactic measures as the supply of an adequate quantity of water for the child during hot weather, the reduction of the carbohydrate percentage in the food, the use of cooling baths and of light and porous clothing, and the proper ventilation of rooms, courts and alleys. New York, which suffers so profoundly from the disease, has taught us a lesson in the provision of rapid means of transport of overheated infants to the fresh and moving air of the sea coast.

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[4] **Niven.** "Summer Diarrhœa and Enteric Fever," *Proc. Roy. Soc. Med.*, 1910, iii (Epid. Sect.), p. 125.

[5] **Pembrey.** "Recent Advances in Physiology," 1906.

[6] **Salle.** "Über den Einfluss hoher Sommertemperaturen auf die Funktion des Magens," Vortrag in der *Gesellsch. f. Kinderheilk.*, auf der 83 Vers. *Deutsche Naturforscher u. Aerzte*, Karlsruhe, 1911.

[7] **Schloss.** "Ueber Säuglingsernährung," Berl., 1912.

Dr. **Langmead** said that there were so many varieties of the condition called summer diarrhœa that it made the whole subject a very difficult one to speak on. It was difficult properly to criticize a paper of this kind in the present condition of ignorance on the flora of the intestinal tract in association with infantile diarrhœa. There seemed to be several forms of infection. For instance, the infection most common in America differed from that most common in London. Those who had read the accounts of the New York epidemics must have been struck by the fact that the greater proportion of children there passed blood and mucus in their stools, whilst a comparatively small proportion of the infants suffering from summer diarrhœa in London did so. The larger proportion of severe cases in London were choleraic cases, whereas in America they were dysenteric. He took it that the infection in the two cases was not the same. It varied also in different epidemics in London, and cases at the beginning and end of a seasonal outbreak often failed to resemble those at the height of the epidemic. We could not yet say whether a particular case was due to a specific infection, or to the direct effect of heat, environment, or faulty dieting.

Section for the Study of Disease in Children.

March 28, 1913.

Dr. J. WALTER CARR, Vice-President of the Section, in the Chair.

Case of Tremor (? Congenital).

By FREDERICK LANGMEAD, M.D.

THE patient, a girl, aged 10 years, has definite tremor of both upper limbs, moderate in amplitude. It interferes somewhat with school work, but otherwise causes no discomfort. It has been noticed particularly for the last six years, but probably dates from infancy. There are no signs of organic nervous disease.

The CHAIRMAN (Dr. Walter Carr) agreed that the case belonged to the class of congenital tremor, concerning the nature and origin of which nothing was yet known. Some years ago he saw a man with marked tremor of this kind, which had started in early life and continued to adult age. It did not seem to vary much, nor to cause much discomfort. The patient was shown before several societies, but no fresh light was thrown on his condition. Probably in this child the tremor would continue.

Case of "Nervous Cretinism."

By FREDERICK LANGMEAD, M.D.

J. M., A BOY, aged 10 years. He is an undersized and mentally deficient boy who first attended the Hospital for Sick Children, Great Ormond Street, at 7½ years, and then exhibited the usual signs of cretinism to a moderate degree. He could not sit alone, stand, or talk, and was "dirty in his habits." His appearance was characteristic.

Under thyroid treatment he has improved considerably in his general condition and can now walk, although badly, and is clean in his habits. His vocabulary is still very limited and pronunciation imperfect, only a few simple words being used and these in a monotone. He has lost most of the aspect of cretinism. The feature of special interest is a co-existing cerebral diplegia affecting chiefly the legs. These are stiff, adducted and their deep reflexes are exaggerated. The plantar response is sometimes definitely extensor. The arms are little, if at all, affected; the triceps jerks being brisk and (?) slight rigidity being present. He stands in the knock-kneed position, with feet abducted and walks with adducted legs, but without scissor-leg progression. The thyroid cannot be felt.

I have called the case "Nervous Cretinism" after Major McCarrison, I.M.S., who has described the association of cretinism with spastic paralysis in his study of endemic cretinism in the valleys of Chitral and Gilgit in India. He regards the paralysis as part of thyroidal inadequacy and obtained some success with thyroid therapy in such cases.

DISCUSSION.

Dr. LANGMEAD said that Major McCarrison had investigated 203 cases of cretinism, in one-third of which there were symptoms of cerebral diplegia as well as of cretinism. He brought this case in order to raise the question of the possible relationship of diplegia to thyroid inadequacy. The patient had been under thyroid treatment, and the usual evidences of cretinism had almost disappeared. He was still stunted in growth, however, and was defective in speech. The paralytic symptoms had improved also.

Dr. CAUTLEY asked what were the symptoms of cretinism in this case. Agreeing with Dr. Langmead's statement that there had been cretinoid symptoms, why should not the case be called cerebral diplegia with cretinoid symptoms? There was nothing extraordinary, though unusual, in the association of cretinism with injury at birth or other causes of diplegia. He objected to the term "nervous cretinism."

The CHAIRMAN asked as to the probable cause of the present degree of mental deficiency, which still seemed to be considerable, although there had been definite improvement. How far was the mental condition now due to the cretinism, or how far to the probable brain lesion causing the cerebral diplegia? This raised the question as to the extent to which cretins improved mentally under thyroid treatment. He had had a well-marked case which was treated with thyroid for some years; the child lost all the outward appearances of cretinism, and the mental condition became so much better that she could go to school, where, however, she remained in a very low standard. He asked what was Dr. Langmead's view as to the prospects in this child.

Dr. LANGMEAD replied that when first seen the child was stunted in growth, there was puffiness of the face, thickness of lips, projecting tongue, fullness about the eyes, the presence of so-called pads of fat, prominent abdomen, umbilical hernia, and severe mental disturbance. He had used the name advisedly in order to provoke discussion as to the relationship between the thyroid gland and the cerebral diplegia. It was a merely tentative use of the word, and there was need for much more investigation before its permanent employment was justified. There had been some improvement of the mental state under thyroid administration, and the diplegia was also somewhat better. The present mental state might be due to the diplegia. He was in ignorance as to the prospects in the case. Cases of cretinism were often seen in which the mental condition did not respond to the treatment, whereas the physical condition did so markedly. Good illustrations of that truth were the cases of two cretins, both of whom came for treatment under him at the same time when 3 months old. They were given thyroid in equal doses. In one the cretinism, both physical and mental, cleared up completely, and that child would now be regarded as a normal individual. In the other the physical side was so improved that the child looked the picture of health; but the mental state underwent a great change. Not only was there no improvement, but he was now a destructive and troublesome imbecile. There was thus need for great caution in predicting the effect of treatment, and sure prognosis was impossible.

Congenital Deficiency of the Cranial Vault.

By FREDERICK LANGMEAD, M.D.

BOY, aged 4 years. At the posterior part of the interparietal suture a small pulsating area, the size of a threepenny-piece, can be seen and felt. In this position there is a small depression admitting the top of the little finger. At the bottom of the depression a small hole exists, which apparently transmits cerebral pulsations. The up-and-down movement of the scalp was noticed by the mother.

Case of Congenital Thoracic Deformity.

By F. G. Crookshank, M.D., and Sidney Boyd, M.S.

This boy, aged 6½ years, is the subject of congenital thoracic deformity affecting the right side. There is a large depression in the upper part of the front of the chest, and an oval gap can be made out in the bony parietes, measuring 4 in. in its long diameter, which passes obliquely downwards and forwards from the axilla, and 3 in. transversely. The defect is apparently due to absence of the anterior portions of the second, third, and fourth ribs. The fourth rib appears to join the fifth. The sternum is asymmetrical and the right upper costal cartilages, which are attached to the sternum, are bent backwards. There is a marked hernia of the right lung. The clavicle and scapula are smaller on the right side than on the left. There is scoliosis of the dorsal vertebræ, convex to the right. The pectoralis major is fairly well developed, but is not so large as on the left. The pectoralis minor and the upper part of the serratus magnus are apparently absent. There is some depression of the corresponding area of the left side of the chest, but no bony

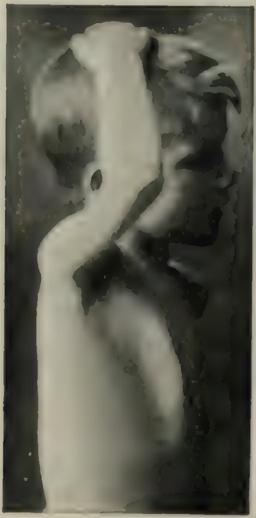

Fig. 1.

Congenital thoracic deformity.

defect. The condition appears to fall within the category described by Dr. J. Thomson in "Teratologia" (January, 1895) as "Congenital Thoracic Deformity." It will be observed that, as in Dr. Thomson's cases, if the arm be flexed at the elbow and carried across the chest, the deformed area is found to be almost exactly covered by the upper arm.

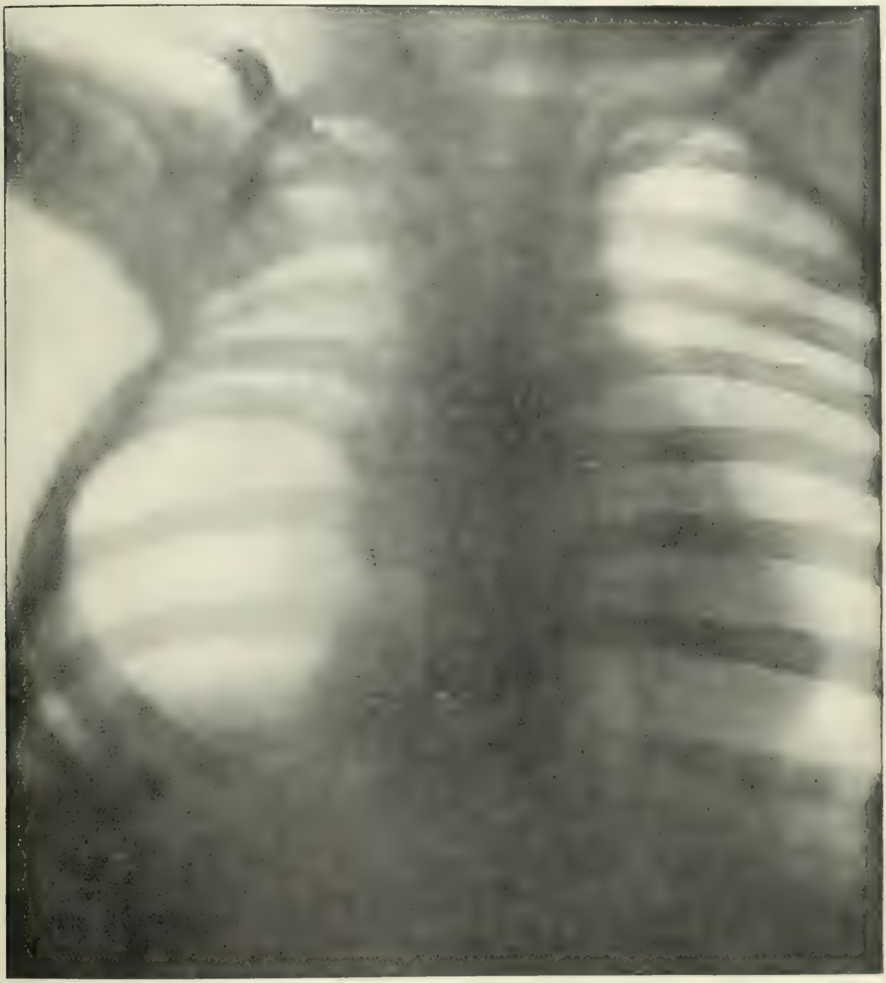

FIG. 2.

Skiagram showing the right side of the patient to the left of the picture. The advanced ossification of the anterior portions of the ribs on the deformed side is clearly indicated.

So far, then, Dr. Thomson's suggestion, that this kind of deformity is due to intra-uterine malposition, is supported. The mother gives two explanations: one, that she was, when pregnant, frightened by a motor car; the other, that the child was "not born so, but that it was done in the wash."

DISCUSSION.

Dr. CROOKSHANK added that, since the skiagrams had been examined a cervical rib had been detected on the left side. Moreover, it was obvious that the anterior portions of those ribs which were complete on the right side were more advanced in ossification than those on the left or "normal" side. Apparently this advanced ossification indicated an attempt of Nature to support, so far as possible, the right lung. With respect to the possible mode of production of this kind of deformity, it was interesting to refer to a case described, together with a good general account, by Auché and Lataste[1]. In their example congenital facial hemiatrophy was linked, on the right side, with deformity of the chest wall, and, as the authors observe, although the second deformity may be explained by malposition *in utero*, the first can hardly be. Mr. Boyd would refer to the question of surgical intervention, which had been considered.

Mr. SIDNEY BOYD said Dr. Crookshank urged him to do something, because he felt that if the child were to have whooping-cough, or any violent cough, it would be a serious matter for that lung. He thought it might be possible to get pieces of two or three ribs from the other side and put them in the gap. He would like to hear whether members had had experience of these cases. He was inclined to use bone-graft rather than periosteal, because he understood the former was more apt to live: and a bone-graft without periosteum was better than bone-graft with periosteum. He had not had experience in using a bone-graft in places where there was no periosteum.

Dr. F. PARKES WEBER thought that such deficiencies in the anterior wall of the thorax were analogous to the deficiencies occasionally met with in the anterior wall of the abdomen. No one would attribute the latter to malposition *in utero*. He did not, therefore, think that intra-uterine malposition was likely to be the correct explanation for the thoracic defect in the present case.

Dr. WHIPHAM agreed that the deformity could not be caused by the position of the child *in utero*. The case was similar to one he exhibited before the old Society some years ago,[2] the difference being that in his case one of the upper ribs came down and bridged over the gap which was on the left side. In that child there was also deformity of the scapula, hemiatrophy of the face, and deformity of the ear on the same side.

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[1] *Journ. de Méd. de Bordeaux*, 1913, xliii, p. 51.

[2] "Reports of the Society for the Study of Disease in Children," 1907, vii, p. 5.

Dr. G. A. SUTHERLAND said that several similar cases had been shown before the Section, but this was the most extreme form of this congenital lesion he had seen. From the skiagrams it did not appear on cursory examination that there was a definite absence of ribs; the ribs seemed to be there, but were widely separated in parts. At the anterior end there was a bending over, and the rib seemed to curve backwards very much, and it seemed to have been drawn or pushed in. He did not know how the case would appeal to the surgical mind, but he thought a physician would be inclined to leave it alone. He granted that there were dangers in the event of a violent cough, but he thought that simpler remedies than transplanting bone were available.

Dr. CROOKSHANK replied that he agreed that the theory of uterine malposition was scarcely tenable; but the fact that the arm did "fit in" gave some support to Dr. Thomson's idea. Dr. Sutherland's was the first suggestion they had heard that the apparently missing portions of the ribs might be present, but he would further study the skiagrams from that standpoint.

Mr. SIDNEY BOYD replied that he could not agree with Dr. Sutherland; he considered the ribs were absent. One could feel the fourth rib joining the fifth. There was an absence of shadow corresponding to what was seen on the opposite side; and one could feel where the third rib stopped short. He regarded it as a case of congenital absence of the bony elements of the ribs. The mother had settled the question of treatment for the time being by taking the child out of the hospital.

Case of Achondroplasia.

By A. S. BLUNDELL BANKART, M.C.

FEMALE, aged 7 years. Parents are second cousins. Deformity of chest first noticed at the age of 5 months. Bottle-fed infant. Suffered from bronchitis in infancy. Treated for rickets when 16 months old Treatment continued for three years without effect. Child stunted in growth. Head normal, not rickety. Teeth good, dentition not delayed. Chest narrow, sternum prominent (pigeon-breasted), very slight if any, beading of ribs, well-marked horizontal sulcus across lower part of chest Trunk short. Well-marked kyphosis. Lordosis. Upper extremities Great enlargement of epiphyses; skiagrams show marked irregularity and "cupping" of epiphyseal lines; diaphyses stunted; extreme laxity of wrist-joints, 180° of rotation at wrists, yet she has a good grip and can use hands well; looseness of elbows, head of radius on each side can be dislocated and replaced with ease. Lower limbs very slightly affected; epiphyses hardly, if at all, enlarged; diaphyses not stunted; slight genu valgum improved by treatment.

DISCUSSION.

Mr. BANKART explained that the diagnosis was tentative. In showing the case as one of achondroplasia; it was not meant to suggest that it was typical of that condition, but merely that the case must be placed in the achondroplasia group.

Dr. CAUTLEY said that if such a condition were seen in a younger child, it would be regarded as rickety; one saw similar deformities in the forearms of young children with severe rickets when they had been allowed to crawl a good deal. The points against it being rickets were that, first, it was noticed in infancy, if that meant at birth, and the head was not involved. It was not achondroplasia, for the shortening of the limbs was limited to the forearms. Rachitic changes are sometimes severe and limited to the long skeleton, the head being unaffected. He thought it was a case of persistent rickets, exaggerated in the forearms by much crawling during infancy. In some cases of achondroplasia there was marked beading of the ribs.

Dr. G. A. SUTHERLAND was inclined to agree with Dr. Cautley that the case was not achondroplasic, and yet he could not regard it as rickets. He was not prepared with a diagnosis, for which, indeed, the evidence submitted was not sufficient. The child might have had rickets in the ordinary sense of the term, but as now seen there were no active manifestations of it. He had noticed very marked spinal curvature, great deformity about the sternum, possibly secondary to the spinal condition, and also changes about the lower ribs. He had seen several similar cases, with marked changes at the epiphyses, at the Orthopaedic Hospital, some of the patients being young adults. One might say it was persistent or late rickets, but at 10 to 12 years and onwards it was difficult to say what the condition was. He preferred to keep an open mind for the present. In some of the cases he was referring to the bones were much bent, and osteotomy was resorted to, but a serious condition ensued, as the bones would not unite. He did not think this case was one of achondroplasia; there was no shortening of the limbs; the hand reached down two-thirds of the thigh, and there was no resemblance to that condition in the skull.

Dr. F. PARKES WEBER thought that the great argument against this case being one of achondroplasia was the absence of the peculiar shortness of the femora and the humeri, that was to say, the absence of the extreme relative shortness of the proximal segments of the limbs (not of the whole limbs), which was a striking characteristic of typical cases of achondroplasia. The present case resembled achondroplasia in one way, namely, in the fact that it was an example of mal-development, chiefly of the limbs, of unknown ætiology.

Mr. BANKART, in reply, said that the child was sent to the hospital as a case of severe rickets. The condition was first noticed as a deformity of the chest when she was 5 months old. He excluded rickets because the condition was noticed so early; it was treated as rickets for years without effect; and, although the affection was apparently very severe, the head was quite normal, there were no curvatures of the long bones, and the lower extremities were hardly affected at all. He had never seen rickets of such severity in the upper limbs and trunk with the lower limbs so slightly involved and the head not at all affected. He called it achondroplasia because there certainly was great shortening of the bones of the forearms and some shortening of the arms. In estimating how far down the thighs the hands reached when the child was standing, one must take into account the fact that there was well-marked kyphosis and shortening of the whole trunk. The skiagram showed the shortening of the radius and ulna unmistakably. There was also enlargement of the corresponding epiphyses, and great laxity of the joints of the upper extremities, amounting to a possibility of 180° of rotation at the wrists. Some laxity of the joints, was, of course, often present in cases of rickets, but he believed that extreme laxity such as this never occurred in rickets, while it was quite characteristic of achondroplasia. He thought that on an examination of the upper limbs alone one would form the opinion that this case was one of achondroplasia; the skiagram also supported that diagnosis; but he admitted that the rest of the skeleton was not characteristic, so that it could not be regarded as a typical example of that condition.

Case of Circumscribed Serous Meningitis.

By LIONEL E. C. NORBURY, F.R.C.S.

F. H. S., MALE, aged 13 months. Full-term child; normal labour; breast-fed. Admitted to the Belgrave Hospital on August 6, 1912. History of convulsions fifteen days previously, followed by right facial paresis and tremor of right hand. No history of trauma; abscess of left side of neck incised two months before admission; discharge from right ear at the same time.

On admission: Well-nourished, but very pale child. Fine, regular tremor affecting right arm and hand, right leg, right side of mouth, and also the tongue and lower jaw. Right facial paresis; slight convergent strabismus; no otorrhœa. No sign of injury to skull. Knee-jerks present and equal, not exaggerated. Abdominal reflexes present. Pot. brom. and hydrarg. cum creta prescribed.

August 7: Tremor more marked, but ceases during sleep.

August 8: Condition unchanged. Lumbar puncture attempted, but failed to withdraw fluid.

August 9: Tremor as before. No changes seen in fundus oculi. Operation: Left Rolandic area mapped out on surface of scalp. Cranial tourniquet employed. Large scalp-flap turned down over left parietal bone; bone trephined over centre of Rolandic area. Pulsations of brain feeble. Very little bulging of dura. Opening in bone enlarged so as to freely expose Rolandic area. Dura incised crucially; pia arachnoid very swollen and œdematous, pulsation feeble. Child's condition became very critical; edges of dura partially approximated and scalp-wound closed rapidly.

August 10: General condition improved after administration of salt solution. Tremor as before. Tremor gradually disappeared, first from leg and then from arm and face. Wound healed by first intention. Cerebrospinal fluid collected under scalp-flap and was evacuated every few days (five times in all) until September 9, when it ceased.

On August 18 (nine days after operation) there was no sign of tremor.

On discharge from hospital, September 20, no tremor, but still slight facial weakness.

Patient seen on March 20, 1913: General condition very good. Very slight facial weakness. Moderate degree of bulging in region of craniectomy wound.

DISCUSSION.

Dr. REGINALD MILLER said he understood that the diagnosis put forward was only a tentative one. One of the difficulties in accepting the diagnosis was the fact that if it were a real tremor, an alternating and rhythmical contraction of antagonistic muscles, the tremor should have been homolateral, due probably to pressure on the frontal area—the tremor of frontal lobe disease; whereas here the tremor was contralateral to the site of operation. He was able to collect, a year or two ago, some cases of acute tremor in children, which he regarded as cases of encephalitis attacking the cerebello-rubrospinal tract. Quite a large number of these cases had now been collected. They were analogous to cases of acute poliomyelitis, only occurring elsewhere in the central nervous system. That diagnosis would fit in with the present case in various ways. The onset was in August, the month in which poliomyelitis and encephalitis occurred. Secondly, it would fit in with the symptom of tremor, with facial palsy, and slight sixth nerve weakness, all of which would place the lesion in the pons, affecting the rubrospinal tract (Monakow's bundle). It would also harmonize with the child's recovery. He had not seen cases which had been operated upon: and that brought one to the question of the presence of the œdema. Lumbar puncture in the present case failed, hence it was not known whether the œdema was circumscribed or generalized, as might occur in encephalitis.

Dr. JEWESBURY asked if the œdematous fluid removed from the case was examined bacteriologically or cytologically. Also, had a Wassermann examination been done?

Dr. LEONARD PARSONS said it seemed to be a typical case of polioencephalitis affecting the upper part of the pons and involving Monakow's bundle. He did not understand the association of a right VII with a right VI palsy if the lesion were cortical. The age of the patient, the suddenness of onset, and the complete recovery were in favour of polioencephalitis, as also the fact that the tremor stopped in sleep first, and then gradually got well, and finally ceased at other times. He asked whether the diagnosis put forward was made before the operation or afterwards.

Mr. NORBURY replied that apparently the case fitted in with Dr. Miller's diagnosis, but he could not understand why there should be so much œdema over the Rolandic area, and why the tremor should be localized to the right side. If it had been generalized œdema there would have been twitching in other parts of the body. No bacteriological investigation was made. As the child did not look syphilitic, and there was no history suggestive of that disease, no Wassermann test was carried out. The diagnosis was made after the operation.

Acute Poliomyelitis affecting the Muscles of both Hands.

By R. C. JEWESBURY, M.D.

E. H., AGED 12 years, had attack of "influenza" six months ago, very feverish for two days, then became very weak in right arm, and to a less extent in her back and right leg. Present condition shows marked wasting of muscles of both hands—also wasting of the muscles of the right arm and shoulder. The triceps is the muscle most affected in the arm.

DISCUSSION.

Dr. JEWESBURY said he had brought this case forward because, so far as he had been able to find out, a bilateral affection of the muscles of the hands in acute poliomyelitis was very uncommon. The mother had brought the child for weakness in her hands, and at first sight the case did not look like infantile paralysis, but on going into the history and seeing that the muscles of the right arm and shoulder were also affected, there was no doubt about the diagnosis. The wasting of the hand muscles had been much more marked and the condition was improving under treatment. He would like to hear whether other members had come across cases of infantile paralysis in which the hand muscles of both sides had been affected.

Dr. F. PARKES WEBER said it would be well, by the help of X-ray skiagrams, to exclude the presence of cervical ribs, which might cause symmetrical wasting of the intrinsic muscles of the hands. Cervical ribs which caused wasting in the hands were generally small ones, and could usually not be detected by ordinary palpation of the neck; the patient's acute illness (acute anterior poliomyelitis) might have given rise to a slight sinking of the shoulder-girdle on the spinal column, and consequently to the commencement of cervical rib symptoms. He, however, thought that in the present case the right explanation was that offered by Dr. Jewesbury, especially as the type of wasting of the thenar eminences was not that usual in cases of cervical rib; there was no marked ridge (due to *selective* atrophy in the thenar muscles) such as Kinnier Wilson had described as typical for cervical rib cases.[1]

Dr. JEWESBURY replied that the possibility of there being a cervical rib had not occurred to him, and he would have a skiagram taken.

Congenital Eversion of the Great Toes and other Abnormalities. Bony Process, on the Left Side of the Neck, liable to be mistaken for a Cervical Rib.

By F. PARKES WEBER, M.D.

THE patient is a healthy-looking female baby, M. T., aged 7½ months. Both great toes are everted and overlapped by the other toes, and the deformity thus produced at first sight reminds one of the eversion of the great toes not rarely met with in elderly persons, often associated with ulnar deflexion of the fingers, and often wrongly ascribed to the wearing of badly shaped boots or shoes (*see* illustration). Both the child's thumbs are turned inwards across the palms, and there is also an abnormal bony projection on the left side of the neck, which might be mistaken for a cervical rib. Careful examination, by palpation, ordinary inspection, and Röntgen rays (Dr. N. S. Finzi), shows that the

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[1] *See* Kinnier Wilson in the "Discussion on Cervical Ribs," *Proc. Roy. Soc. Med.* (Clin. Sect.), 1913, vi, p. 133.

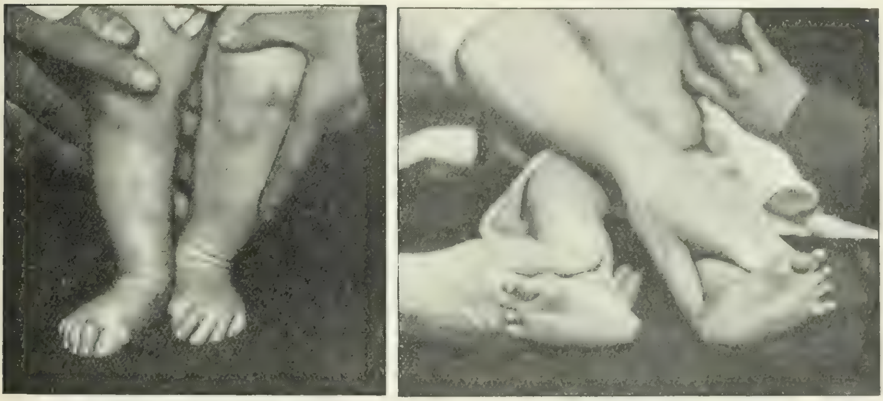

FIG. 1.

Photographs of the child's feet, showing position of the great toes.

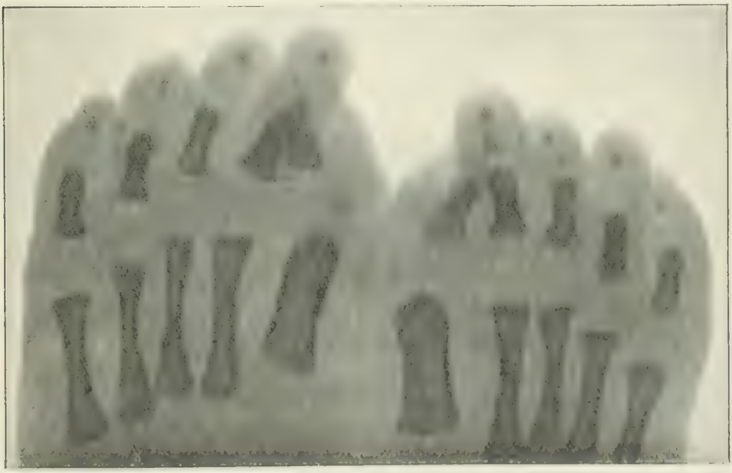

FIG. 2.

Skiagram of the child's feet (eversion of great toes).

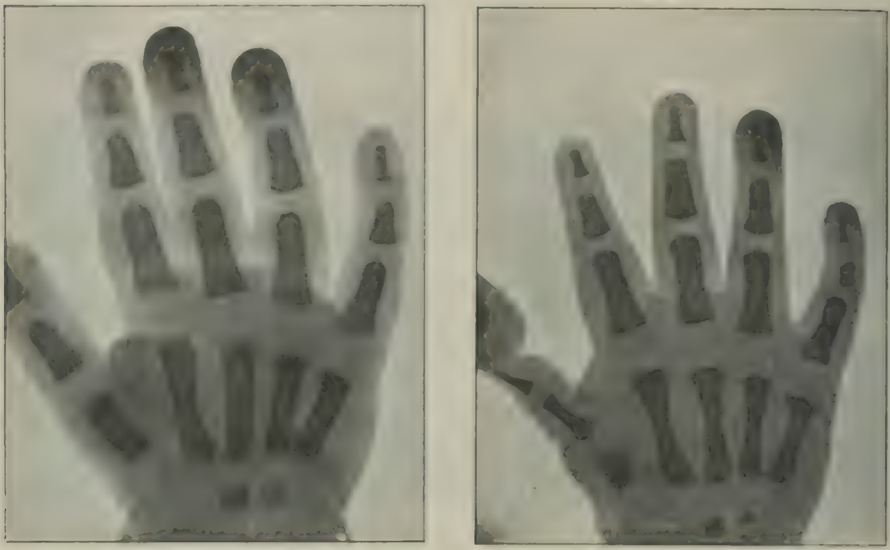

FIG. 3.

On the right is a skiagram of one of the patient's hands, showing the smallness of the metacarpal bone of the thumb; the thumb is being forcibly held in position. On the left is a skiagram of the normal hand of a male child, aged 10 months.

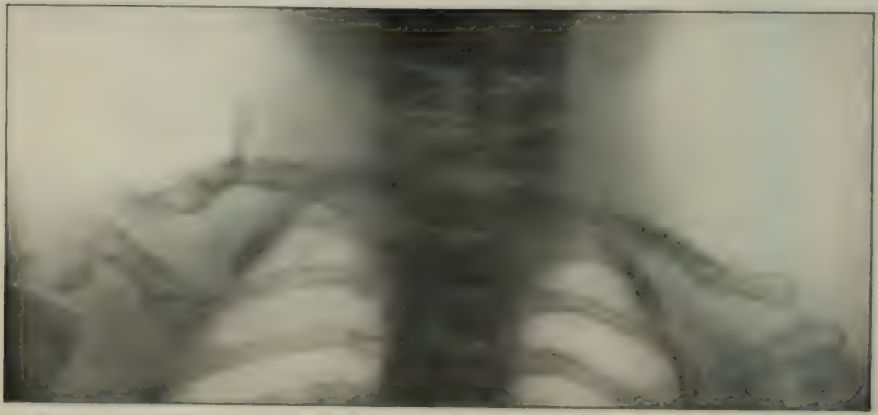

FIG. 4.

Skiagram of the child's cervical region from the back, showing the bony outgrowth from the left clavicle.

deformities in the feet and hands are quite symmetrical. The everted great toes (*see* skiagram) are abnormally short and altogether small, though the first metatarsal bones are of about average size. Both thumbs are abnormally short and slender, the shortness being especially well marked in the first metacarpal bones (*see* skiagram). There is "congenital ankylosis" (or more probably *non-development*) of the interphalangeal joint of each great toe and each thumb. The projection on the left side of the child's neck is due to a thin bony outgrowth (somewhat like the styloid process of the temporal bones at the base of the skull) from the back of the middle of the left clavicle; it projects about ½ in. upwards, and its upper end can be felt almost immediately below the skin (*see* skiagram). In regard to this abnormality in the neck (which is not an exaggeration of a so-called "deltoid tubercle" of the clavicle), Dr. Weber knows of no similar case, but he believes that similar congenital deformities in the great toes and thumbs, or in other toes and fingers, are occasionally met with. He is indebted to Mr. Alwyne Compton for sending the patient up to him.

The patient, who seems otherwise quite normal, is the only child of healthy parents, both young. There is no history of any similar malformation in any other members of either the mother's or the father's family, nor is there any definite history of "maternal impression."

Acute Tuberculosis (Hilum Phthisis). Specimen from a Boy aged 3 Years.

By J. T. Leon, M.D.

The boy had measles four months before the onset of the acute illness, but had not really been well since the measles. He was sent to my out-patient department from the Portsmouth Tuberculosis Dispensary as a case of probable empyema and there was complete dullness and absence of breath sounds over most of the right lung. The right side of the chest also appeared bulging as compared with left. The child was evidently extremely ill, temperature 101° F., and much emaciation. A needle was inserted into the right side of the chest, but no pus was found. The next day there was less dullness, and bronchial breathing over the lower part of back. In the hospital the child improved markedly for about a week, then rather rapidly got worse with pyrexia, 104° to 106° F., and died three weeks after admission. There were signs of consolidation also at the left apex. There was no generalized tuberculosis.

The CHAIRMAN said that, judging from his post-mortem experience, tuberculous disease of the lungs of this kind in young children was by no means uncommon. It was a special feature of tuberculous disease in early childhood for the mischief to start in the bronchial glands and spread from them into the lungs, the tubercles being more sparsely scattered as the periphery of the organs was approached.

Sections from a Case of Progressive Spinal Muscular Atrophy of Infants (Werdnig-Hoffmann Type).

By LEONARD PARSONS, M.D.

THE sections were obtained from a boy, aged 3 years, the third child in a family of six. He was normal until the age of 10 months. Wasting commenced after an attack of bronchitis. At 1 year he could move the arms well at the elbows, but movements at the shoulder-joints were poor; the legs could be well moved. Wasting in the lower limbs was first noticed in the upper part of the thigh. He was never able to sit up alone or to crawl—i.e., weakness was first noticed in the back muscles and in the muscles of the pelvic and shoulder-girdles. During the third year he gradually lost the power of holding his head erect, and his voice became feeble.

State: Marked wasting, weight 9 lb., intelligence good. Unable to sit up alone, and when propped up scoliosis to the left in the lower dorsal and lumbar regions develops. Back muscles wasted. The head flopped about. Arms: Marked wasting and flaccidity, shoulder-girdle muscles almost complete paralysis, supination impossible, wrist-drop. Movements of fingers possible but weak, some extension and flexion of the elbow also possible. Fingers long, thin, with characteristic overaction and slowness in performing movements. The hands, which are not wasted, assume a rather characteristic position. Legs: Very thin, adducted, internally rotated, and slightly flexed at the hip- and knee-joints. Right talipes equino-varus, left talipes equino-valgus. The only movements possible are weak flexion and extension at the ankle and of the toes; the paralysis is of the flaccid type. No wasting of the feet noticeable. Abdominal muscles very atrophic and ballooned. Intercostal muscles completely paralysed, and respiration is entirely diaphragmatic. Thorax shows a "trichterbrust" deformity. Knee-, ankle-, and arm-jerks absent, abdominal reflexes absent, organic reflexes normal. No sensory changes. Marked diminution in excitability to electric current with slight R.D. Cerebrospinal fluid normal.

The most striking feature about the case was the marked degree of flaccid palsy of the limbs.

Progress: Whilst under observation—six months—the paralysis of the lower limbs increased somewhat. Movements at the ankle-joints became impossible, and those of the toes became very feeble. Death was due to broncho-pneumonia.

At autopsy the wasting was found to be even greater than appeared before death, since a fair amount of subcutaneous fat persisted. The erector spinæ, glutei, latissimus dorsi, rhomboids, trapezius (particularly the lower half), sternomastoid, pectoralis major and minor were all very pale and atrophic. The sternal head of the sternomastoid and the upper part of the pectoralis major were less affected than the other muscles. The deltoid was wasted to an extreme degree; the biceps and coracobrachialis, although much wasted, yet contained a fair number of apparently healthy fibres. The psoas, iliacus, and quadriceps extensor were extremely pale and atrophic. The condition of the muscles was the same on both sides of the body. The fibres of the intercostal muscles and of the muscles of the anterior abdominal wall had almost entirely disappeared. The diaphragm alone of all the muscles examined was of a good colour and not wasted.

Sections of the muscles stained by Van Gieson and by the Marchi methods are shown. There is a considerable increase in fibrous tissue between the muscle-bundles. The muscle-fibres are altered in two ways, some are larger than normal and show a rounded contour on cross-section, but by far the greater number are smaller than normal (0·01 mm. and less), some indeed are so small as to be almost on the point of complete atrophy. These small fibres show an enormous increase in the number of nuclei, so that the section in parts is almost black with them. They appear to be formed by the splitting of the large and normal-sized fibres into a great number of small ones, and at the point of fission multiplication of the nuclei takes place. Striation even in the small fibres is, on the whole, very well preserved. The altered fibres are spread irregularly through the muscle and are not localized to any one spot. The muscle-spindles, even when situated in the middle of changed fibres, show a normal structure, the intrafusal fibres being 0·025 mm. in thickness.

The Marchi-stained sections show an increase in the amount of interstitial fat. A large number of the fibres show no fatty change, but some, and those chiefly of a normal size, show a certain amount of true fatty degeneration.

In Weigert-Pal sections of the cord the anterolateral columns stain rather more lightly than the posterior columns, but no definite tract is affected. The fibres of the anterior root are seen to be markedly diminished in number and less than those of the posterior root; they also take the stain more feebly. The sections shown stained by Nissl's method exhibit a most striking diminution in the number of the cells of the anterior horn. Full two-thirds of the cells normally present are missing, and those that persist are considerably smaller than normal. In the cervical and lumbar regions it is a rarity to find a normal-sized cell, and many appear to be on the point of complete disappearance. A slight degree of chromatolysis is not uncommon, and a few cells, particularly in the lumbar region, show complete chromatolysis. The atrophy and fibrosis of the anterior roots is well seen in the sections stained by Van Gieson's stain. The posterior root and their ganglia and the medulla were normal by all the methods used. Marchi sections of the spinal cord showed that no recent degeneration was present.

DISCUSSION.

Dr. PARSONS added that the condition seemed to be myelopathic. Dr. Batten[1] had shown from a series of post-mortems what was probably the true sequence of changes, viz.: that in the early stages the anterior horn cells were not diminished in number or size, showed little chromatolysis, and that the Marchi method showed degeneration in the anterior roots, both intra- and extra-medullary; whereas in older cases the cells diminished in size and number, chromatolysis increased, there was no Marchi change, and fibrosis of the anterior nerve-roots occurred.

Dr. JEWESBURY said that during the past year he was engaged with Dr. Topley in investigating histological changes in muscles from many cases of wasting diseases with the exception of neurological ones. They had had great difficulty in getting good sections of muscle, and they had found the celloidin method the most satisfactory one. He asked how the sections had been cut in this case. The marked increase in the nuclei referred to by Dr. Parsons was also seen in all the cases of wasted muscle which he and Dr. Topley had examined. In many cases the muscle-fibre nuclei were massed together, and in a few the nuclei were arranged around the periphery of the muscle-fibre, and in transverse section the appearance closely resembled that of a tubercular giant cell. In certain tuberculous muscles there were tubercular giant cells present and also muscle-fibres with this peripheral nuclear arrangement, and it was difficult to distinguish the one from the other—the main point of difference was the presence of a fine striation in the muscle-fibres. Dr. Parsons had said that fatty degeneration was present in the muscle-fibres of his case. He (Dr. Jewesbury) asked what stain was used to show this, and if it was often present in the muscles in nervous diseases. In non-neurological cases fatty degeneration of muscle was rare and only occurred in a few conditions, such as certain blood diseases and acute specific infections.

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[1] Batten, *Brain*, 1910-11, xxxiii, p. 43; *see* also joint paper with Dr. Gordon Holmes. Batten and Holmes, *Brain*, 1912, xxxv, p. 38.

The CHAIRMAN asked Dr. Parsons if he would state what was meant by the Werdnig-Hoffmann type of muscular atrophy.

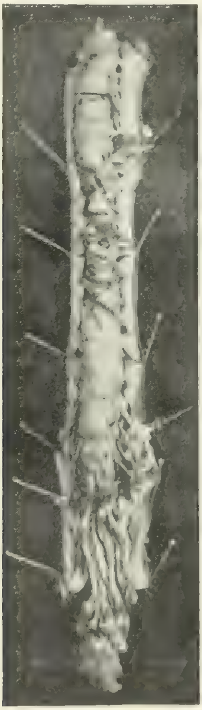
Dr. PARSONS replied that the Werdnig-Hoffmann type of atrophy was one which began around the shoulder and the pelvic girdle, and was progressive. The condition occured in families and always commenced *after* birth. It was a flaccid palsy of spinal origin which progressed towards the extremities of the limbs. It was first described by Werdnig, and later by Hoffmann. This child could only move his hands and elbows slightly, and could flex and extend the foot and toes. When once an advanced case had been seen he did not think there was difficulty in recognizing another, for there was something very characteristic about them. It was not much unlike amyotonia congenita, but the latter was present at birth, the intercostal and abdominal muscles were not paralysed, and the condition tended to improve. He had been much indebted to Dr. Jewesbury's paper on muscles, for from it he got a great amount of help in rightly interpreting his sections. They were cut in celloidin, which was the only way to get satisfactory results. The atrophic fibres were formed from normal fibres by fission. He did not find the giant cell formations which Dr. Jewesbury referred to. The Marchi stain did not show much fatty degeneration, but there was a peculiarly fine dusting all through the muscle-fibre, and this was present chiefly in the normal-sized fibre.

Case of Diffuse Sarcomatosis of the Brain and Spinal Cord.

By LEONARD PARSONS, M.D.

THE specimens illustrating this case were taken from a child, aged 2 years, who was an in-patient under Dr. Kauffmann at the Queen's Hospital, Birmingham. I am indebted to Dr. Kauffmann for the clinical characters of the case, which were as follows: The symptoms, which lasted four months, began with headache and attacks of violent screaming. Vision gradually failed and there was present double optic neuritis; at the end of three months the child was apparently quite blind. Walking was possible until a fortnight before death occurred, and a few days before death he was able to stand in his cot. For some time before death he would, in an attack of screaming, hold on to the cot sides tightly and retract his head; when this attack of screaming passed off he would talk quite naturally. Hearing, speech and intelligence were normal until after the onset of the final pressure symptoms, which commenced some ten days before death.

Post mortem: On opening the dura mater over the brain the convolutions were seen to be flattened, and on attempting to remove the brain from the skull there was found adherent to the petrous bones what appeared to be brain tissue, and which had to be cut through before the brain was free. On the basal surface of the brain and scattered indiscriminately over this surface of the cerebrum, pons, medulla, and cerebellum, were many small growths which were attached to the pia arachnoid. These growths looked very like brain tissue. Many of the cranial nerves received an investment of tumour growth. Thus the optic chiasma and the optic tracts received a small coating, and the stalk of the infundibulum was surrounded by growth. The third nerves received a much larger deposit, but the fourth nerves appeared to have escaped. The fifth cranial nerves received a large investment, which extended to and clothed the Gasserian ganglia, the latter structures being as large as filbert-nuts. The seventh and eighth nerves also received a large investment, and it was this growth which had to be cut from the surface of the temporal bone to enable the brain to be removed from the skull. The ninth, tenth and eleventh nerves also received a smaller deposit, but the growth markedly thickened the ganglia of the vagus and glosso-pharyngeal nerves. The growth appeared quickly to fade away after the nerves had left the interior of the skull. On dissecting the brain there was found a large mass in the cerebellum. This appeared to be arising in the middle lobe of the cerebellum, but had infiltrated the lateral lobes and had also extended into the fourth ventricle, which it had distended and completely filled. The iter was completely blocked by tumour substance and the ependyma of the lower part of the third ventricle was covered over with a film of the growth. This filmy growth extended through the foramen of Munro—but did not block it—and then faded away over the ependyma of the posterior horns of the lateral ventricles. On removing the lamellæ the theca appeared to be greatly distended. When the theca was opened it appeared perfectly normal, but, as the photograph shows, the cord was found to be entirely ensheathed by growth. This investment consisted chiefly in an infiltration of the pia; the arachnoid could be reflected from its surface in many places (*see* figure). The growth was most



Diffuse sarcomatosis of the brain and spinal cord.

abundant in the lumbar region, and in all regions was most marked on the posterior surface of the cord. It was least abundant in the upper dorsal region, where it was rather nodular. The spinal nerves received an investment of the growth and the posterior root ganglia were much enlarged from the same cause, those of the cauda equina being very large indeed. The growth, after a short course outside the spinal column, gradually faded away, but its presence along the radicles of the lumbar plexus is shown in one of the specimens. Along the intrathecal course of the nerves forming the cauda equina were seen many fusiform tumour growths.

Microscopically the growth is seen to be a sarcoma; the cells vary a little in size and shape, but most of them are small round cells. The growth is also found to have infiltrated, compressed, and distorted the cord.

This case closely resembles one described by Dr. Stanley Barnes,[1] whose explanation of the condition is probably the correct one. It is also almost identical with another case of the same disease recorded by Dr. Batten.[2] The primary growth in my case was in the cerebellum, and it owes its dissemination along the spinal cord and the various cranial and spinal nerves to the flow of an "infected" cerebrospinal fluid. The spread into the third and lateral ventricles is against the current of the cerebrospinal fluid, but this probably occurred by direct extension after the other growths had occurred and when the final hydrocephalic symptoms were arising.

Dr. REGINALD MILLER said that when he was at Great Ormond Street Children's Hospital he made the post-mortem examination on the case which Dr. Batten showed before the Neurological Section, and the photographs from brain, cord, and cauda equina could not be distinguished from those in the present case. It would be very interesting if someone could definitely work out the symptomatology of these cases.

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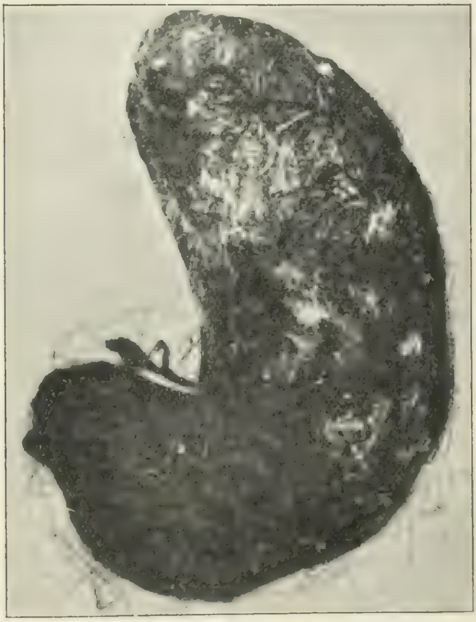
[1] Barnes, *Brain*, 1905, xxviii, p. 30.

[2] Batten, *Proc. Roy. Soc. Med.* (Neurol. Sect.), 1908-9, p. 12.

Hair-ball removed from the Stomach of a Child.

By Seymour Barling, M.S.

The patient, a female child, aged 7 years, was admitted to hospital for vomiting and severe pain in the upper abdomen of two days' duration. The child was rather small for her years, anæmic, but otherwise healthy and normal mentally, except that she was described by her parents as being rather sly. Since an attack of dysentery four and a half years ago she has been in the habit of chewing up pieces of string, tape, &c., and occasionally fragments of such things have been found in the motions. Her appetite was good up to ten days before the time she was taken ill, food being taken in normal quantity and without discomfort.



Hair-ball removed from the stomach.

On examination, a hard lump exactly resembling the outline of the stomach could be felt descending from beneath the left costal arch, and passing transversely across the epigastrium. The outline of the tumour together with the history led Lieutenant Ritchie, R.A.M.C., who first saw the case to make a correct diagnosis.

At operation, the specimen was easily removed through a longitudinal incision into the anterior wall of the stomach about 3 in. long, the dilatation of the organ allowing the mass to be turned on itself and delivered endwise through the opening in the stomach. The opening in the stomach was closed by a double layer of sutures, and the child made an uneventful recovery.

The specimen consists of a felted mass of hair and string, the former predominating; it presents an exact cast of the stomach showing the fundus, greater and lesser curvature, pyloric antrum and pyloric canal. When dried it weighs 4 oz., and measures 10 in. from the fundal to the pyloric end.

DISCUSSION.

Mr. BARLING added that he found that up to 1909 forty-two of these cases had been recorded, and they were collected in an American paper by Dr. Butterworth.[1] A history was traced in thirty-three, and seventeen of the patients had died of inanition or perforation. When there was a history of abdominal pain and vomiting, especially in a child, and the presence of a crescentic-shaped tumour in the epigastrium, the possibility of there being a hair-ball should be considered.

Mr. NORBURY said he saw, at St. Thomas's Hospital, a case in a girl, aged 18, with an enormous hair-ball in the stomach, with a kind of pig-tail in the duodenum, extending towards the duodeno-jejunal junction. She was not a lunatic, yet she strenuously denied having swallowed hair.

Dr. REGINALD MILLER said there was a similar case at St. Mary's Hospital recently, in which the woman hotly denied the possibility of having eaten her hair; but she was given away by the fact that she wore a wig. The diagnosis was suggested on the ground that the tumour did not resemble any ordinary pathological condition.

Dr. LANGMEAD asked if the colour of the hair in the hair-ball was the same as that on the child's head. In one case he knew of it was very different and gave rise to doubt as to whether the girl dyed her hair or whether the colour had been altered by the gastric juice.

Mr. BARLING replied that he had seen a case in which a tail of hair went down the duodenum, and there were cases in which some of the hair extended into the œsophagus. He believed hair went dark after a long residence in the stomach.

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[1] *Journ. Amer. Med. Assoc.*, Chicago, 1909, liii, pp. 617-24.

Section for the Study of Disease in Children.

April 25, 1913.

Mr. A. H. TUBBY, President of the Section, in the Chair.

Two Cases of Hereditary Syphilis treated by Intravenous Injections of Salvarsan and Neo-salvarsan.

By J. L. BUNCH, M.D.

Case I.—The child is now aged 2 years, and was shown at this Section[1] some eighteen months ago. Shortly after birth the child developed virulent symptoms of hereditary syphilis, and, when 8 weeks old, was given by me an intravenous injection of 0·03 grm. salvarsan ("606"). A fortnight after a second injection all the syphilitic symptoms had disappeared, and the child had increased 4 lb. in weight and has remained free from symptoms up to now.

Case II.—The child is now aged 3 months. When aged about 5 weeks he began to show symptoms of hereditary syphilis—rash, muco-purulent discharge from the nose, very offensive condylomata, &c. He was admitted to the Queen's Hospital for Children on April 11, 1913, and a skiagram taken of the right wrist. This showed marked disease of the lower end of the diaphysis of the radius, with cancellation and rarefaction of the bone, and periostitis spreading well up the shaft and also involving the ulna. There seems little doubt that these bone appearances are due to syphilitic disease. The Wassermann reaction was strongly positive. At 2 p.m. on April 12, 0·06 grm. neo-salvarsan was injected into the left external jugular. At 10 p.m. on the same day the temperature rose to 103° F., but fell again next day almost to normal. The pulse rose from 120 to 136 at night. The injection of neo-salvarsan was repeated on April 19, and the symptoms have all greatly diminished, with the exception of the bone changes. The mother still suckles the child at the hospital three times a day, in addition to artificial feeding.

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[1] *Proceedings*, 1912, v, p. 16.

DISCUSSION.

Dr. BELLINGHAM SMITH asked whether the cases had had salvarsan alone, or mercury in addition. Also, had Dr. Bunch treated many other cases in this way, and had he had any considerable mortality from this method of treatment?

Dr. BUNCH replied that no mercury was used in either of these cases. The second child received the second injection only a week ago. The first had cleared up so quickly that he did not think continuation with mercury was necessary, though a Wassermann test should be occasionally done in all such cases. Statistics showed that the results obtained by intravenous injection of salvarsan in children were extremely satisfactory, and he had not himself seen any unfavourable symptoms follow such injections.

Tuberculosis of Kidney.

By J. PORTER PARKINSON, M.D.

W. M., A BOY, aged 8 years, with a healthy family history, was admitted into a hospital for children two years ago, shortly after an attack of scarlet fever. He was sounded for stone in the bladder, and then the right kidney was explored from behind, but nothing abnormal detected. The urine, which had contained pus, cleared up and he was discharged.

On January 20, 1913, he was admitted into the wards of Queen's Hospital for Children for pain in the right side of the abdomen of two weeks' duration and wasting. He is thin but not emaciated, the right rectus is at times very rigid, but occasionally a hard tumour may be felt in the region of the right kidney, extending two fingers' breadths below the costal margin; it is sometimes tender. The urine is normal in amount, alkaline, contains a variable amount of pus, and about 0·1 per cent. albumin; no blood. Staphylococci and tubercle bacilli are found in it. X-ray report: Increased density in renal region; several shadows scattered over right loin, suggesting calcareous masses in an enlarged kidney or calcareous mesenteric gland. Two very dense shadows at lower part of right ureter suggesting calculi or calcareous matter. The left kidney presented nothing abnormal, and there are no bladder symptoms. The heart and lungs are normal. Temperature as a rule normal, but with occasional rises to 102° or 103° F., apparently due to retention of pus in the pelvis.

The boy's condition has improved in hospital, and the local tenderness and rigidity have disappeared, so it is easy to palpate the diseased kidney. It is proposed to remove the diseased kidney shortly.

DISCUSSION.

Dr. PORTER PARKINSON added that he was inclined to let the case alone as the kidney had shrunk so much under medical treatment, and the boy had gained weight. He asked for opinions as to operative procedure. There was no evidence of any other deposit in the body.

Mr. WHITELOCKE said he would advise an operation on a tuberculous kidney in that state; not excision, but cutting on to the kidney, exploring, and perhaps draining.

Mr. PHILIP TURNER expressed his interest in the way these calcareous masses were shown by the X-rays. He recalled one case in which the patient was supposed to have a stone in the ureter. X-rays revealed a shadow suggesting it, but no stone could be found at operation, the trouble being tuberculous. From the particulars available in Dr. Parkinson's case, he would certainly favour operation.

Dr. MORLEY FLETCHER said he would advise operation in this case if the other kidney were healthy. The method of separation should be employed, to investigate the condition of the urine from the other kidney.

Dr. PARKINSON replied that his inclination to leave the case alone was due to the high mortality from nephrectomy; and the child was at present healthy still. He realized that a source of potential danger was being left behind.

Dr. J. T. LEON suggested a preliminary course of tuberculin. He had at present under care a boy suffering from a similar condition, and that treatment improved him considerably. If the disease could be localized, an operation such as drainage might do good. He would think there was in this case more tubercle than met the eye. The doughy consistence of the abdomen led him to think there might be tubercular peritonitis in addition.

The PRESIDENT (Mr. A. H. Tubby) admitted that the proposed operation was a serious one, but not so serious as that for malignant disease of the kidney. His impression was that where it was possible to make sure that the tuberculosis was limited to one kidney, the mortality was not heavy—about 20 per cent. The danger arose in the cases where both kidneys were involved. In this case there seemed no evidence as to the condition of the other kidney, and he agreed with Dr. Morley Fletcher that this was the main fact concerning the advisability of operation.

Mr. LOCKHART MUMMERY remarked that the whole question of the mortality of the operation in a fairly healthy boy who had his kidney removed turned upon the condition of the bladder. In this case there did not appear to be any cystitis at present, and he would not be in a hurry to remove that kidney, though he would do so if there were signs of cystitis.

A Successful Case of Cerebral Decompression for Convulsions, Jacksonian in Type, in a Child, aged 4 Years.

By R. H. Anglin Whitelocke, F.R.C.S.

J. E. W., aged 4 years, was admitted at 9.15 p.m. into the Radcliffe Infirmary, Oxford, on February 2 of this year, with the history that he had been in perfect health until that morning, when he complained of slight earache. He did not go to school, but in the early afternoon went for a walk. At 5.30 he fell asleep, and at 7.30 awoke feeling sick, complained of headache, and soon vomited. Soon after he vomited his face began to twitch and he began to have convulsions which were confined to the right side of his body. He was at the time quite conscious but spoke indistinctly. As the spasms continued his mother put him into a warm bath and sent for medical assistance. As the fits began to increase in duration and severity the family physician sent him to hospital. On his arrival I happened to be going round the wards with a class of students and took the opportunity of demonstrating to them the symptoms and varying phases of the case. The spasms began in the face and rapidly spread to the thumb, wrist, forearm and arm, and then downwards until the lower limb had been completely involved. The child was then hardly conscious. Between the seizures there was but a momentary period of muscular relaxation, and fit followed upon fit with marked regularity. There was no incontinence of urine, and no biting of the tongue. As the condition was steadily becoming worse, and as the fits were entirely localized to the one side, I determined to perform an exploratory operation with the double object either of removing, if possible, some localized focus of cortical irritation, or of diminishing intracranial pressure in the event of there being some more deeply situated cause, such as a neoplasm or collection of fluid.

As the symptoms were so clearly localized and defined the situation chosen for the opening was the neighbourhood of the Rolandic area. As soon as the surgical preparations had been made, and with chloroform anæsthesia, a semicircular flap of skin and pericranium with its base below was turned downwards over the side of the head; the bone on exposure presented nothing unusual, though the percussion note seemed a little duller in the region of the parietal eminence. With the aid of a 1¼-in. circular trephine a disk was removed, whereupon the dura mater bulged into the space owing to the presence of increased intracranial pressure. A flap of dura mater was made and drawn aside. The blood-vessels on the surface of the exposed convolutions were somewhat engorged, but there existed no definite lepto-meningitis or gross lesion; and to all intents and purposes the exposed portion of the brain was healthy. A fine exploring cannula was then passed into the cavity of the left ventricle and withdrew a few drops of clear cerebrospinal fluid. Suspecting the presence of a deeply placed neoplasm or perhaps a tuberculous focus, the bony disk was *not* replaced, whilst the dura mater was only partially sutured. The skin flap was restored and held in position by interrupted silkworm sutures to allow of leakage, for no other means of drainage was considered advisable. The fits ceased from the moment the dura mater was incised. The wound healed perfectly, and the child made a complete and uneventful recovery. He is now in perfect health and spirits. The interesting features for consideration are :—

(1) The still unascertained cause of illness, no lesion having been found.

(2) The one-sided limitations of the spasms, with at first no loss of consciousness, no incontinence of urine, and no biting of the tongue.

(3) The complete cessation of the fits so soon as the intracranial pressure was relieved by operation.

(4) The complete and rapid restoration to health after such a sudden and severe nerve-storm.

The opening in the skull may still be felt, and through it the brain pulsating. A rude cap is worn more as a placebo than a protection. It is advisable not to close the bony aperture for the present, lest there should be a neoplasm ; it can always be closed later, if required, by bone-grafting.

DISCUSSION.

Dr. C. O. HAWTHORNE said he regarded the case as a very important one, and asked whether it was shown merely as an interesting experience, or whether it was presented as an illustration of the treatment which ought to be adopted in cases of this order. Was it the proposal that a healthy child taken with unilateral convulsive seizures ought to be trephined? There might be something to be said in favour of this, but he regarded it as a serious proposition. A certain number of such cases got well without the aid of surgery, and others were benefited by merely tapping the cerebrospinal fluid. On the other hand, did the case belong to the group in which an attack of unilateral convulsions is followed by hemiplegia? In other words, was the case one of encephalitis, and had Mr. Whitelocke's energetic treatment saved the child from being hemiplegic? These were the questions which seemed to be raised by this very interesting case.

Mr. WHITELOCKE said the result was so dramatic that he did not regret the procedure he had adopted. The child was suddenly attacked, and when seen was rapidly becoming exhausted and unconscious. He therefore, assuming some gross lesion to be present, operated at once. Trephining was not nowadays a serious procedure. As soon as the dura mater was incised the fits ceased. He was not aware of what Dr. Hawthorne said, that a good many such cases were followed by hemiplegia. If the condition was inflammatory in this case, his treatment had been fortunate.

Enlargement of the Thyroid Gland (Goitre) in a Family of Five Children, Four Boys and a Girl.

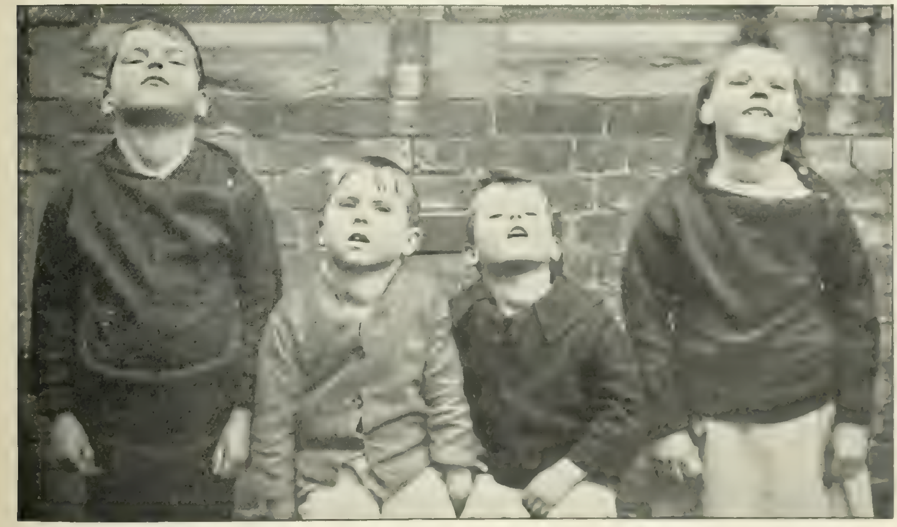
By R. H. ANGLIN WHITELOCKE, F.R.C.S.

THE respective ages are: Hurrell 5, George 7, Gwendolen 9, Sidney 10½, and Henry 12 years. The family history is that the four elder children were born in Surrey, where they lived on high ground, the youngest at Eynsham, near Oxford, a low-lying village near to the Thames. The parents are healthy and intelligent people, the father a builder's labourer earning only 12s. a week.

George is said to have been born with an "enlargement in his throat," so that for the first four weeks of his existence his life was despaired of, and the monthly nurse almost daily informed his mother that he could not live. For some months later he was liable on the least excitement to suffer from "fits of suffocation." This went on till he "cut his eye-teeth." Attention was first drawn to the condition of Hurrell and Gwendolen by the school inspecting doctor eighteen months ago. Sidney's neck began to enlarge when he was aged 10 years; Henry's, the eldest, only became apparent during the last six months, and not before he was approaching the age of 12 years. The eldest boy is said to have been very delicate until he had an attack of typhoid fever three years ago; since then he has grown considerably in size and strength. With the exception of an attack of scarlet fever from which the girl suffered recently, they have all been remarkably free from illness.

For the last eighteen months I have had the four youngest children continuously under my observation and treatment as out-patients at the Radcliffe Infirmary. During this time there has been very little change excepting that George's neck, the congenital case, has grown a little larger, and Gwendolen's become less. The eldest boy has only lately come under treatment, for his enlargement has been very gradual and somewhat slow, and is of only recent development.

As to ætiology nothing very definite can be established beyond the fact that George is evidently a congenital case, and that the two eldest did not show symptoms until after the age of 10 years.



Enlargement of the thyroid gland in a family. Photograph of the four younger children.

The conditions of climate do not help, for in the younger three the disease began while they were living in the uplands of Surrey, whilst the two elder children have lived in the Thames valley during the periods of onset and development of the disease.

The children, as may be noticed, and as is shown in the photograph, are well nourished and well cared for, in spite of the father's weekly wage being only twelve shillings. Whenever the mother has been questioned as to their supply of food she has remarked in a cheerful way that sometimes kind friends help them, and that if at any time the children require a little extra nourishment her husband and she go without.

The pathological condition is, I venture to think, one of simple parenchymatous enlargement. The gland shows equable and general increase, both lobes as well as the isthmus being involved.

Treatment has consisted in an increased weekly pecuniary allowance, the administration of cod-liver oil, iodine preparations, and occasionally iron. The home surroundings have been improved as regards warming, increased light, and general hygiene. This regime, steadily enforced for over a year, seems to have arrested the glandular increase in most cases. The congenital case still remains the largest, as may be readily seen by reference to the photograph.

Amongst the interesting points are to be noted :—

(1) The existence of the disease in *all* the children of the family, the parents being at the present time wholly exempt.

(2) That the history points clearly and definitely to the fact that one case at least is congenital.

(3) That the thyroid became enlarged in all the children whether they lived on bracing and high ground or in the relaxing atmosphere of a low-lying and somewhat damp watershed.

(4) The excellent general health and spirits of the children in spite of the poor pecuniary circumstances of their parents.

We know, speaking generally, so little of the ætiology of thyroid enlargements and of the factors which enter into their increase and diminution that it seems not unbefitting that a few correlated facts should be noted and presented to a Society such as this, concerned with the study of disease in children and young persons.

Goitre is a comparatively rare affection in the very young.

DISCUSSION.

Mr. WHITELOCKE added that when he sent the note he was not aware that the father had had a "big neck"; he did not know whether he had had a thyroid condition which had become cured; at the present time he had not a bronchocele.

Dr. LANGMEAD said a series of cases in the same family might be explained on one of two hypotheses: (1) That all the children were subject to some unusual intoxication, or (2) that their thyroids did not have the same protective action as was common to normal thyroid. But as in this series four of the children contracted goitre while living under different climatic conditions in a different county from the fifth, the first hypothesis could probably be excluded. It was probable that the protection exercised by the thyroid gland was less than normal, and consequently the glands had hypertrophied in order to do the necessary work. The second hypothesis was supported by the appearance of the mother, who was well below the average stature, and although only slightly cretinoid in appearance, it was well known that certain people of diminutive stature improved on thyroid treatment. He favoured the view of congenital deficiency in the protective power of the thyroid gland.

The PRESIDENT said these cases were very useful, as they rather tended to upset the theory relating to the absence of iodine in the water supply. Some of the children in Mr. Whitelocke's series were born in Surrey, and one in the Thames Valley, where the water was such as to make one doubt the correctness of the idea that the problem of thyroidism could be solved by pointing to the absence or otherwise of a minute quantity of iodine in the water.

Dr. WILLIAM EWART asked whether the blood-pressure had been ascertained in these five cases of goitre.

Mr. WHITELOCKE replied that he did not ascertain the blood-pressure, but would now do so. He thanked the President for the points he raised in connexion with the water supply; he did not know that the Sutton water supply had been interfered with. So far as he knew, the water supply of the village from which these children came was quite good; it was the Thames water which he himself drank. The theory advanced by Dr. Langmead no doubt suited the cases admirably, so far as the mother was concerned, but he would have liked members to have seen the father, who happens to be a big, burly man.

Operative Myxœdema—Cachexia Strumipriva.

By H. MORLEY FLETCHER, M.D.

A CASE in a girl, aged 13 years. In October, 1907, at the age of 7 years, she was admitted to St. Bartholomew's Hospital with a small, elastic, freely movable swelling, the size of a marble, medianly situated in the neck at the level of the thyroid cartilage, and apparently attached to the thyroid gland. The swelling was first noticed two years previously, and had been gradually increasing in size. It was removed without difficulty, and was found microscopically to consist of thyroid gland tissue. She was discharged, but was readmitted on November 22 in a state of cachexia strumipriva. Three weeks after the operation she became drowsy, and swelling of the face and hands with puffiness under the eyes was observed. On admission the temperature was subnormal; the urine contained no albumin. She was treated with thyroid extract, and discharged on December 11 in a considerably improved condition. Treatment was continued intermittently in the Casualty Department until December, 1908, when she came to the Children's Department. She had taken no medicine for at least six weeks, and presented the typical appearance of myxœdema. She was very lethargic, the skin was dry and rough, the hair over the temples very thin. The urine was natural. Pulse 66 per minute; temperature 98° F. Since then she has been taking thyroid extract continuously, but in spite of this has not progressed satisfactorily. The mental condition is good. At the age of 11 years she was in Standard V, but was put back to Standard IV. Bodily growth is backward; height, 4 ft. 3 in. She has never menstruated. She still presents a puffy, pallid appearance, suggestive of myxœdema or chronic nephritis, though the urine has been always found to be normal. At times she becomes lethargic, though as a rule she is bright and active-minded. The addition of suprarenal extract to the thyroid extract has made no apparent difference.

DISCUSSION.

Dr. MORLEY FLETCHER added that it was the first case of operative myxœdema occurring in a child that he had seen. The portion of tissue removed appeared to have been the only functional portion of the thyroid gland, and represented either the isthmus of the gland or the median lobe, or it might have been thyroid tissue in the thyro-glossal duct. The tissue was increasing in size to meet her need for thyroid secretion, and might be regarded as a compensatory hypertrophy. She was readmitted three weeks later with a very typical appearance of myxœdema. Treatment with thyroid extract at first produced rapid improvement, and she was transferred to the Casualty Department to receive that treatment regularly, but for nearly two years treatment had been neglected. From her facies one would say at present that she had either chronic nephritis or myxœdema; he laid a good deal of stress on the thickening of the upper eyelids. An important point was that when a child had been deprived of thyroid secretion for a considerable period the mental condition might subsequently become normal, but the physical condition never seemed to resume its normal character.

Dr. BELLINGHAM SMITH wished to corroborate what Dr. Morley Fletcher said about the difficulty of improving the bodily aspect of these cases, while the mental state could be fairly well restored. For the last fifteen months he had had under his care a little girl, aged 11 years, who developed symptoms of thyroid insufficiency at 5 or 6 years of age. For the whole of this period she had been under treatment with thyroid gland extract. The child's mental condition was moderately good; she was in Standard III of the ordinary elementary school, and apparently did her work well. But despite treatment, the curious conformation of the body, the facial appearance, the dry skin, &c., still existed. He compared her with some five ordinary cretin babies which had been brought for treatment at different times, and the condition was markedly different in the two. Three of the five babies were absolutely normal now as regards intelligence. The other two presented a degree of mental deficiency which he thought was due to treatment being commenced rather late. On the other hand, the bodily condition of all was perfect. He would like to hear of some treatment which would improve the bodily as well as the mental conditions of these children.

Mr. MIDELTON asked whether Dr. Morley Fletcher had tried a preparation of the internal secretion of the liver, as recommended by Dr. Leonard Williams for such cases.

Dr. MORLEY FLETCHER replied that he had not tried extract of liver, and would be glad to learn further particulars. In answer to Dr. Bellingham Smith, he thought a distinction should be drawn between cretinism in children and juvenile myxœdema; in the former a child is born with a defective thyroid, and in such cases the condition manifests itself in the first few months of life. In juvenile myxœdema the child develops normally for two or three years and then development becomes arrested and the condition of myxœdema makes its appearance. The two conditions can be distinguished from one another. He had tried, in the case of this child, nearly every preparation of thyroid on the market, and in various doses, the child having had as much as 4 gr. three times a day, and as little as ½ gr. each day. When the dose was reduced, she became stupid and sleepy. Except for the suprarenal extract he had not tried any other internal secretory preparation. At the convalescent home she was put on large doses of thyroid extract, and it was reported that she became uncontrollable and almost maniacal at times.

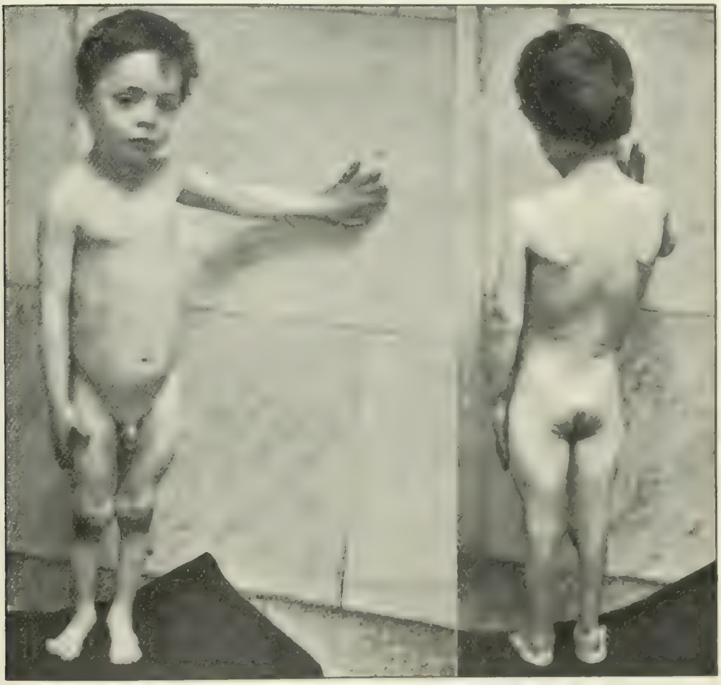
Distal Myopathy.

By E. A. Cockayne, M.D.

The patient, N., is a boy, aged 6 years. The parents are healthy. The mother had five brothers and five sisters who reached adult life, and were normal. The father has one brother and one sister, both normal. There is no history of any similar condition in the family. The patient has two brothers, aged 14 and 8 years, and one sister aged 10 years, all well-grown and healthy. The mother states that the patient has always been undersized, and has suffered from birth with incontinence of urine and fæces. At 3 years of age she noticed that he had considerable wasting of the muscles below the knee, and that his buttocks were small. She is doubtful if the wasting has progressed since it was first noticed. The boy seems to fall over rather easily.

The boy is small for his age, 37¼ in. in height, but is of normal intelligence. There is slight ptosis on the left side—a condition which is also present in his brother, aged 14. The face, arm, and trunk muscles appear natural. There is very marked symmetrical wasting of the glutei and of the leg muscles below the knees. The thigh muscles are well developed and appear to be hypertrophied. There is pes cavus on both sides. The child can rise from a lying or kneeling position without difficulty, walks upstairs without using his hands to help him, and can carry out all the normal movements of the legs and feet. There is sometimes some cyanosis of the legs below the knees. No fibrillation has been observed. Sensation to touch, pain, heat and cold, over both arms and legs is natural and muscular sense is preserved. The knee-jerks are brisk, but neither ankle-jerks nor plantar response have been obtained. Dr. Lyster has kindly examined the electrical reactions on two occasions, and reported that there is no alteration either qualitative or quantitative even in the wasted muscles. He stated that a skiagram showed a narrow pelvis, and changes in the epiphyses of the long bones characteristic of rickets, but that there is no spina bifida present. The boy has no alteration of gait or ataxia, and complains of no pain, no feeling of cold, nor any other subjective symptoms. But for the implication of the glutei muscles the distribution of the wasting resembles that seen in an early case of peroneal atrophy of the Charcot-Marie-Tooth type.

Soca has described a case of this disease in which the arms were unaffected, and in which the glutei, though normal in appearance, showed well-marked R.D. Against this diagnosis is the fact that there is no alteration of sensation, and no change in the electrical reactions. Though it does not fall into any definite group of the myopathies, I have preferred to call it a distal myopathy rather than a myelopathy, but there is the third possibility that it is a congenital deformity.



Distal myopathy.

The PRESIDENT remarked that he was seeing a number of cases of myopathy, and he suspected there were several groups other than those which had been named and described.

Congenital Absence of Abdominal Muscles and other Defects.

By E. Bellingham Smith, M.D.

Baby, F. V., aged 2 months. This infant is the first child of healthy parents; he is brought to hospital because "there is something wrong with his stomach." On examination the abdomen is flattened from before backwards, while there is a distinct bulge in either loin, which is markedly accentuated whenever the infant screams or strains. The skin is loose, transversely wrinkled in places as if scarred, and the normal umbilicus is absent and is replaced by a longitudinal groove. Centrally there is a wide, flattened median band, stretching from the ensiform to the pubis, which represents the rectus sheath. If the abdomen is palpated on either side of this central band the coils of intestine can be readily felt, apparently only covered by skin, fascia and peritoneum. Both kidneys are readily felt, and the lobulated nature of the organ can be readily determined. The bladder is distinctly palpable, and is situated well above the pubis. The central sheath itself can be easily picked up between finger and thumb, and suggests to the feel merely a band of fibrous tissue without any intervening muscular tissue. The whole of the abdominal wall fails to react to electrical stimulation. In addition to this apparent lack of muscular tissue in the abdominal wall, the infant also presents other congenital defects. The right leg is kept slightly flexed at the hip- and knee-joints, and is said to be moved but very little. There is a moderate degree of genu valgum and talipes calcaneo-valgus present on both sides; the latter condition is more marked on the right side than on the left. Over the præcordial area there is a faint systolic murmur, which is loudest in the region of the impulse. In all other respects the infant appears to be healthy.

DISCUSSION.

Dr. Bellingham Smith added that while it was difficult to be absolutely certain that the abdominal walls contained no vestige of muscular tissue, he had formed the diagnosis on the following grounds: (1) The extreme tenuity of the abdominal walls, which allowed all the contents of the abdomen to be freely palpated; (2) the entire absence of any form of electrical response;

(3) the similarity of the case to four or five others, which had previously been reported. It differed only from these in the fact that no marked abnormality, other than the high situation of the bladder, could be detected in the urinary organs.

Dr. LANGMEAD said the case belonged to a definite group of cases as Dr. Bellingham Smith had said. A paper was published in 1905[1] by Dr. A. E. Garrod and Mr. Ll. Wynne Davies, recording more cases than Dr. Bellingham Smith had just spoken of. A paper by Stumm included seven cases, that which he described himself making eight. Dr. Garrod described another, and mentioned a case under the care of Dr. Batten, bringing the number up to ten. He (Dr. Langmead) had seen one since. They were all very much the same. There were: (1) An apparent absence of abdominal muscles to palpation; (2) in many cases deformity of the chest, most often a protrusion forward; (3) a thickened and abdominally placed bladder, attached above to the umbilicus by a short and thickened urachus; (4) often abnormalities in the ureters and kidneys (in the case described by Dr. Garrod and Mr. Davies there was one kidney very small, and in others the ureters had been large and distended, even as large as or larger than the child's own small intestine; (5) a curious linear vertical marking of the abdominal wall; (6) a slit-like umbilicus; (7) talipes. It had been suggested that in these cases there was some abnormality in the spinal cord. Dr. Batten examined one case, including in his examination enumeration of the cornual cells, and found no evidence that the condition was primarily a nervous one affecting the cord. A certain amount of abdominal muscle was probably present, unstriped rather than striped.

Two Cases of Ranula.

By P. MAYNARD HEATH, M.S.

Case I.—Boy, aged 10 years. Swelling under right side of tongue noticed four months ago. Was then soft, but has got much harder lately. It gives rise to no symptoms, but is tender on pressure. The tonsils are very large, and there are enlarged glands at both angles of the jaw.

Case II.—Girl, aged 3 years. A swelling under the tongue has been noticed for six weeks. It varies in size from time to time. The swelling is soft, bluish in colour, and lies symmetrically on each side of the frænum of the tongue. The child is a mouth-breather. There are enlarged glands in each side of the neck.

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[1] *Med. Chir. Trans.*, 1905, lxxxviii, pp. 363-82.

Pathological Specimen: Diphtheria of the Œsophagus.

By J. D. Rolleston, M.D.

Boy, aged 6 years. Admitted to hospital on March 22, on fifth day of disease. Death occurred within one hour of admission.

Condition on admission: Profound toxæmia, pulse imperceptible; temperature 99·4° F. Palatal and faucial œdema, membrane on tonsils, pillars and palate, considerable neck swelling, oral fœtor and profuse nasal discharge. Slight stridor, croupy cough, and recession.

Specimen shows normal condition of upper part of œsophagus. The lower $1\frac{3}{4}$ in. show longitudinal areas of congestion and submucous hæmorrhages. A small piece of membrane is visible at the upper part of this congested area, and there are larger patches of membrane present at the cardiac end. Membrane was also present in the nasopharynx, larynx and trachea. Stomach normal. Pure cultures of Klebs-Loeffler bacilli were obtained from lesions in throat and œsophagus.

The specimen illustrates an early stage of the invasion of the œsophagus by diphtheria, and should be compared with that which I showed before this Section in October, 1911.[1] In both cases the lesions were localized in the lower end of the œsophagus, and in both there were multiple diphtheritic lesions elsewhere associated with profound toxæmia. The first case, however, was in a much later stage, as the membrane in the œsophagus had entirely separated, leaving a necrotic area.

Probably diphtheria of the œsophagus is not so rare as is generally supposed, although there are details of only twenty-four cases on record, including these two of my own. Fourteen of these occurred in the pre-antitoxin era, and in the rest antitoxin was either not given at all or too late. Mallory[2] found definite membranes in the œsophagus in twelve cases, or 4·7 per cent., among 251 necropsies of diphtheria cases. During the last eighteen months—i.e., since I showed my first specimen—I have examined the œsophagus in ten cases of diphtheria which died in the acute stage, and in two cases, in which the present is included, I have found œsophageal membrane present. The other case was that of a girl, aged 6½ years, admitted to hospital with very severe faucial, nasal and laryngeal diphtheria on the ninth day of the disease. Death occurred within twenty-four hours, in spite of large doses of antitoxin, and post mortem the lesions in the fauces, nasopharynx, larynx and œsophagus were found to be almost identical with those seen in the present case.

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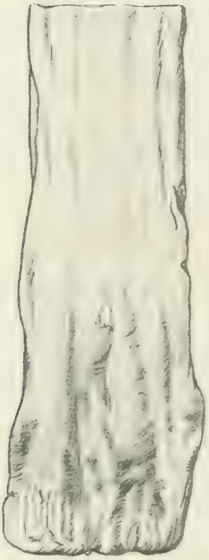
[1] *Proc. Roy. Soc. Med.*, 1911, v (Child. Sect.), p. 16.

[2] "Bacteriology of Diphtheria," ed. by G. F. H. Nuttall and G. S. Graham Smith, Cambridge, 1908, p. 97.

In connexion with unusual situations of diphtheria in the alimentary tract, it should be noted that while diphtheria of the œsophagus and stomach[1] is usually fatal, four cases of diphtheria of the intestines have recently been recorded, all of which recovered, while only two were followed by paralysis.[2] A few cases of diphtheria of the œsophagus have ended in recovery, but with the development of a stricture. I referred to a certain number of these cases in my last paper, and since then another case has been published by Réthi.[3]



Diphtheria of the œsophagus.

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[1] Tylecote, F. E., *Brit. Journ. Child. Dis.*, 1912, x, p. 211.

[2] *Lancet*, 1913, i, p. 129.

[3] Réthi, A., *Berl. klin. Woch.*, 1912, xlix, p. 2405.

Two Successful Cases of Operation for Strangulated Inguinal Hernia in Female Infants, of the Ages of 22 and 17 Days.

By R. H. Anglin Whitelocke, F.R.C.S.

Case I.—In September, 1912, a female infant, aged 22 days, was admitted under my care into the Radcliffe Infirmary. The mother stated that the baby was entirely breast-fed, and that for the last five days nothing had passed through her bowels. The family attendant, without seeing the child, advised the mother to give it castor-oil; this, however, was invariably vomited as soon as swallowed. The infant had vomited for three days, had moaned almost continuously as if in great pain, and had slept hardly at all during the whole of this time. When examined, a firm, tense swelling was found in the right inguinal region the size of a small hen's egg. Both legs were tightly drawn up and the child was blanched and obviously very ill. A few drops of chloroform were administered, and after the necessary surgical preparations had been made, herniotomy was performed. In the hernial sac were found the right ovary and Fallopian tube, together with a small loop of the small intestine. This last was severely strangulated, was purple black, but shiny. The circulation was gradually restored by the aid of a warm, saline wash, and the colour soon improved. The contents of the sac were returned to the abdomen, the neck of the sac ligatured off while the sac itself was excised and the operation rapidly completed. Recovery was complete and quick and the babe is to-day well grown, fat, and shows no after-effects of the operation.

Case II.—On March 6 of this year I was called into the country to see the female infant of a lady who had been confined only seventeen days previously, and who was still in child-bed. The family physician informed me that the child had been screaming much since birth, that it had vomited all its food for nearly three days, that its bowels had not moved even with the use of glycerine suppositories for four days, and that it refused the breast. In the right groin was a swelling, tense, tender and tympanitic, the size of a small tangerine orange. I decided to operate at once, realizing that we had to deal with a strangulated inguinal hernia in a very young and feeble subject. After the necessary preparations had been hurriedly made, and with the help of chloroform, I cut down upon and opened the sac. The contents consisted of a swollen and very œdematous cæcum with a long discoloured appendix vermiformis. The appendix was exceptionally large, I thought, for such a young infant, and owing to the presence of a very short mesentery it had become doubled upon itself so as to produce a condition of almost complete strangulation in the distal half. This organ was quickly removed, and after considerable difficulty at the internal ring, I succeeded in returning the swollen cæcum. The internal abdominal ring admitted the tips of two fingers. The sac was ligatured, and the wound closed and dressed with collodion. In a very short time after the little one took the breast, passed water, and fell asleep. The bowels were moved naturally a few hours later, and the wound healed without any trouble. I have recently heard that she is now thriving and is in all respects well.

Several points of interest to the surgeon may be gathered from the experience of these cases, and amongst them may be narrated :—

(1) The early ages at which strangulation occurred, and with apparently no definite cause.

(2) The unusual nature of the hernial contents, in the one case an ovary and tube as well as small intestine; in the other, an unduly mobile cæcum with large appendix measuring 3½ in.

(3) The successful issue in each case even after the obstruction and symptoms of strangulation had lasted for over three days.

(4) The absence of post-operative shock after a general anæsthetic and herniotomy, and in the younger infant after appendicectomy in addition.

Herniotomy for strangulation in such young infants must be exceptional, and a successful appendicectomy at the age of 17 days is certainly so.

A Case simulating Meningitis, in which the Symptoms were caused by the Escape of Threadworms into the Peritoneal Cavity through a perforated Appendix Vermiformis.

By R. H. Anglin Whitelocke, F.R.C.S.

The patient, a girl, aged 5½ years, was admitted into the Oxford County Hospital in May, 1910. She was sent in with the diagnosis of tuberculous meningitis, seeing that a year previously an enlarged gland had been removed from the side of her neck. When examined, it was noticed that she seemed very ill, had a temperature of 102·4° F., and a pulse-rate of 112. There was marked converging strabismus with contraction of both pupils, and some photophobia. Her mother stated that for thirty-six hours or more she had been sick, was restless, and now and then screamed out when apparently dozing. She was very constipated, and an enema had produced but a small result. She slept badly and often refused food; any attempt at drinking brought on immediate vomiting. She had urinary incontinence. There was no disease of the ears or air sinuses.

For the first two days after admission her condition and symptoms remained about the same, but after this she began to pick at the bed-clothes, to throw herself about in bed in a restless, irregular manner, and to wet herself more frequently. At this period I noticed for the first time that she kept her right lower limb fully flexed and drawn up to the abdomen, whilst she moved the left from time to time, often extending it.

Palpation of the lower abdomen gave the impression of increased rigidity in the right rectus muscle and caused her to call out when even gentle pressure was brought to bear over the caecum. There was no distinct dullness on percussion anywhere. The existence of these physical signs on the right side together with the increasing sickness, quickening pulse, and general weakness determined me to explore the region of the appendix. The mother readily assented to the operation, and under complete ether narcosis I operated, utilizing the McBurney or gridiron incision.

When the peritoneum was opened a small quantity of semi-purulent fluid escaped, emitting a faecal odour. An inflamed process of omentum was found covering in the anterior surface of the cæcum and the appendix completely. In peeling the inflamed omentum from the appendix I came upon a colony of about a dozen dead threadworms. After the appendix had been cleared and delivered through the wound it was found to possess a pin-point perforation ¾ in. from its distal extremity on its mesial aspect. Through the minute perforation a living threadworm was to be seen wriggling until it finally extruded itself on to a swab held to receive it. In quick succession three or four others of smaller size, and presumably of less mature age, passed in single file through the opening. The base of the appendix was crushed and ligatured, and the organ excised according to custom. The omentum was then drawn into the wound and carefully examined, and, as a result, three more live worms were removed from its meshes at some distance from the inflamed and previously attached portion. In swabbing the pelvic peritoneum with hot saline in search for others, four more live ones were caught and removed. The wound was then closed. The appendix when cut open was found to be crammed full of oxyurides of all sizes and ages. The very small and immature worms greatly predominated. The mucous membrane was considerably ulcerated, especially in the immediate neighbourhood of the perforation.

The opening was so small that a fully grown oxyuris squeezed itself through with decided difficulty. Sincere regret must be expressed for my inability to show the specimen, and for the sad reason that whilst I was searching for a suitable receptacle an onlooker let it slip from his fingers into the sink, whence it could not be recovered. After the operation the nervous symptoms and sickness very soon disappeared, the pulse and temperature fell to normal on the following day, and from that time onwards the recovery was rapid and complete.

The case presents many points of interest, and amongst them may be enumerated:—

(1) The difficulty in making a rational diagnosis in the first instance was increased by the existence of an almost positive tuberculous history on the one hand, and by the entire absence of any other known cause on the other.

(2) The nervous symptoms played such an important rôle, and the pain was so generalized at the first and subsequent examinations, that there was little to direct attention to the local focus in the lower abdomen.

(3) The only truly localizing and diagnostic symptom was the continued flexure of the right lower limb.

(4) The imitation of a true organic nerve lesion or lesions by the reflex irritation produced by the active animalcules wriggling about on the exquisitely sensitive peritoneal surface.

(5) The immediate cessation of the urgent symptoms as soon as the cause had been removed.

(6) To the surgeon especially the behaviour of the omentum in coming to the rescue in this case, as it so often does in others, by limiting and preventing the extension of local mischief. In this particular case it appears not only to have checked the perigrinations of many of the worms, but it seems to have actually killed them. The colony of dead worms was to all appearances enwrapped by the adherent omentum and the individuals were held in bondage, as it were, in the immediate neighbourhood of their place of exit from the cavity of the appendix.

(7) The absence of infection as the result of the perforation in spite of the free escape of an odoriferous semi-purulent discharge on to the peritoneal and wound surfaces. A drainage-tube was used only during the first twenty-four hours after the operation. The case is unique in the operator's experience, and he has neither heard of a similar case nor has he seen an account of one in surgical literature.

DISCUSSION.

Dr. BELLINGHAM SMITH asked whether Mr. Whitelocke attributed the appendicitis to the presence of the threadworms: he had seen threadworms present in the appendix after appendicectomy on three or four occasions. In one case an operation for recurrent attacks of appendicular colic revealed nothing but a quantity of worms in a normal appendix. Also, were the meningeal symptoms due to the appendicitis, or to the threadworms? He had seen meningeal symptoms produced by round worms in the intestines, all the symptoms being relieved when the worm was vomited.

Mr. WHITELOCKE said he could only reply that in this case there was a perforation by the worms which presumably had been residing in the peritoneum for some days. As soon as the appendix had been removed and these worms extracted, the symptoms were at once relieved. He knew there were reflex nerve symptoms from the presence of worms in even the intestine itself, but in this case the peritoneal surfaces had been irritated.

Section for the Study of Disease in Children.

June 21, 1913.[1]

Mr. A. H. Tubby, President of the Section, in the Chair.

Case of Perineal Ectopic Testicle in a Boy, aged 17 Years.

By R. A. Milligan, M.D.

Patient sought advice on account of a lump in the perineum which at times swelled and became painful. Examination showed the scrotum to be empty. The right testicle was situated in the inguinal canal, while the left formed the lump complained of in the anterior part of the perineum.

DISCUSSION.

The President (Mr. A. H. Tubby) said that though he had read of these cases of perineal ectopic testicle, he had not hitherto come across such a case. There were plenty of instances of other kinds of misplacement of the organ. It had been shown that the position of these misplaced testes depended very much on which of the three processes of the gubernaculum testis was at fault. The strongest process was the middle one, which was attached to the bottom of the scrotum and drew the testis into its normal position. If it should happen that the process, which was attached to the front of the pelvic bone, between the symphysis pubis and the obturator foramen was stronger, it anchored the testis above the scrotum. In some cases it happened that the third process, which went to the perineum, was stronger than the others, and then one found a perineally misplaced scrotum. His experience in operating on misplaced testes was not large; but of late years experience had led him to endeavour to detach the testicle from all its surroundings, leaving it attached only by the spermatic cord and its vessels. Then one found that, up to a given point, the more thoroughly one separated the testicle the easier was the replacement, and the difficulty of keeping it in place was less. He counselled that procedure in this instance, and if it did not succeed, removal could still be carried out. The general opinion was now against removal straight away.

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[1] Provincial Meeting held at the General Hospital, Northampton.

Mr. CLOGG said his impression was that a perineal misplaced testicle was generally fairly well developed, but the present case was an exception, as the testicle was small for a boy of his age. He agreed with the President that the testicle should be put into its right position, and suggested that an attempt should be made to place the right testicle (which was in the inguinal region) into the scrotum. He had not been so successful in placing inguinal testes in the scrotum as had the President. He thought it easier to do so at an earlier age than the present case. He had several times divided the spermatic vessels, leaving only the vas and its differential artery, thus allowing the testicle to be placed in the scrotum. But in a few months the testicle was found at the upper part of the scrotum in front of the pubes. These testicles, when placed in the scrotum, did not develop. It seemed, in his opinion, to make no difference in the development of the testicle whether the spermatic vessels were divided or not.

Mr. R. H. A. WHITELOCKE said his experience with misplaced testes had not been so happy as had the President's or Mr. Clogg's. He had dealt with a good many such cases, but he had not yet satisfied himself that the testicle grew satisfactorily. They enlarged for a time, but later they shrank. Having watched several cases over years, he regarded them as disappointing. He considered that the enlargement following operation was usually in reality an orchitis. Recently he had taken to doing the complete separation which the President advocated, dividing all the concerned structures as freely as possible, thinking there was going to be a good reposition. He might not have done the perfect operation, but in three or four years the condition was just as bad: he knew of only one case in which the testicle had what approached proper functioning. Some surgeons with whom he had recently discussed the matter had gone so far as to advocate the removal of the organ, and inserted paraffin into the area to render the loss less obvious, and probably also for psychological reasons.

Case of Congenital Spastic Paraplegia, with Congenital Optic Atrophy.

By P. S. HICHENS, M.D.

F., AGED 6 years. According to the mother the child, though backward, was a normal child till she was 3 years old. She was then frightened by a Salvation Army band, and had two convulsions and developed paralysis of the right arm and leg, and paresis of the left leg. The child appears to be slightly microcephalic. She has congenital optic atrophy and spastic paraplegia of the right arm and leg, and, to a less extent, of the left leg. Despite the history, the case is probably one of congenital spastic paraplegia.

Cases of Familial Jaundice.

By P. S. Hichens, M.D.

Mother and two sons all suffering from familial jaundice. Mother has had ten children and five miscarriages. She has been anæmic all her life and became jaundiced during her first pregnancy; she has been jaundiced many times since. Children:—

(i) Female, 1892. Born yellow; died after five days.
(ii) Female, 1893. Born yellow; died after ten days.
(iii) Male, 1894. Born yellow; has frequent slight attacks of jaundice, otherwise in fair health.
(iv) Male, 1896. Slightly jaundiced.
(v) Female, 1898. In good health.
(vi) Male, 1901. In good health.
(vii) Female, 1904. In good health.
(viii) Male, 1906. Born yellow and has occasional attacks of jaundice.
(ix) Female, 1907. In good health.
(x) Male, 1910. In good health.

The mother and Nos. iv and viii are shown to-day.

Mother: Liver not enlarged; spleen extends below costal margin. Heart: Systolic murmur with its maximum at third left interspace and conducted upwards and to the apex.

Son (iv): Liver not enlarged; spleen just palpable. Heart: Systolic bruit with maximum intensity at third left interspace and extending up and down.

Son (viii): Liver not enlarged; spleen enlarged with a hard sharp border. Heart: A loud blowing systolic murmur is heard over the whole præcordial area and through to the back.

The other son (not shown) presented much the same murmur.

I would invite opinion on the condition of the hearts of these patients, as I have not seen such murmurs mentioned in other reported cases of familial jaundice, and it is remarkable that all these cases appear to have some heart lesions, possibly congenital.

DISCUSSION.

Dr. LEONARD GUTHRIE asked whether Dr. Hichens regarded these cases as examples of obstructive or of acholuric family jaundice. Did the urine contain urobilin but not bilirubin? And were the stools dark in colour? If so, that favoured the view that they were acholuric cases, as also was the enlargement of the spleen. He did not regard the hæmolysis test as of great value. He would like to know whether Dr. Hichens was in favour of excision of the spleen in these cases.

Dr. HICHENS replied that the intra vitam staining of the red cells was not done in this case. These patients were all too well for one to think of attacking the spleen. The mother and the eldest son had attacks of abdominal pain when the jaundice exacerbated. The little boy was very delicate, and it was not certain that he would grow up. The cases contained no bilirubin in the urine, and in the one case properly investigated in the hospital the urine only once showed a faint trace of urobilin.

Case for Diagnosis.

By P. S. HICHENS, M.D., and F. A. WAGSTAFF.

CHILD, aged 2 years and 2 months. Breast-fed and weaned at the age of 10 months; then fed on wheat flour, Robinson's patent groats, &c. At the age of 15 months began to swell all over and hair began to grow copiously all over her body; her gums became spongy and four of her milk teeth were shed; she became extremely irritable and passionate; some pigmentation was noticed over the lower part of the abdomen, and peeling of the hands and feet. There appeared to be much fat in the limbs or else a myxœdematous condition; no cushions of fat above the clavicles. Slight rickety changes in the limbs but no pain in them. The child has been treated with thyroid extract 5 gr. daily and has made a good recovery.

DISCUSSION.

Dr. HICHENS added that the changes about the long bones showed very slight rickets, but in some respects it looked like myxœdema, though the growth of hair did not seem to support this. He drew a bow at a venture, and gave thyroid extract. The child did not improve quickly on that, but was now very much better. He had thought it was possibly a case of progeria.

Mr. WAGSTAFF said that when he first saw the child the œdema was extreme, as also was the growth of hair. The child looked like a cretin, except that there was no obvious flattening of the skull, and the child was decidedly intelligent, though very irritable. The gums seemed like those produced by acute rickets, but there was no enlargement of the ends of the long bones, and no tenderness of them. There was exaggerated œdema of the forehead and face, but there was no albumin in the urine. He did not regard the swelling as fat, but the patient looked like the subject of acute nephritis.

Dr. LEONARD GUTHRIE said he supposed that the successful result of the treatment must show that the thyroid gland in this child was implicated. But hirsuties was associated with disease of the adrenal cortex, and was nearly always present in association with hyper-nephroma. Therefore there might have been some inter-relation between the suprarenal gland and the thyroid gland in this case. The suprarenal affection probably induced the hirsuties, but he did not see why the condition disappeared under the administration of thyroid. It was very rare to find hirsuties in myxœdema, for as a rule in that condition there was alopecia. He had only heard of one case of a cretinous child having precocious sexual development and hirsuties, and that child, aged 9 years, was shown at a meeting of the British Medical Association, held at Barnstaple, by Mr. F. Wellesley Kendle. Under thyroid treatment hair disappeared from the body, the breasts became smaller, and menstruation, which had occurred at the age of 5, ceased.[1]

Dr. HARRY CAMPBELL drew attention to the fact that the child had had considerable pigmentation over the abdomen.

Dr. D. STONE said he understood that when thyroid was given to monkeys it produced a peculiar condition of the hair. Hale White's book contained an illustration of a monkey in which the hair on the forehead had disappeared after the administration of thyroid.

Dr. ROBSON asked whether the child suffered from a low temperature, because one of the symptoms of hypothyroidism was an abnormally low temperature. The children with this condition suffered from cold hands and feet, although they were properly clad.

Dr. G. PERNET said he had been much interested in this case, and since he saw it several things had occurred to him. Growth of hair was not usual in hypothyroidism; it was the other way about. He suggested the hypophysis cerebri might be responsible for the growth of the hair which had been observed in this case.

Dr. LEONARD GUTHRIE said that cases of adiposis cerebralis were usually associated with loss of hair on the body, and not hirsuties. In a case of adiposis cerebralis with loss of sexual power there was recovery of that power after the operation, and the hair began to grow again. It was difficult to say which gland was primarily at fault, and how far the condition was due to an upset of the relationship between the glands mentioned.

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[1] Cf. *Brit. Med. Journ.*, February 4, 1905.

Dr. G. A. Sutherland said he considered that the success of Dr. Hichens's treatment had embarrassed the diagnosis. Dr. Hichens had succeeded in removing all the symptoms, and there was only the history to go upon. He understood the administration of 5 gr. daily of thyroid had been going on for twelve months. That seemed a large dose for a child, especially if that child was suffering from a deficient thyroid, because in those cases big doses tended to produce alarming symptoms. His impression was that the facial expression distinctly suggested a cretinoid condition, and that the thyroid gland was at fault. The stamp of previous hypothyroidism was on the face still. He would be inclined to stop the administration of the thyroid, and watch the patient.

Dr. Hichens, in reply, said that the temperature of the child was not taken, but Mr. Wagstaff informed him that the temperature of the hands and feet did not seem to be below the normal.

Case of Purpura.

By W. M. Robson, M.D.

C. A., male, aged 10 years. On May 8, 1913, trod on a rusty nail and developed a poisoned foot. On May 11 red spots appeared on both legs below the knees. On May 18 he began to vomit and had griping pains in the abdomen. Admitted on May 28 with vague pains in his knees, elbows, and wrists; temperature 99° to 100° F. On the limbs, especially about the knees and wrists, a purpuric rash was present. The heart is normal and the joints were not swollen or red but tender on pressure. The urine contained albumin and blood. Microscopically: Pus cells, red blood disks, but no casts. On June 8 the rash became more profuse, there was more blood in the urine, and the joint pains again more marked. There is no history of previous hæmorrhages.

DISCUSSION.

Dr. Robson added that he wished to arrive at a conclusion as to why the purpura had arisen. The persistence of the pain inclined him to think it was rheumatic. The patient had always been somewhat constipated and abdominal distension had been present. He could not feel the liver or the spleen. One member to-day thought he had ascites, but others did not think so. The blood showed little abnormality, the red cells being 5,400,000, the whites 15,000, and the hæmoglobin 90 per cent. He thought it might be Henoch's purpura, or it might be a septicæmic condition arising from the septic foot.

Dr. G. PERNET said he could not agree that it was real purpura. He would call it a purpuric rash in a boy in a toxic condition. In some of the cases in which albumin was present in the urine, dryness and scaling of the skin occurred as in this case. In such cases, however mild or slight the purpuric rash might be, it was always well to remember the possibility of visceral hæmorrhages. In the present patient there had been hæmaturia, which confirmed the view which he always took when he found purpuric rashes about the body. He did not know what the cause was in this case; he regarded purpura as a symptom only. The term "Henoch's purpura" was used in a loose way, he thought, and he did not think that Henoch, if he were alive now, would agree with some of the cases associated with his name. The original paper showed that Henoch's purpura was a fulminating purpura; it affected the skin markedly over large areas, and visceral hæmorrhages occurred. These cases were generally fatal. As far as he remembered there was no mention of intussusception in Henoch's book.

Dr. LEONARD PARSONS called to mind one case which he saw not long ago in which there was a rash similar to the one present in this case and a lump in the abdomen. The lump was due to an intussusception produced by hæmorrhage into the bowel. The mass was excised by Mr. Seymour Barling and the child recovered. It was a question whether in the present case the condition was a septic one. He thought many cases of so-called Henoch's purpura were really septicæmias; but how was one to draw the distinction? It seemed to him frequently impossible. A patient of Mr. Hugh Lett's died after a second attack following the operation for relief of intussusception, and in that case the skin was extensively involved; this occurrence was rather against the view expressed by Dr. Pernet. In one or two cases of Henoch's purpura operated upon for supposed intussusception it was not present; the condition turned out to be tuberculous peritonitis.

Mr. SEYMOUR BARLING considered that the case was one of Henoch's purpura because of the abdominal pain, the joint conditions, the association of the purpura, and above all the presence of renal hæmorrhage. He agreed that the term "Henoch's purpura" was loosely applied, though he had not read Henoch's original description. Henoch's purpura was usually associated with certain abdominal symptoms, and sometimes itself originated an intussusception. He referred to the case of a boy about the same age as this patient, in whom the distribution of the rash was almost identical, though the rash was not a marked feature. There was constantly recurring abdominal pain, and on the appearance of the purpura the condition was diagnosed; later on, intussusception occurred. He thought there was a liability of these cases being mistaken for intra-abdominal conditions which called for operation. Some of them were so fulminating that they seem to call for exploratory laparotomy to distinguish the condition from intussusception. But that procedure, where the condition was doubtful, was not without danger. In some cases purpura had recurred with great hæmorrhage into the skin, which had consequently sloughed and added to the risks of the patient.

Mr. Percival Mills said he once made the mistake to which Mr. Seymour Barling referred. Some years ago he operated upon a boy, aged 10 years, for what he supposed to be an intussusception. He found in the small intestine about twenty patches of congestion and œdema, which were arranged at regular intervals down the intestine, such patches being separated by portions of normal intestine. He was sure many of these cases were missed at first, even when they were operated upon, because the surgeon, having found a patch of œdema, considered he had come upon the site of a reduced intussusception. The wound in the case he referred to suppurated somewhat, but eventually recovery was good.

Case of Septic Dermatitis.

By W. M. Robson, M.D.

L. T. B., male, aged 4½ years, began to have blisters on him three years ago; they were never profuse. In July, 1912, a more profuse eruption began on the legs and gradually spread to the abdomen, arms, face and neck, and lastly, to the back. The groins and axillæ have been more affected than the rest of the body. Other children of the same family normal. Admitted May 14, 1913. Some of the bullæ were small and contained clear fluid, others were opaque. On May 18 some of the bullæ became septic and the temperature rose to 101° to 102° F. A culture taken from an apparently non-pustular blister gave a pure culture of *Staphylococcus aureus*. Treatment has been increasing doses of liq. arsenicalis, boracic baths, and injections of *Staphylococcus aureus*.

Dr. G. Pernet said the case was masked by treatment and by the large amount of secondary pus infection which the child presented, even at the present time. He did not doubt that when first the patient came under care the septic condition was much worse than now. But on trying to reconstruct the case in its original condition, he concluded it was an instance of dermatitis herpetiformis, with much secondary pus infection. Some impetigo contagiosa was also present. In impetigo contagiosa in children bullæ might occur. He had recorded in the *Transactions* of the old Children's Society the cases of four children who suffered from impetigo contagiosa bullosa, as a result of pediculosis capitis[1]. Taking all the facts into consideration, he diagnosed the present case as one of dermatitis herpetiformis, and suggested the giving of salicin instead of liquor arsenicalis. He would treat the pus lesions with diluted ammoniated mercury ointment, giving also weak tar baths, and supplementing by a vaccine to combat the secondary pus infection.

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[1] *Reports of the Society for the Study of Disease in Children*, 1902, ii, p. 37.

Complete Congenital Absence of both Radii in a Boy, aged 6 Years.

By W. M. Robson, M.D., and N. B. Odgers, M.Ch.

R. S., aged 6 years, has had "club" hands since birth. Both hands are kept in the extreme varus position, but, since an operation at the Royal National Orthopædic Hospital three years ago, can be straightened with a splint. An X-ray photograph shows complete absence of both radii.

The President said it was comparatively common for those who saw much of deformities to see cases somewhat like the present. Some of the cases had the thumbs developed as this patient had, but most had no thumbs. In other cases there was partial development of the radius, especially the upper part. In other cases there was no sign of radius. Another broad distinction was that some of the cases had very little power in the hands, while others had good power in the hands. All the cases were alike in this particular, that there was great difficulty in making a useful member of the deformed part. He had tried every possible way which had been described. He had most commonly split the thickened ulna vertically, after doing the necessary tenotomies, and shifted the outer part of the ulna as far as possible to the outer part of the carpus, and anchored it there with silkworm gut. In one or two cases he thought he had improved the stability of the hand. But a week ago he went further. Extensive division of the tendons had been done, and osteotomy of the ulna, to straighten it as much as possible. In all these cases one found there was a very curved ulna, as well as a thickened and deformed one, and in this case he exposed the upper part of the radius, and then made a long channel downward between the muscles; and having just previously removed the astragalus from another patient, he used one portion with cartilage attached to form a new wrist-joint, and the other part of the astragalus he cut into long columns of bone, and laid it carefully into the groove. He did not know yet how it would turn out, but he hoped the result would be to make a new radius. There was, however, no prospect that the arm would have the power of pronation or supination, because the arrangement of the tendons and muscles was very abnormal. The hand, however, would probably be more stable by being fixed and attached to two bones instead of one bone.

Synovitis of Hands, Feet and Knees, in a Congenital Syphilitic.

By W. M. ROBSON, M.D., and N. B. ODGERS, M.Ch.

D. E., FEMALE, aged 18 years. At the age of 8 years she began to have rheumatism which has affected her hands and feet and knee-joints at different times. The joints of both hands and wrists are swollen and deformed; she has marked nerve-deafness, the signs of old interstitial keratitis and Hutchinson's teeth.

DISCUSSION.

Mr. SYDNEY STEPHENSON said the case reproduced one shown by Dr. Partridge and himself at the old Society (1903). Both cases were markedly syphilitic, both had suffered from interstitial keratitis, both were deaf, both had the small joints of the hands affected, and in neither did the affection appear to involve the bone; it was entirely periosteal. The cases reminded one of the truth of the adage enunciated by Sir Jonathan Hutchinson years ago—viz., that when one met with a condition like osteo-arthritis in a child the cause was, in nearly every case, inherited syphilis.

Dr. PERNET agreed with Mr. Stephenson with regard to the syphilitic point of view. He regarded the condition of the hands as part and parcel of the congenital syphilitic syndrome.

The PRESIDENT remarked that in this case the appearances of the hands were quite different from what one would see in osteitis deformans in a child, for here the swellings were largely bulbous and limited to the joints. There were no spindle-shaped swellings of the shafts, nor was there a thickening of bone, as shown by X-rays. It was a good case of a congenital syphilitic polyarthritis, and he was glad to have seen it.

Case of Coxa Vara.

By W. M. ROBSON, M.D., and N. B. ODGERS, M.Ch.

H. C., FEMALE, aged 4 years. For some months the child has walked badly. The left leg is ½ in. shorter than right. An X-ray plate shows nothing abnormal in the femoral head and neck.

Case of Lymphangioma of the Tongue.

By N. B. ODGERS, M.Ch.

A. N., MALE, aged 29 years. Since birth he has had a "nævus" of the left side of the tongue and this has steadily got bigger; lately it has been more painful.

DISCUSSION.

Mr. ODGERS said he was inclined to excise half the patient's tongue, and had prepared the patient to consider the loss of half of it, especially as he was beginning to have some bleeding from it, and it was getting steadily bigger.

Mr. WHITELOCKE thought half the tongue should be removed, as it was evidently growing and causing considerable irritation. Though the patient had hardly reached the cancer age, the irritation to which the tongue was subject might result in cancer if it were left.

Mr. SEYMOUR BARLING said he had treated a similar case by deep punctures with a galvano-cautery, and the patient was doing fairly well. It seemed a drastic procedure to remove half the tongue. The cautery treatment should be extended over some time to prevent too severe a reaction setting up on any one occasion.

Dr. PERNET said that an analogous congenital condition, lymphangiectodes, might affect the skin. The cautery point was useful in such.

Dr. ROBSON referred to a case which Mr. Odgers and he treated some years ago, to draw attention to the fact that treatment by causing clotting of the blood in a big nævus was not always devoid of danger. It was a case of nævus of lip and inside of mouth treated by electrolysis, under anæsthesia. After the first dose the boy suddenly died in the night. Death was due to pulmonary embolism.

The PRESIDENT remarked that it was well known that these lymphatic conditions were by no means confined to the tongue. This man said he occasionally got swellings about his neck. A lymphatic condition of the tongue was almost always associated with hygroma of the neck, therefore removal of half the tongue would probably do no more than ameliorate the local condition. During the last ten years he had been seeing a girl with an extensive vascular nævus of half the tongue, soft palate, pillars of the fauces on that side, the cheek, the lip, and the neck. She presented herself once each six months, and he applied a blunt thermo-cautery, and that kept the tongue within reasonable compass.

Dr. D. Stone commented on a case of sarcoma which was sent to Paris for radium treatment. The patient came back temporarily improved, but soon became worse than ever.

Mr. Odgers, in reply, said the institution did not possess enough radium to try it in this case.

Case of Symmetrical Swelling of the Nasal Bones.

By N. B. Odgers, M.Ch.

F. L., male, aged 18 years. The anterior nares are almost completely occluded by the hyperostoses of the nasal processes of the superior maxillæ; he is evidently a congenital syphilitic.

The President said Mr. Stephenson and he looked at this case, and simultaneously agreed that it was more like leontiasis ossea than congenital syphilis. The bilateral affection of the nasal bones and the superior maxilla, and the curious facial conformation were very like three cases he had seen. In 1888 he saw a case of leontiasis ossea in Fraenkel's clinic and he had seen others since. He had recently seen two unusual cases, one of which Mr. Stephenson also saw, with localized hypertrophy affecting the bones of the cranium. In one there was hypertrophy of the supra-orbital ridge. He removed the portion of projecting bone and found it to be quite normal bone. Another case, that of a Jewish boy, had a curious enlargement affecting the parietal and frontal bones. He removed a portion, and here, too, it was normal bone. Thus there were two kinds of cases of leontiasis ossea, one in which the condition was symmetrical and diffuse, and the other in which it was localized and asymmetrical.

Case of Fibro-sarcoma of the Semi-membranosus Muscle.

By N. B. Odgers, M.Ch.

T. B., male, aged 14 years, has since birth had a tumour in the right thigh; for six months it has been getting bigger. He had on the inner aspect of the back of the thigh a solid tumour which appeared encapsuled. In March, 1913, I operated on this and found it growing from the sheath of the semi-membranosus muscle, which it infiltrated; this latter was removed in its whole length with the tumour. The tumour appears degenerated in many places; in its upper pole is well-formed bone. Microscopically it appears to be a fibro-sarcoma.

Case of Cellulitis of the Forearm.

By F. A. WAGSTAFF.

M. J., AGED 24 years, fractured her left forearm at the age of 8 years. For the last three to four years she has had attacks of pain at the seat of fracture lasting for two to three days. Six weeks ago she came with a condition resembling a cellulitis of the forearm with exquisite tenderness at the seat of the old fracture and at various spots below it; in a week the cellulitis subsided. Tenderness and disability remained for some weeks. X-rays show dotted about the forearm a dozen or more discrete shadows, some of them the size of split peas, the majority being smaller.

DISCUSSION.

Dr. ROBSON said that when he first used the screen to inspect the case he asked the girl whether she had ever had a gunshot wound, and she replied that she had had nothing but a broken arm. The skiagram showed nodules scattered about the whole forearm. The muscles showed as a deeper shadow in the more translucent shadow of the fat and skin. All the nodules were in this deeper shadow. He concluded that there were little phleboliths in the deep veins. If so, they must have been there a long time before the acute onset of pain. Probably an acute inflammation brought the condition under notice because of the pain it caused.

Mr. PERCIVAL MILLS agreed that they were phleboliths, and pointed out that when the patient clenched her hand so as to make the veins prominent, the nodule which was visible under the skin was rendered more prominent.

Dr. SUTHERLAND thought it might be commencing myositis ossificans.

Mr. WHITELOCKE said that he was at a loss for an explanation, because the nodules seemed not to follow any anatomical structures at all: they were not in the course of veins or nerves. He could not say from the skiagram whether they were in front of or behind the bones or on both sides of them.

Dr. PERNET said the condition suggested to him fibro-neuroma.

The PRESIDENT said the striking feature about the case was the persistence of the cellulitis. There did not seem to be any very definite help to be given.

Case of Ectopia Vesicæ.

By D. STONE, M.D.

J. O. K., MALE, aged 2¾ years. He is an apparently healthy boy; he has complete epispadias, while the posterior wall of the bladder forms a deep red bulging mass about the size of a crown, from which urine slowly oozes. On the rudimentary penis is a mark resembling the urethral opening; the testes are both present in the inguinal canals.

DISCUSSION.

Dr. STONE added that, contrary to the usual experience, the boy had grown to the usual size and weight, and, so far, there was no septic inflammation; there was a red mass where the bladder ought to be and there was a slight depression just where the urethral channel should be. The symphysis pubis was also absent. Seeing that the boy was suffering only from the inconvenience, he raised the question whether it would not be well to treat him with some apparatus—a kind of pad with a circular ring round it.

The PRESIDENT said the only successful case of the kind he had seen was a brilliant one seen when the Section visited Edinburgh four years ago. It was Mr. Stiles's case, and the patient was 8 years of age. Mr. Stiles had implanted both ureters into the pelvic colon, and the patient was able to retain the urine in the rectum for a couple of hours or more at a time. It would be agreed that these cases did not usually offer a good opportunity for plastic operations, and surgeons were rather unwilling to attempt to treat them.

Case of Cerebral Tumour.

By D. STONE, M.D.

I. R., AGED 6 years and 10 months, born healthy, grew to average height and weight and well till last August. She then contracted whooping-cough, which was followed immediately after by an attack of measles. Since this period her present illness has lasted till now. Her only sister, aged 15 years, is in good health. Between the two there have been three miscarriages, one at four months' gestation and two at three months'. The mother suffers from pain in the left arm of an obstinate character, and worse at night. Four years ago she lost a good quantity of hair from one spot over the right temple. This left a bald patch for some time. She also suffered at times from sore throat accompanied by loss of voice, and has had enlargement of the glands in the right side of the neck. Before the child was seized with whooping-cough she suffered from strabismus—internal, from the mother's description. Since last August she has been confined to bed or the couch with paralysis of the right side of the body. Up to the last few weeks she had attacks of vomiting, accompanied by headache.

Present condition: Right half of face, right arm and leg paralysed, with coldness of the hand and foot. There is absolute inability to move hand or foot on that side, and, in smiling, the right half of the face remains motionless. On the same side there is an exaggerated patellar reflex, ankle clonus, and the Babinski sign. The left eye exhibits several changes. There is ptosis, very marked, the pupil is widely dilated, and there is paralysis of the muscles of the eyeball, almost complete. Slight movement outwards is the only movement detectable. There is no drawing up of the eyebrow on that side. The pupil is inactive to light or accommodation (ophthalmoplegia interna). The head is square-shaped and the forehead is high and prominent. The teeth are short and overcrowded. She apparently hears well, but replies to questions in monosyllables, and in a slow and hesitating manner, and is inclined to sleep throughout a good part of the day. On tapping the skull near the junction of the right parietal bone with the frontal some pain is elicited.

Dr. HARRY CAMPBELL said he thought this was a case of lesion of the left crus cerebri, and as there was a history of syphilis, presumably the lesion was syphilitic, possibly a gumma. The peculiar point was that, in spite of the unmistakable hemiplegia on the right side, with evidence of degeneration in the pyramidal tract, the paralysed muscles were in a condition of extreme flaccidity. This flaccidity, however, affected all the muscles of the body. He believed it to be connected with the condition of extreme coma present. Cases of hemiplegia in which rigidity was conspicuous by its absence were not very rare and had not yet been satisfactorily explained.

Case of Hydatid Cysts of the Liver.

By D. G. Greenfield, M.D.

G. S., male, aged 10 years. When he was aged 3 years his mother noticed a lump on the right side of his abdomen; a year later there was a large swelling extending downwards below the right costal margin. Between 1909 and 1911 three hydatid cysts were drained in Guy's Hospital by Sir Alfred Fripp. A year ago he had slight hæmoptysis on two occasions; his liver is still considerably enlarged.

Dr. Greenfield added that there was a thrill. At first he thought the swelling was connected with the kidney, but at Guy's Hospital Dr. Hale White considered it was continuous with the liver. When operated upon there was found to be a hydatid cyst. The other cysts seemed to be contained in a large mother cyst. This was drained, and a year later two other cysts developed. Those also were drained, not enucleated. Since then the patient had had two slight attacks of hæmoptysis. The liver was still very large and displaced downwards. Before operating, the liver was well below the umbilicus.

Case of Congenital Eye and Heart Defects.

By D. G. Greenfield, M.D.

H. A., male, aged 11 years. A full-time child weighing 1 lb. 15 oz. at birth. His mother has had no other children or miscarriages. He had convulsions as an infant and at the age of 8 months developed a "cataract": he has always been deaf and can hear nothing with the right ear and very little with the left. Since the age of 5 years he has had a left otorrhœa. He can read with the left eye, but the right eyeball is small. He has a congenital heart lesion. There is no evidence of congenital syphilis.

Cases of Congenital Ocular Lesions.

By E. H. Harries-Jones, M.D.

Case I.—J. H., aged 9 years. Right eye: Persistent capsulo-pupillary membrane (pigmented). Left eye: Pigmented area on anterior capsule of lens.

Case II.—E. C., aged 23 years. Right eye: Coloboma of iris and choroid downwards and inwards (usual kind).

Case III.—M. S., aged 23 years. Right eye: Coloboma of iris and choroid (downwards and inwards). Left eye: Marked microphthalmos, with coloboma of iris and choroid.

Case IV.—J. F., aged 11 years. Left eye shows persistent hyaloid artery—not blood-bearing—passing from the optic disk to the posterior pole of the lens.

Case V.—Child showing congenital dislocation of crystalline lenses, outwards and downwards.

Case of Paralysis of both Internal Recti with Slight Ptosis.

By E. H. Harries-Jones, M.D.

F. C. S., aged 5 years. Almost complete paralysis of third nerve on both sides; eyes divergent, with widely dilated pupils which do not react to light or accommodation. Ptosis only partial. The eyeballs do not move upwards, downwards, or inwards. The optic disks are pale on their temporal sides, but the vessels are normal, and there is no sign of there having been any optic neuritis. The history of the case is peculiar, the mother stating that the child went to school last January (six months ago) "quite right," but returned in the afternoon with the eyes in the present condition. There have been no further symptoms—no headache, giddiness, or sickness.

DISCUSSION.

Dr. Harry Campbell suggested that this case was one of acute nuclear lesion, similar to acute anterior poliomyelitis (polio-encephalitis superior). Such cases were rare. The disk pallor somewhat complicated the diagnosis, but in cases of moderate pallor of the disk it was difficult to be sure that actual atrophy was present. He emphasized the suddenness of the onset. Paralysis might occur suddenly in cerebral tumour, but he thought that the sudden occurrence of extensive symmetrical ocular palsy argued against the diagnosis of tumour.

Dr. LEONARD PARSONS said the case showed bilateral paralysis of third nerve accompanied with slow tremor brought out by movement. He suggested it was tumour of the corpora quadrigemina, involving the third nerve nuclei and the red nucleus, or at least the region between the red nucleus and the optic thalamus. He had seen three similar cases. One Dr. Batten reported, with the post-mortem findings, before the Ophthalmological Society in 1907. He did not regard it as polio-encephalitis. In this case there were no constitutional symptoms, which was against tumour somewhat, but more against polio-encephalitis; and it occurred in January, when polio-encephalitis was not rife.

Dr. LEONARD GUTHRIE said that in polio-encephalitis superior it more frequently happened that some of the cells were affected more than others, and it was rare to find such a complete bilateral condition unless the nerves were involved. He thought the more likely diagnosis was tumour of the corpora quadrigemina, causing practically complete paralysis of both third nerves.

Dr. LEON remarked that the mother stated that since the squint came on the patient suddenly stumbled if he tried to walk faster.

Mr. SYDNEY STEPHENSON remarked that the symptom spoken of by Dr. Leon might be accounted for by the condition of the muscles; the patient might have diplopia, and fall in consequence of that. Two apparently similar cases which he could recall turned out to be instances of meningitis; and a third case was cerebral tumour. Involvement of the internal recti, especially on both sides, was a rare manifestation of polio-encephalitis superior. He published a paper on the subject a year ago, in which he recalled cases of paralysis of external recti from, as he considered, polio-encephalitis superior. In the present case there was also ptosis, and other branches of the third cranial nerve were also affected.

Remarks on some Fatal Cases of Eczema in Children.

By P. S. Hichens, M.D.

This little paper is offered to the Section for the Study of Disease in Children, not because it professes to throw any particular light on the subject dealt with, nor because it embodies any research worthy of the name, but because it deals with what is, I believe, rather a rare occurrence, and because I hope it may elicit from the learned medical men gathered here to-day some light on catastrophes which have puzzled me personally very much.

I have been a physician to the Northampton General Hospital for the last twelve years, and I find on looking through my notes of the cases treated in the children's ward, that I have admitted and treated in that ward twenty-eight cases of eczema, and that no fewer than six of them ended fatally. Such a proportion of unfortunate results may be regarded as particularly unlucky or particularly fortunate and interesting, according to the light in which they are taken, but at any rate they distressed me very much at the time, and I could see no adequate reason for their occurrence.

It may be well, therefore, to give in very brief detail the five fatal cases on which I have notes. The notes of the sixth have unfortunately been lost.

(1) F. C., male, aged 7 months, admitted May 8, 1904, with chronic moist eczema of the head and face; had had eczema since he was 14 days old. Treated with lotio plumbi and unguentum plumbi carbonatis. Began to seem very ill on May 9, and died on May 11, with temperature rising to 103° F.

(2) M. B., female, aged 6 months. Admitted October 22, 1904; she had had eczema for the last five months. It began behind the ears and then spread acutely on to the head and face. It is also present on the nates. On the second day after admission the temperature rose to 104° F., and then rapidly fell to normal. The patient was treated with liquor plumbi subacetatis diluti. She made rapid improvement and seemed in a fair way of complete recovery, when she suddenly became extremely pale and collapsed and died in the course of a few hours on November 4, 1904.

(3) D. H., female, aged 6 months. Admitted April 29, 1905.

Chronic moist eczema of the head and face since 5 weeks old. She had bronchitis and pneumonia when 3 months old. The child looked very ill, cried a lot, and did not feed well. On admission the temperature was 105° F., but dropped the same day to normal and remained at that level till May 29 and 30, when it rose to 100° F. and then 101° F. The child was treated with liquor plumbi subacetatis diluti and then calamina præparata 1 dr. and unguentum zinci benzoati to 1 oz. May 28: Eczema quite dry and beginning to disappear. May 30: Eczema gone; the patient suddenly became very ill, pale and collapsed, and had twitchings of the right arm and leg. May 31: The pulse became weaker and weaker; there was general twitching, unconsciousness and slight convulsions, and the patient died without regaining consciousness.

(4) G. E. S., male, aged 6 months. Admitted May 2, 1907, with chronic moist eczema of the face, scalp and right arm. Had had bronchitis at the age of 2 months. He was treated with calamina præparata 1 dr., unguentum zinci benzoati ad 1 oz. The eczema slightly improved, but on May 14 he was seized with diarrhœa and vomiting, and sank and died on May 15.

(5) M. G., female, aged 1 month. Admitted with a universal acute eczema on November 15, 1910. She was treated with tinct. lavandulæ, 15 minims, emplastri plumbi, olei olivæ āā partes æquales ad 1 oz. The temperature was 102·8° F. on admission, and fell to subnormal before death, which occurred in a state of collapse on November 18.

I am sorry to have to confess that in not one of these cases was I able to obtain a post-mortem examination. Regrettable though this is, I am inclined to think, however, that in all probability an examination would have thrown very little light on the cause of death.

Of the five cases recorded, the second and third in the series seem to me much the most interesting from the point of view of discussion. The first and the fifth cases were in the hospital such a short time that not much can be said about them, and of the fourth case it might be said that the diarrhœa and vomiting, which came on before and apparently caused the fatal issue, were intercurrent affections and quite enough in themselves to carry off a weakly child. The second and third cases, however, seem to me extremely interesting. In the second case the recovery from the eczema was rather unusually rapid, and all seemed to be going on to a completely successful issue, when without any warning all the phenomena of shock with collapse developed, and the patient died in the course of a few hours. In the third case the recovery from the eczema was much more gradual, but on recovery the same symptoms supervened, though the fatal issue was not quite so rapid, and was accompanied with symptoms of cerebral irritation.

To my mind it is very difficult to find any adequate theory to account for deaths in such cases, and from the small search I have been able to make in the comparatively slight allusions to this matter in medical literature, little light is thrown on the subject.

Dr. Leonard Guthrie,[1] Dr. Pernet and Mr. Arthur Edmunds allude to such deaths occurring in cases of skin lesions.

Dr. Guthrie alluded to it more especially in cases of syphilis, but asked whether members of the Society had met with such deaths in cases of chronic eczema of long standing, which had been followed by rapid healing, and then sudden and unexpected death. He mentioned a case in a child, aged 2 years, which had been treated with such success that in ten days the eruption was nearly gone. The improvement was so remarkable that the matron drew the mother's attention to it, and the mother remarked, "I feel sure you are curing that too quickly." The next day the child had symptoms of gastro-enteritis and died. He also mentioned a case of ringworm of the scalp which was treated too vigorously by the mother with an ointment containing cantharides and red oxide of mercury. The child died in twenty-four hours. In the case of eczema the post-mortem examination showed a marked condition of congestion in almost the whole gastro-intestinal tract; Peyer's patches were raised above the surface and dark red in colour, and the mucous membrane was swollen and inflamed and marked by areas of injected arborizing vessels. In the case of ringworm only hyperæmia of the brain and congestion of the kidneys were found. He believed that in some instances the death might be due to poisonous applications absorbed through the cutaneous surface, which was denuded of epithelium. But that would not account for all, and he was inclined to think there was some ground for the prevalent belief in the harmfulness of "driving in" an eruption as it was called.

Dr. George Pernet said that, in a large experience of syphilitic and eczematous children at the Skin Department of University College Hospital, he had never known such an event occur, although they endeavoured to cure the eruption as quickly as possible. Perhaps the "*status lymphaticus*" might have something to say in the matter. He had certainly been told by old patients that when their weeping eczema was cured they did not feel so well in general health. He had felt there might be something in the contention of such patients. Perhaps the oozing surfaces gave the inadequate kidneys a rest, and helped them in that way.

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[1] *Reports of the Society for the Study of Disease in Children*, 1905, v, pp. 136-7. In a discussion on "Cases of Sudden and Unexpected Death in Children."

Mr. Arthur Edmunds mentioned a case of an extensive burn in a girl, in which every time skin-grafting was done over too large an area the patient became very ill, with a temperature and a mild attack of septicæmia; he attributed this to the corking up of drainage products. "In the cases of that kind if a limited checking of free discharge could produce septicæmia, it was only a matter of degree for it to be capable of causing death."

Maille[1] records two cases of eczema ending in sudden or rapid death. The first case was a child with extensive eczema and concurrent diarrhœa and enlargement of the liver. The child died in coma accompanied by a subnormal temperature, and post-mortem granular and fatty changes were found in the liver with partial necrosis, and the kidneys were not healthy. The second case was an infant, aged 8 months, with general eczema, which was treated with wet dressings, and died in convulsions. Fatty and granular changes were found in the liver.

Variot,[2] in a discussion of infantile eczema, says that all manifestations are preceded or accompanied by digestive trouble, vomiting, diarrhœa, and constipation; all pointing to intestinal toxæmia. He treats the cases with weak remedies, as too rapid a cure may react on the nervous system, causing sudden death with cerebral symptoms.

Dr. J. D. Rolleston tells me that Gaucher, in his lectures at the St. Louis Hospital, used to warn his audience against treating cases of eczema in children too energetically, owing to the possibility of sudden death occurring.

Looking at my own cases, I do not think the treatment can have caused sufficient absorption of metallic poisons to cause death. If that were so, one would expect death to occur while there were still large weeping surfaces, and not when the disease was almost or entirely healed; and, moreover, many other cases have been treated in the same way without death. Neither do I propose to bring in that blessed phrase "*status lymphaticus*" as a *deus ex machina*. I am not sure

[1] *Semaine med.*, 1907, xxvii, p. 255.

[2] *Gaz. d. Hôp.*, 1906, lxxix, p. 951.

that there is any such thing as *status lymphaticus*, and, at any rate, there was no special evidence of it in these cases.

It seems to me more likely that these untoward results must be explained by some sort of humoral pathology. On the one hand, one might suggest that the weeping surfaces of the skin were a manifestation of some internal toxæmia finding a vent on the surfaces of the body, and that the healing of the skin forced the poison upon the internal vital centres with fatal effect. Some obscure analogies of this kind may, I think, be found in certain gouty manifestations. Or, on the other hand, the fatal issue may be due, not to a toxic but to a physical alteration in the balance of the fluids in the body causing congestion of fluids in the brain and a fatal effect by pressure on the fourth ventricle. However, these are pure hypotheses, and everyone is at liberty to form his own conclusion.

I will only add that I have been so impressed by these catastrophes that I have become accustomed to warn mothers that their children may possibly be better with eczema than without it, and that its cure is not always an unmixed blessing.

DISCUSSION.

Dr. G. PERNET regarded the paper as one of great interest and lucidity. He could only repeat what he had expressed at a meeting of the Children's Society. Though he had not seen such cases as Dr. Hichens described, he remembered hearing of such from Gaucher in Paris. He (Dr. Pernet) had come across a number of cases in which a very irritable dermatitis, a papular, pruriginous, and also eczematous type, alternated with attacks of asthma. When the skin in those patients was in a very bad condition, they were not troubled by the asthma; but the asthma returned when the skin improved. Alternation had been noted of active tuberculosis and maniacal attacks in asylums. It might be that, especially in children, when a large surface of the body was involved, severe nerve or other disturbance might occur. Possibly, therefore, Dr. Hichens's suggestion as to a humoral explanation was correct. In some respects medicine appeared to be going back to the old humoral views of disease.

Mr. SYDNEY STEPHENSON said he could point to a familiar instance in the case of eye disease. Strumous ulceration of the cornea was frequently associated with eczema of the skin, and when the skin was healthy the eyes were bad, and vice versa. He believed there was much truth in what mothers said as to the danger of "driving the disease into the system."

Dr. LEONARD GUTHRIE said that probably many had doubted the occurrence of such cases as they did not happen to come within their own experience. He wondered whether the popular idea of the danger of "driving in" an eruption or curing a chronic ulcer was prevalent in this district. Up to the end of the eighteenth century, people who had "issues" used many precautions against healing them too soon, because of the supposed danger of a fatal result. Old women sometimes derived most of their income from exhibiting chronic ulcers which they would not allow to be healed. He knew of a case of chronic ulcer of the leg which had persisted for fifteen years. Under treatment healing occurred and within three months the patient had died of cerebral haemorrhage. Dr. Sutherland and he remembered the case referred to in the paper. The congestion of the intestines was very marked, their walls were a brilliant scarlet, due to the injection of the vessels there. The alternation of asthma with eczema had often been observed, though some skin specialists seemed disposed to deny it. Often he had seen children with eczema who had been treated in every possible way without much effect, and he had predicted that they would lose the eczema at about two years of age and become asthmatics. A lady had been a martyr to asthma for twenty years, and ten years ago she had a very disfiguring acne rosacea. For two years, in consequence of this, she was unwilling to leave the house and only did so when wearing a thick veil. During that eruption she had no asthma, but when her acne was cured by a skin expert the asthma returned. The profession must not despise the views which had been held from time immemorial, and he was very glad Dr. Hichens had brought this subject forward.

Dr. ROBSON said he had had one case similar to those recorded in the paper, in a private patient, a child 8 months old, apparently quite healthy. It had been brought up on the breast and was fully grown for its age. Apart from its eczema, it looked a bonny boy. It was not a very moist eczema; there were small dry scabs over the body. He prescribed the ordinary treatment for eczema, and was surprised to learn two days later that the child had died suddenly in the night. The special nurse had fed the child at 2 o'clock in the morning and got into bed herself; almost immediately she heard a slight noise and found the child was dead. No post-mortem examination was allowed and at the time he suggested that the child having just had its food, some of it might have regurgitated and choked the child. But that was merely supposition.

Nocturnal Enuresis.

By W. M. Robson, M.D.

The subject of enuresis is one that, from time to time, has received a good deal of attention from physicians, but which, nevertheless, is still in an unsatisfactory state, as regards both its ætiology and treatment. The number of surmises as to the cause, and the many different suggestions in the treatment, indicate that further inquiry into a large number of cases is still necessary before we can admit that our knowledge of the subject approaches the ideal. My intention to-day is not to dogmatize, or bring forward new material, but rather to place before you a short summary of the subject, as I have construed it from the literature and personal observation. That enuresis is the stumbling-block of many doctors, and the distress of every mother whose child is afflicted, is my excuse for choosing this malady for discussion. The newly born child normally passes urine reflexly, whenever the bladder becomes full enough to act as a stimulus to the centre in the spinal cord. About the end of the first year of life the child, during waking hours, has gained a higher or mental control, and is able to hold its urine until such time as the nurse or mother attends to its requirements. At the age of 2 to 2½ years most children can control their bladders during sleep as well as waking hours. Beyond this age absence of control becomes enuresis. About half of the cases are of this variety, in which the higher control has never become established. Of acquired cases, practically all begin before the age of 10 years. Sex incidence is variously stated by different authors, which rather indicates that, on the average, the sexes are equally affected. Slightly more than half of the cases occur during sleeping hours alone ; less than half during both waking and sleeping hours ; and hardly any during waking hours alone.

Most modern writers consider that enuresis is a symptom, the true cause of which must be diligently sought for and treated. I will mention the commoner causes, which have from time to time been credited with its origin, but, as it is my intention to limit this paper to cases in which no gross pathological lesion is found, it will be necessary merely to allude to some of the lesions, lest we should forget their existence.

Fluid in physiological excess, by giving rise to the secretion of an abnormally large amount of urine, may cause nocturnal incontinence. in a child otherwise healthy, from over-distension of the bladder. For a similar reason, drugs which cause diuresis have likewise been quoted as being responsible for the trouble. Much has been written on hyper-acidity and concentration of the urine as a cause of the malady, owing to its irritating effect on the neck of the bladder. If present, this may possibly be a factor in prolonging the trouble, but the majority of cases do not have it, and some others even improve in the summer months, when the urine becomes more concentrated and strongly acid. Excessive alkalinity has also been stated to be present in other cases. It has not, however, been proved that either of these conditions is present over extended periods in chronic cases, and probably too much stress has been laid upon them. Dr. Thursfield has pointed out that many cases have marked bacteriuria, and attributes the enuresis to this condition. On the other hand, it is stated that in some of these at least the infection is brought about by the urine-sodden and dirty condition of the clothing, setting up a vulvitis or balanitis, which spreads by the urethra to the bladder. Many cases with bacteriuria improve or get well when the urine again becomes sterile, but bacteriuria has also frequently disappeared when the foul clothing has been strictly attended to. Malformations, calculi, tubercle, tumours and polypi of the bladder are all local gross lesions, which must be borne in mind, but which I will not here discuss; whilst caries of the spine, spina bifida, diabetes mellitus and insipidus, are other causes of incontinence, which must not be lost sight of.

Reflex irritation has always held a prominent position in the list of causes of enuresis, under which heading are included intestinal parasites —especially threadworms—elongated prepuce, pinhole meatus, balanitis, vulvo-vaginitis, anal fissure and rectal polypi. It is difficult to estimate correctly the bearing which these conditions have on the affection as causal agents. Any one of them may be present in an individual case, but probably in the majority of instances not one of them is to be found. Moreover, each condition is found frequently in children whose control is perfect.

During recent years enlargement of the tonsils and adenoid growth have been recognized as having a definite connexion with enuresis. In two series of published cases (totalling 1,150) which were operated on, enuresis was noticed in 14 per cent. On the other hand, another writer examined fifty cases of enuresis, and found only eight who had adenoids, and of these only one was cured by operation. In this connexion it must be remembered that rheumatic children are susceptible to both maladies, and that the enuresis may be attributed to adenoids when rheumatism is really the underlying factor.

More recently still, attention has been directed to hypothyroidism in children who suffer from incontinence. Dr. L. Williams, quoted by Dr. Langmead in the *Medical Annual* (1913), has noticed a certain group of cases who are undersized and backward; they suffer from dead fingers and toes, have highly arched palates, always feel cold even though overclad, and have subnormal temperatures, especially at night. Dr. L. Williams attributes these signs to thyroid inefficiency.

One other condition, that of epilepsy, may give rise to nocturnal incontinence and must not be overlooked. In these cases the incontinence is not a constant feature, but is intermittent in character. This very intermittence should always lead one to suspect epilepsy.

In addition to the cases which may present one or other of those defects previously mentioned, there is still the larger number in which none of them are found. In such instances it is frequently noticed that the child is pale, anæmic, and listless, with perhaps signs of underfeeding, and may suffer from night terrors, migraine, hysteria, or other signs of nervous instability. On careful inquiry into the history of all cases of enuresis it is found that a very large number give evidence of nervous phenomena. The child is either mentally dull or is hypersensitive. Fright or profound emotion is the starting point of many, and it is often found that children who are run down from illness, and, therefore, whose nervous control is below par, are subject to enuresis who at other times are free from the malady. There are many points of close contact between enuresis and this nervous instability; in fact, so closely are they related that there is a growing tendency to make the nervous factor the one to which all other causal agencies are subservient. When enuresis is acquired we find that the majority of cases begin during the period of the second dentition—i.e., when a child's nervous system seems to be prone to disorder, and when other nervous phenomena, such as habit spasm, are apt to occur. Rheumatic children are notoriously "nervy," and this nervous factor, common both to rheumatism and enuresis, may explain their connexion. Threadworms, which so frequently are associated with enuresis, are, more commonly still, related to a symptom complex, of which nervous irritability is an outstanding feature.

As nocturnal control is the later to be established, so also is it earlier than diurnal control to go. Many writers lay stress on very deep sleep as a causal factor—the child sleeping so soundly that the warning of a distended bladder passes unheeded. If this be true, we have here an example of nervous origin, pure and simple, the higher central control being abolished as by a narcotic. I know a child who on two occasions, after chastisement by his father, has developed a temporary enuresis, which must surely have been purely nervous in origin. Another boy, aged 11 years, had enuresis till he was aged 9½ years, associated with night terrors, shyness, and a peculiar temperament. He would eat no meat, fish, or vegetable, and his mother had the greatest trouble with his diet. Occasionally he would go to bed for three or four days, waking only to drink some water, but refusing food. According to the mother, the enuresis stopped eighteen months ago without any special treatment being adopted. When I saw the boy recently I could find none of the exciting or reflex causes of enuresis, with the exception of slight adenoid growth without nasal obstruction. Had these been removed eighteen months ago, we should have had to report a clear case of cause and effect. The operation of circumcision may frequently stop incontinence, but I do not think we need therefore conclude that the phimosis was the only, or indeed the chief, cause of the trouble. I have known a case of persistent vomiting with gastrodynia cured by an exploratory laparotomy. Moreover, circumcision has been the starting point of some cases of enuresis. It is not my object, however, to minimize the relationship which these reflex causes have to enuresis, but rather to urge that, while keeping them in view, we do not fail to recognize, what may prove to be, the true cause lying in the background. In many instances the exciting cause is so slight that, were the child otherwise normal, incontinence would not occur. Conditions such as vesical calculus, however, will break down the barriers even in a child otherwise healthy.

And so we come to the proposition that I would place before you—namely, that excluding gross disease, such as tubercle or calculus in the bladder, and spinal disease, &c.—enuresis is primarily a functional nervous disorder.

In the treatment of enuresis there is no specific upon which we can depend for a cure. Too often, however, little, if anything, is done to shorten the course of what must be a secret worry and misery to the affected child. As a result it grows shy and retiring, and develops a dislike for the society of other children. Should the incontinence be allowed to continue into the years of boarding school life, the child will further be subjected to the ridicule or scorn of his fellows, and so acquire an outlook on life which may irreparably blight his future career.

It is of the first importance that the disease be treated vigorously, and not in the half-hearted manner that is so often done. This applies equally to the parent as to the doctor, and hence the physician must make it his duty to instil into the mind of the mother the necessity for sustained and possibly prolonged treatment. A thorough examination of the child should never be omitted, and the urine must be examined for any abnormality. Hyperacidity and concentration will require citrate of potash, and a diet likely to restrict the formation of excess of uric acid. Bacteriuria must be treated by giving urotropine in 3-gr. to 5-gr. doses three times a day, according to age; whilst the clothing must be kept free from fouling. As a rule, however, the urine is found to be normal. Threadworms, if present, are to be vigorously attacked until they are overcome. In this case santonin 1 or 2 gr. given on an empty stomach, and followed by a purgative or given with calomel, will prove the most helpful drug.

Should any of the signs of thyroid inefficiency be noted, $\frac{1}{2}$ gr. of the dried thyroid may be given twice a day, with a slightly larger dose for children over 6 years of age. Leonard Williams has had very promising results in such cases from this treatment.

As regards surgical treatment in cases of adenoids and phimosis, it is difficult to make any hard and fast rule. Should the adenoids interfere with nasal respiration, or cause a condition of ill-health, their removal should be advised. Whether, however, the removal of adenoids or the operation of circumcision should be lightly undertaken in all cases is more than doubtful. The effect of a surgical operation in cases of enuresis, as regards immediate cure of the disease, is probably purely psychical, just as in any other case of functional neurosis.

Similarly the application of silver nitrate or electric current to the neck of the bladder, the injection of saline into the lower end of the spinal canal or perineum, and massage of the vesical sphincter by a finger in the rectum, are all methods of producing a psychical effect, which might equally well be done by firing off a gun. Moreover, they have the distinct disadvantage that they so often fail to cure. Children are often wakened up and made to micturate before they have wetted the bed. This is an excellent method of keeping the sheet dry, but does not cure enuresis, for if the child be not waked up the incontinence will occur. I mention this because one so frequently hears it propounded as part of the treatment of the malady. An ingenious apparatus has been devised consisting of two metal plates separated by a layer of absorbent material, which are placed under the child's buttocks in bed. Each plate is connected by wire to one pole of a battery and electric bell. When the child passes urine the absorbent material becomes wet, allowing the current to pass, and so ringing the bell, which wakes up the child. This, after several applications, is said to have been efficacious in many cases.

The method of treatment, however, which has proved to be most valuable in the largest number of cases, is that by which the general health is built up on sound lines, and the nervous instability combated by suitable drugs. Should it be found necessary to curtail the amount of fluid taken in the latter part of the day, this should be made up by allowing a little more in the earlier hours. The diet may be of the ordinary variety, so long as there is no gastro-intestinal disturbance. Dr. Still advises the restriction of sugar, fruit, and potato, when there are signs of carbohydrate dyspepsia, and always disallows tea and coffee, because of their exciting effect on the higher centres. Open-air life and freedom from mental strain, especially the stress which a board school education so often entails, are to be advocated in all cases. The child must not be punished for the bed-wetting habit, but may be advised to try to wake up before it wets the bed.

Of the drugs which act beneficially in restoring the nervous control we may limit our attention to two or three. Many others have been lauded at various times, but have not stood the test of acting consistently well over a large number of cases. Belladonna or atropine holds by far the best record, and it does not seem to matter much in which form the drug is given. As, however, the tincture of belladonna is so much more easily prescribed, it is usually preferred to atropine sulphate. All authorities are unanimous that the drug must be given in large doses and for a prolonged period. It is necessary to begin with moderate doses (say 5 minims of the tincture t.d.s.) for a child aged 5 years, and to increase the dose by 2 or 3 minims every week, until the limit of toleration is reached. When the child begins to show signs of intolerance, such as a dry sore throat, blurred vision, marked flushing or delirium, the dose should be reduced by 2 or 3 minims and kept at this level, provided always that toxic symptoms disappear with the reduction. Should incontinence stop before intolerance is reached, the dosage is kept at, or raised slightly above, that at which the incontinence stopped, for a week or ten days, and then gradually reduced each week, until the initial dose is again reached. Other drugs which are likely to be of value are bromide of potassium and strychnine. but these will be tried only when belladonna has proved ineffective.

Hypnotism is a mode of treatment which has never been favourably received in this country, although in France it is credited by one author at least (Voisin) as being successful in some cases.

I will here draw my remarks to a conclusion, hoping that the subject may receive more critical comment from my audience than I myself have brought to bear upon it.

DISCUSSION.

Dr. G. A. SUTHERLAND remarked that it was a pity the reading of the paper was compressed, because he was hoping to hear some fresh method of treatment propounded. Not that he would be particularly hopeful even if Dr. Robson had done so, for he had lived through several new forms of treatment for the condition, all of which had the same ultimate merit, or lack of it. If he had been writing a paper on the subject he would have followed much the same line of thought as the author had. He gave a list of possible local lesions which might be present, and which must always be considered in connexion with neuroses. He then passed to the important class of case where no definite local cause could be found, and then gave his view of what was at the back of the condition, and what it was the physician had to treat. The author found that there was some disturbance in the central nervous system, and he (the speaker) agreed with him. The defect of the nervous system did not take any one definite form. Reference had been made to subjects of this condition who were dull and lethargic; and another class were those who were very bright and neurotic. There was the child in general lowness of health, associated often with defective nerve energy. He considered this was due to delay in development of the nervous system. One met with incontinence in children who had definite mental defects, and who were the subjects of more or less gross brain disease. But there was a class in whom the brain did not seem to have developed at the ordinary rate. Prognosis depended on what the growth of the brain was likely to be. He did not think it followed that because a child up to a certain age might be backward mentally and suffer from incontinence, it had bad prospects for the future. The bright, excitable child he found it more difficult to treat successfully. Rheumatism had also been mentioned, and that was a distinct example of disturbance of the cerebral function directly induced by a poison in the system. That class seemed to be more amenable to treatment than any other. It had been very interesting to him to note the various treatments for the condition as they came out, and which died out in a few months. The question of employing atropine or belladonna was a vexed one, but in some cases he did not doubt it was a valuable drug. He did not think any guide was yet known as to what particular cases it was suitable for.

Dr. LEONARD GUTHRIE remarked that the subject was so wide that it was difficult to know where to begin in discussing it. Dr. Sutherland had referred to two types of children in whom the condition was found, but he (the speaker) thought it was quite as common to find nocturnal enuresis in children who were quite healthy in every way and who were not neurotic; and yet they were in the habit of wetting the bed at night. It was difficult to ascribe a cause, but he thought he had noticed that it might be strictly described as hereditary. He remembered the case of a healthy schoolboy, who had nothing the matter with him except this unfortunate habit; and the father said he was exactly the same until he reached the age of 18 years. He believed some of the cases were amenable to suggestion; there seemed a likelihood that the brain might be cultivated to exercise a control over the bladder during sleep. Still, it would be difficult to choose the particular cases in which one could carry that out. It was remarked in the paper that diurnal enuresis alone was scarcely ever known to occur. He agreed that it was very rare, but still it did occur. He met fairly recently with a rather painful case in a young school girl, aged 15 years, well developed physically, but rather backward and childish mentally. She was continually wetting herself by day, but no such accident had been known to occur at night. He spoke to her about it and asked her whether she could not control it. She replied that she could, and when asked further why she did not, she said she supposed she did not try hard enough. Both persuasion and punishment had been tried, but she was imperturbable, and it did not seem that any reason why she should stop it had ever occurred to her. Yet the habit made her a nuisance to the whole school. With regard to acquired enuresis, both by day and night, it was nearly always due to bacteriuria, usually the *Bacillus coli*. Those cases were treated in days gone by with alkalies, but without knowing exactly why. He agreed with Dr. Sutherland as to the disadvantages of pressing the atropine treatment; he had seen it produce far more discomfort than benefit.

Dr. ROBSON, in reply, said there were so many different treatments for the defect he had referred to that it would require a long time to deal with them. He thought one ought nowadays to give more attention to the cases in which there was hypothyroidism; they seemed to be a class of cases apart. And although these patients might be mentally unstable, or have an irritable nervous system, perhaps the hypothyroidism might be the cause of their instability.

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Clinical Section.

October 11, 1912.

Sir WM. OSLER, Bt., F.R.S., President of the Section, in the Chair.

Case of Coarctation of the Aorta.

By A. M. GOSSAGE, M.D.

THE patient, a woman, aged 53, the mother of thirteen children, twelve alive and well, was shown to the Clinical Section by Dr. Langmead on May 31 last. She is again brought before the Section because the diagnosis of coarctation of the aorta, advocated then by Dr. Parkes Weber, is now quite clear, and because there are certain important alterations in the physical signs from those described by Dr. Langmead.

Her complaint has been mainly of pain in the right side of the abdomen, apparently arising from a movable right kidney. There is a history of one or two slight attacks of hæmatemesis, but except for this she had always been healthy until three years ago, though she had always suffered from cold extremities. Her large family and a negative Wassermann reaction preclude the idea of any past infection with syphilis (Dr. Langmead also obtained a negative Wassermann reaction). In August, 1909, she had an attack of right hemiplegia, but in the succeeding six months the paralysis almost disappeared. More recently her breath had been a little short, but beyond this she had had no symptoms referable to the chest.

She is a rather thin woman, who looks older than her stated age. Under the skin down the whole of the back of the thorax there are numerous tortuous arteries, and some are also to be found in the axillæ and over the front of the thorax, but no enlarged arteries can be felt over the abdomen. The heart is somewhat enlarged, apex in the fifth space 1 in. outside the nipple, and the beat is rather forcible. Some pulsation can be felt over the upper part of the sternum, and the percussion note is here somewhat impaired, but the X-ray examination failed to reveal any dilatation of the aorta. Pulsation can be felt in the episternal notch, and the subclavians are abnormally high in the neck. The axillary, brachial and radial pulses are normal except for some slight arterio-sclerosis, but only very faint pulsation can be felt in the abdominal aorta and femorals. A systolic murmur can be heard over the upper part of the sternum, and more loudly at the back on both sides above a line drawn between the two scapular spines: here, too, a thrill can be felt on both sides. The murmur can also be heard more faintly over the next eight or nine vertebræ below this level.

A number of skiagrams in various positions were kindly taken for me by Dr. Worrall. Those from front to back showed some peribronchial fibrosis and a rounded shadow on the left side, evidently due to something in the posterior part of the thorax, which was apparently not connected with the aorta. This shadow, found by Dr. Langmead, was considered by him to be due to an aneurysm of an intercostal artery. Screen examination from side to side and a photograph showed no shadow behind between the heart and the vertebræ, and in the upper part of the photograph it can be seen that the aorta ends at about the third dorsal vertebra, the obstruction being apparently complete (*see* figure).

There are several interesting points connected with this case. Coarctation of the aorta of the adult type arises just after birth at the time of the closure of the ductus arteriosus, and to this type the case would certainly seem at the present time to conform. The fact that Dr. Langmead[1] last May found no enlargement of the heart and normal pulsation of the abdominal aorta and femorals, raises the question whether the condition has arisen recently and whether there has been progressive development of the atresia since her last appearance here. Against this view is the extreme rarity of the development of atresia of the aorta in adult life, and the absence of any sufficient cause in this patient for the onset of such a condition. It is important to note the strong evidence against there having been any syphilitic infection.

It is difficult to decide whether the obstruction in the aorta is, or is not, complete. The pulsation in the abdominal aorta and in the femorals, particularly if this was normal last May, suggests that there is only a narrowing of the aorta, and this view is borne out by the marked systolic murmur heard over the upper part of the back and the accompanying systolic thrill. Further, the murmur can be heard over the spinous processes as low as the last thoracic vertebra. The complete absence of a shadow behind the heart in the skiagram, while it proves the absence of a normal vessel in this situation, does not preclude the presence of a small descending thoracic aorta, such as would probably exist even if the obstruction were complete.

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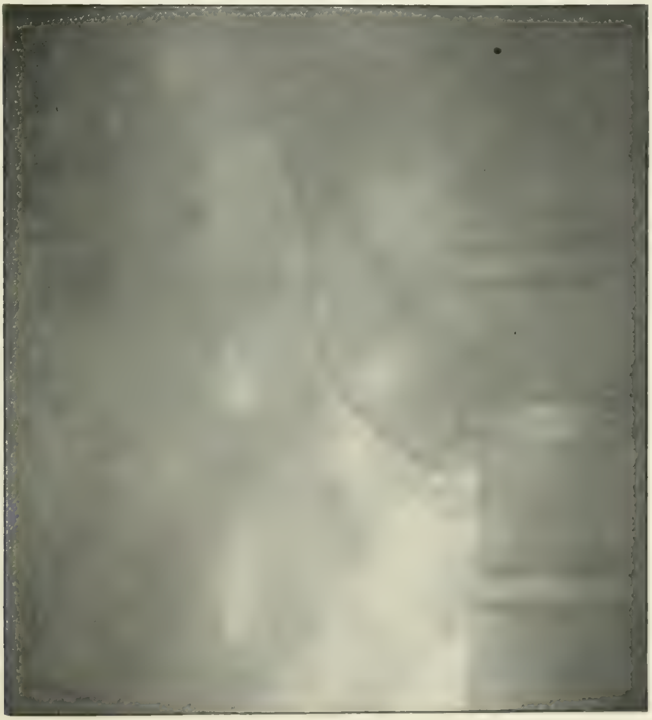
[1] Langmead, *Proceedings*, 1912, v (Clin. Sect.), p. 194.



Skiagram from side to side with plate on left side of chest. Shows in front the heart and ascending aorta; behind the spine. Above, just in front of spine, is the descending thoracic aorta, which apparently ends at the third dorsal vertebra. Between the heart and the spine there is no shadow.

No doubt in these cases the blood-pressure is always high, so as to overcome the difficulty of getting the blood through the narrow arterial channels. In this patient the maximal pressure was from 200 to 225 mm. of Hg. and the minimal 100 mm. (Oliver's auditory method), but it has to be remembered that there is some sclerosis of the radials to account for the rise in pressure. In Parkes Weber's and Price's case,[1] where there was considerable vascular degeneration, the maximum pressure was 230 to 290 mm., and in Moon's case,[2] in which there was chronic nephritis, the pressure was 180 mm. It is regrettable that the pressure was not taken in this last patient, when he was under my care as an out-patient before the onset of the kidney trouble, and shown by me to this Section.[3]

The cause of rounded shadow on the left side of the chest, which seems to cause no symptoms, is very obscure. To the writer it seems more like an enlarged gland than an aneurysm of an intercostal artery.

DISCUSSION.

Dr. R. O. MOON said he did not doubt that the case under his care a year ago, which he described in the *Lancet*, was exactly the same, pathologically, as that now shown—i.e., it was of the congenital adult type. The infantile type was generally associated with other cardiac lesions, and proved fatal at a very early age; whereas the adult type, though at birth there was obliteration of the ductus arteriosus, was compatible with a fairly long life. He learned that Dr. Gossage had seen his (Dr. Moon's) case two years previously at his out-patient department, and he then correctly diagnosed it as coarctation of the aorta. Two years later that diagnosis was verified at autopsy. Dr. Russell Wells did the autopsy, and he might say what he found. His own case might be said to be normal in its abnormality; the sex was male, which was usual, the ductus arteriosus was not patent, and death occurred at about the age usual in these cases—i.e., between 20 and 40. The patient was undersized, being of about the size of a boy aged 16, whereas his age was 20. His general aspect seemed to give the impression that he was suffering from some form of heart disease; but there were really no lesions of the valves: the constriction of the aorta at the level of the ductus arteriosus was the only lesion. His death was due to interstitial and subacute nephritis.

Dr. RUSSELL WELLS pointed out that the skiagram of the case demonstrated showed a very large space between the heart and the vertebral column. He asked if Dr. Gossage believed that there was a considerable portion of the aorta obliterated, or that it was simply that the aorta was very small, and consequently there was the open space between the aorta and the spinal column. He believed that in most of these cases the amount of aorta obliterated was very small. In Dr. Moon's case there was complete atresia of the aorta just where the ductus arteriosus joined it. There was a test-tube-like end of the aorta from both distal and proximal sides. From the distal side the aorta was pervious, and about half its normal size. Obviously an extremely good collateral circulation had been established, and the aorta from the valves to the point of obliteration was normal. The patient had very thick skull-bones. Both subclavians were nourished from the left ventricle in the normal manner, but the lower extremities were small, and the legs were shorter than normal, as also was the abdominal aorta. It was difficult to say how much smaller than normal the legs were as there was œdema. The common iliacs were not larger than normal radial arteries. The boy was in fair health until a short time before his death. These abnormalities were of great interest, and he thought every case should be put on record.

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[1] Parkes Weber and Price, *Lancet*, 1912, ii, p. 692.

[2] Moon, *Lancet*, 1912, i, p. 1531.

[3] Gossage, *Proceedings*, 1909, ii (Clin. Sect.), p. 210.

Arteriovenous Anastomosis for Gangrene due to Syphilitic Endarteritis.

By H. Morriston Davies, F.R.C.S.

This patient was shown at a meeting of the Clinical Section a year ago.[1] The history of the case is briefly as follows: R. F., a male, aged 47. He has had syphilis and has been a heavy drinker. He first noticed pain in the left foot and discoloration of the middle toe in March, 1911. The toe became gangrenous and was amputated in June and the wound was left open. At the beginning of August, when the patient came under my care, two further patches of gangrene appeared, one on the inner side of the fourth toe and one on the outer side of the second toe. The foot was cold and painful, and there was purple discoloration of the great toe, the dorsum of the foot, and round the head of the fifth metatarsal; the amputation wound was unhealed. An arteriovenous anastomosis was done in Hunter's canal on August 15, 1911; the proximal end of the superficial femoral artery being united to the peripheral end of the femoral vein and the proximal end of the vein and distal end of the artery being ligatured. The pain and discoloration disappeared after the operation, the foot became warm, the gangrenous patches separated off, and the wound at the base of the middle toe healed. The patient began walking a month after the operation and was discharged from the hospital after another four weeks. A week later he was readmitted under Sir J. Rose Bradford suffering from a right-sided hemiplegia and aphasia thought to be probably due to syphilitic thrombosis and to be unconnected with the anastomosis.

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[1] See *Proceedings*, 1912, v, p. 25.

The patient has slowly recovered from this attack and has been able to walk with the help of a stick for some months. The condition of the left foot has remained good. There is now (almost fourteen months after the operation) no pain, discoloration, or œdema. The patient had a chilblain on the great toe of the left foot last winter, but that cleared up completely.

DISCUSSION.

Mr. MORRISTON DAVIES said he thought the case could be regarded as presenting evidence of the value of arteriovenous anastomosis, especially as it was now fourteen months since the operation and the initial satisfactory improvement had been maintained. He took the opportunity of again showing this case as, so far as he could trace in the literature, the subsequent history of those cases in which an arteriovenous anastomosis had been beneficial for the first three or four months had not been recorded. The opponents of the operation might, of course, say that, as gangrene did sometimes clear up spontaneously, the improvement in the present case was of this nature; but the suddenness with which the pain disappeared and the foot became warm after the operation, the rapid disappearance of the gangrenous patches and return to normal of the discoloured areas, could only be regarded as directly due to the anastomosis. The arteries in both limbs were very degenerate, and the right hemiplegia prevented the man from working, but he was able to walk, and even in the evening after being up and about all day there was no œdema of the left foot.

Dr. F. PARKES WEBER said that in cases of threatened gangrene from obstruction in chronic non-syphilitic arteritis obliterans, the prognosis was not always entirely bad without operation. In December, 1907, he showed a case[1] at the Clinical Section in which the tendency of most surgeons would have been towards amputation, but the patient was still getting about in much the same condition, and he had now less pain than formerly, but he had had attacks of superficial phlebitis, and once or twice ischæmic ulceration on the affected foot, which healed very slowly. In such chronic cases Nature made a great and prolonged effort to establish a *capillary* collateral circulation, and that accounted for the hyperæmia and turgidity of the affected foot when it was allowed to hang down, a condition which had been confused with "erythromelalgia" of nervous origin.

The PRESIDENT (Sir Wm. Osler) said it must be remembered that the veins themselves might be involved, as in the remarkable series of cases record by Leo Buerger in Russian Jews, among whom this type of gangrene was common.

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[1] *Proceedings*, 1908, i (Clin. Sect.), p. 44; *see* also F. P. Weber, "Arteritis Obliterans of the Lower Extremity with Intermittent Claudication (Angina Cruris)," *Lancet*, 1908, i, p. 152

Unusual Rashes in Two Brothers.

By A. E. Gow, M.D.

(For W. P. Herringham, M.D.)

The patients are two brothers, H. and E. L., aged 5 and 2 respectively, now in St. Bartholomew's Hospital under the care of Dr. Herringham. The younger was exhibited, by Dr. Adamson, before the Dermatological Section of this Society in May of this year.[1] Both children, from a few months of age, had dusky hands and feet, and an eruption identical in character with that now present on the younger. This eruption has become, however, in the elder boy, much less marked during the past few months, and with its subsidence the interphalangeal joints, which were previously swollen, have become red and painful. The character of the eruption, as seen at present in the younger child, is briefly as follows: On the forearms, hands, and legs are papules about 3 mm. in diameter, reddish-brown in colour; papules, 5 mm. in diameter with black crusts in centre; circular, crateriform areas 10 mm. to 30 mm. in diameter, with raised, reddened margins and a large central scar; several healed lesions resembling vaccination marks. The papule apparently evolves through its stages in from two to three weeks. The elder child has never been able to walk properly, probably owing to genu valgum, which has become worse during the last year, and he is stated to have lost flesh during this time. There is a fusiform peri-articular swelling on all the fingers. A skiagram shows no alteration in the bones. On admission there were palish red, slightly raised spots, somewhat resembling patches of lichen planus, but rosier, on the feet. On the hands there were more of these spots; one on the right metacarpus shows the same loss of subcutaneous tissue as do those of his brother (E. L.). Spots tend to come out on the metacarpophalangeal and proximal interphalangeal joints. There is no muscular wasting, the nails are natural, and the tendon reflexes brisk. There are a few palpable glands in the anterior triangles of the neck. The spleen is not palpable, and there is no fever. Since he has been in the hospital the peri-articular swelling has largely subsided.

It was suspected that the lesions were of the nature of a tuberculide. Von Pirquet's cuti-reaction is negative, but after injection of 0·0002 c.c., 0·0005 and 0·001 c.c. of old tuberculin there was a general reaction with rise of temperature to 101·4° F. in the case of the younger boy. There were, however, both previously and subsequently, unexplained rises of temperature. The elder showed no reaction to tuberculin, even with a dose of 0·004 c.c. The Wassermann reaction is negative in each case, and the blood shows no cytological change. As the result of treating the younger brother with tuberculin (T.R.) there is local reaction in the spots, and many heal without passing through the scab stage, the resulting scar being not nearly so marked.

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[1] *Proceedings*, 1912, v (Derm. Sect.), p. 131.

DISCUSSION.

Dr. **JAMES GALLOWAY** said that he supposed that in the very loose nomenclature used at present the lesion from which these two children suffered might be called by some a "tuberculide." But he wished to protest vigorously against the use of this name, possessing so obvious a suggestion, as applied to conditions of localized necrosis of the skin. In these very cases, indeed, the looseness in the use of the term was very well borne out by some one who suggested that they were "a sort of a tuberculide"! He was of opinion that such a term should not be used without very good reason, and it must be perfectly clear that in any case there was no such close connexion between the lesions known as "tuberculides" and tuberculosis as with the lesions called "syphilides" and syphilis. In large numbers of the so-called tuberculides careful and prolonged examination had failed to establish the existence of tubercle in any recognizable form in the sufferers, just as in the case of the two patients brought before the Society. Indeed, the only evidence seemed to be of the nature of the well-known statement that everyone had a little tuberculosis if they lived long enough. It was true, just as in the case of these two patients, that there did occur fairly frequently cases of localized necrosis of the skin, sometimes accompanied by a process of softening with suppuration, and in other cases without these two latter complications. In his own experience he had been much impressed with the fact that, in the case of many of the patients so suffering, there were obvious defects in the circulation, especially of the cutaneous circulation; and in some of the cases it seemed to be proved fairly conclusively that the inflammatory or necrotic lesions had their origin in close association with the small vessels of the cutis or subcutaneous tissue. It was well known that degenerative changes occurred in the arterioles, venules, and capillaries in cases where marked degeneration of the larger vessels might not be noticed. He felt much more inclined to consider that these minute areas of necrosis originated in defects of the circulation produced by damage of the small vessels, ending in thrombosis and loss of nutrition of the small areas supplied or drained by the disorganized vessels. Cases did occur in which the inflammatory exudation assumed characters closely resembling the structure of a tuberculous granuloma, but it should be remembered that the mere occurrence of giant cells in an inflammatory exudation did not necessarily prove the presence of tubercle. He was interested to observe in Dr. Gow's cases that both children suffered from defective and stagnating circulation in the periphery. The occurrence of the small areas of necrosis, as proved by histological examination to be in close relation with the blood-vessels and their distribution, corresponding to the areas of vascular supply of the cutis, suggested that these cases also were connected with defects in the cutaneous circulation rather than of a specific tuberculous infection. A good deal was already known as to the degenerative changes in the larger blood-vessels, but information was only now beginning to be collected as to the degenerative changes in the smaller vessels—the arterioles and venules. The study of the necrosing lesions of the cutaneous structures had already given information, and would no doubt help in the future towards the elucidation of this problem.

Dr. F. PARKES WEBER said that, as Dr. Galloway had somewhere pointed out, when necrosis of the skin occurred in the class of cases to which he referred, the favourite spots seemed to be behind the internal and external malleoli. Those might be the only parts where there was a tendency for indolent ulcers to occur.

Case illustrating Circulatory Disturbance with Cervical Rib.

By Sir WM. OSLER, Bt., M.D., F.R.S.

H. C., AGED 20, was admitted to the Radcliffe Infirmary on September 20, 1912, under Dr. Collier, to whom I am indebted for the permission to show the case. A healthy girl with a good family history. In February, 1912, she fell and bruised the right elbow, apparently not a serious injury. About the end of May she noticed that after using the right arm it became darker in colour, the hand swelled, and the veins in the neck became distended. She looks well; physical examination of the chest and abdomen negative; no enlargement of the glands. The right pupil is a little larger than the left. At rest the right arm looks natural, but measures above the elbow ¾ in. more than the left. There is no atrophy of the muscles of the hand; sensations are perfect. After using the muscles of the right hand for a few minutes the following changes occur: The skin reddens, at first on the inner side above the elbow, then the redness becomes general; the arm swells, increasing ¼ in. by measurement; the pulse in the right radial becomes smaller, the blood-pressure falls from 115 mm. of mercury to 90 (it is normally a little less than on the left side), the veins in the neck become enlarged, particularly the external jugular, and there is prominent venous swelling above the inner end of the clavicle. Continuing the exertion, the arm feels numb and dead and she has to rest.

The X-ray pictures show cervical rib on both sides, the right a little longer, both of moderate size. The case belongs to a group of cases of cervical rib which I have described,[1] in which the arm is normal when at rest, but on exertion becomes swollen and livid, and muscular effort has to cease—features resembling the condition known as intermittent claudication. In many cases of cervical rib the subclavian has been compressed in the angle between the rib and the scalenus anticus. In the present instance the rib looks short, but it is possible that there may be a cartilaginous extension. When at rest, and with slight muscular effort, sufficient blood reaches the limb, but the demand for increased blood following upon exertion is not met, and there is stiffness with numbness and vascular changes. Cases have been reported suggesting Raynaud's disease, and Keen states that there are at least seven instances of local gangrene of the finger associated with cervical rib.

DISCUSSION.

Sir WM. OSLER said that cervical rib disturbances could be arranged into three groups—motor, sensory, and vasomotor, or vascular. In the vascular group, of which there were some instances recorded in literature, associated with cervical rib on one side, there had been symptoms of Raynaud's disease, and even superficial gangrene. He considered that the present case belonged to a vascular group which had not been much recognized. This was the fourth case he had seen. The first he showed at the Neurological Society, Philadelphia, many years ago, that of a carpenter, whose arms were perfectly normal when at rest. But as soon as he began to use the right arm it became red, swollen, livid, and hot, and finally dropped. The second case was a woman in the out-patient department of the Johns Hopkins Hospital, with identical features. The third case he saw a few years ago, in a strong, robust woman, who had precisely the same symptoms.

Dr. JAMES GALLOWAY said that the problem to which Sir William Osler had drawn attention—namely, why the symptoms due to the presence of a cervical rib should make their appearance at some definite time, while the malformation had existed since birth—had also presented difficulties to him on certain occasions recently. Earlier in the day he had seen a woman, aged about 52, who had come to him some time previously with symptoms of tinglings, stiffness and pain in the forearms and very obvious atrophy of the muscles of the thenar eminence of the left hand. On further examination it was shown that she possessed a pair of accessory cervical ribs. The rib on the right side was pronounced and noticeable, running well towards the front of the neck. On the left side the X-ray picture showed merely a long and easily noticeable transverse process of the seventh cervical vertebra, with a very small extension only, yet it was on this side that the symptoms were most pronounced and the atrophy of the muscles was seen. The symptoms had been noticed for about a year previous to the time when the patient sought advice. By means of rest to the arm and a course of careful massage, the painful symptoms had greatly disappeared and the muscles of the hand were larger and stronger. But the question arose, why should this woman, suffering from this congenital defect, have suffered from these symptoms for the first time only two years ago? In one or two similar cases under his observation the same question arose. In one case he thought the fact that the patient having been previously employed as a clerk and suddenly having to undertake manual labour might offer an explanation of the onset of symptoms. The unusual movement and the development of the muscles might press the vessels of the neck, or the nerves of the brachial plexus against the projecting rib, its cartilaginous extension, or the fibrous band which passed from its tip towards the sternum. In younger patients the ossification of the cartilaginous extension might afford a firm resistance against which vessels or nerves could be pressed. He was much interested to have seen the clear demonstration in this patient of the symptom described by the President, namely, of the swelling and redness of the extremity on muscular exertion.

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[1] *Amer. Journ. Med. Sci.*, Philad. and New York, 1910, n.s. cxxxix, p. 469.

Dr. F. E. Batten said he thought that many cases such as the President had described were placed in the group of occupation neuroses. Some of these cases were probably due to vasomotor disturbance occurring in relation to a prominent cervical rib, but in others no rib was present and the symptoms were due to a band attached to the transverse process. He mentioned the case of a boy, a telegraphist, who, after a short time at telegraphing, was unable to continue his work from what was regarded as telegraphist's cramp; he was therefore transferred to letter-sorting, but a few months of this work again produced a muscular disability. These cases were divisible into certain groups: first, the motor group, in which there was disability of muscle, sometimes called cramp, though there was not necessarily any cramp; secondly, there was the sensory group, in which there was a definite loss of sensation on the ulnar side of the forearm; and thirdly, there was the vasomotor group, well exemplified by the case shown by the President. He thought the motor and sensory groups were more common than the vasomotor. He asked if the President had any explanation to offer for the occurrence of the vasomotor condition.

Dr. F. Parkes Weber said the symptoms in the President's case were very decided. When the patient worked with her right arm she thereby caused distension of the veins on the right side of her neck, and then intense reddening of the outer upper part of the right arm. With Sir William Osler's case some of the cases of localized flushing (and sweating) of the skin might perhaps be compared. Dr. Weber saw a striking example in a young man who had a scar in the left parotid region, in which some nerve-fibres had evidently become entangled. Whenever he ate a meal he developed intense redness on that side of the face as if he had just received a blow there. The redness was soon followed by localized sweating.[1] There were several similar cases on record in which there had been a scar, probably from suppurating lymphatic glands, which must have involved some nerve-fibres. He asked the President what connexion he thought there was in the present case between the distension of the veins at the root of the neck and the flushing of the arm. He (Dr. Weber) did not think the intense reddening on the outside of the arm could be due to simple engorgement from venous obstruction at the root of the neck; he thought it must have a nervous origin, whereas the swelling of the cervical veins might be due to mechanical obstruction.

Mr. ELMSLIE asked if the President intended to advise removal of the cervical rib in his present patient, and if he could give the meeting the results of removal of the rib in previous cases which he had seen.

The PRESIDENT, in reply, said it was not an easy matter to give an explanation of the case. He thought it was due to pressure, and the closest analogy was borne by the cases of arterial obstruction in the lower extremities with intermittent claudication. The limb when at rest was natural, but as soon as used it flushed, and if the motion persisted, it became livid and the veins increased in size and the skin became hot. Following, was muscular disability. He had seen an instance in which the condition affected the arteries of only one leg, which when used had very much the same appearance as this patient's arm. In this girl patient the sympathetic was involved in the neck on that side, as the pupil was enlarged. He did not regard the condition as analogous to the so-called parotidean sweating, which in the only case he had seen had sweating only, without flushing. He would advise operation in this case, as there was disability interfering with her occupation.

Giant Urticaria of Five Years' Duration.

By F. PARKES WEBER, M.D.

THE patient, W. C., aged 31, is a well-nourished man, of medium size. He is subject to "swellings" of various sizes, which may affect any part of the surface of his body, and are generally accompanied by a sensation of itching. The skin over these swellings is always more or less reddened. Different parts of his trunk, extremities and head have been affected, notably the hairy scalp, and sometimes the forehead or a lower eyelid; perhaps the most disagreeable sites are the palms of his hands and the soles of his feet. The swellings are sometimes large enough for a single one to cover the whole of a shoulder or a buttock. When smaller and multiple they seem to merge into ordinary urticaria. Sometimes the swellings occur so frequently that he does not become free from them for any length of time. Muscular exertion, he thinks, favours their occurrence, and he is decidedly less subject to them when he has no work to do and is resting. Scratching his skin makes them worse. Diet apparently plays no part in inducing them. Temperature (cold bathing, &c.) seems to make little difference, but he has never tried a Turkish bath. The swellings sometimes last two or three days, but sometimes (provided that he rests) they disappear earlier; in fact, they may even vanish within twelve hours. The patient says that as yet nothing has done him any good, and that the affection is neither better nor worse than it was when it commenced about five years ago. He says that it commenced two weeks after an attack of rheumatic fever, for which he had been confined to bed for six weeks, but that the rheumatic fever had been preceded by an irritable rash of some kind all over his body. Otherwise he has enjoyed fairly good health. There is no history of any similar disease in other members of the family, as there often is in typical cases of angio-neurotic œdema, nor has the inside of the patient's mouth, or his pharynx or larynx, ever been affected. Nor is there evidence of any special irritability of the mucous membrane of the digestive tract, except that he is subject to sudden diarrhœal attacks if he drinks hot tea, &c. No "dermographia" or "factitious urticaria" can be elicited by drawing a finger-nail along the skin. Ordinary examination of the patient's blood shows nothing abnormal, no eosinophilia, &c. On June 27, 1912, before the calcium therapy was commenced, the coagulation time (by Sir A. E. Wright's tubes, at 35° C.) was about four and a half minutes. Examination (Dr. G. Dorner) of the fæces has shown nothing abnormal beyond the presence of the ova of *Trichocephalus dispar*. The urine is free from albumin and sugar. The thoracic and abdominal viscera appear healthy, except that there is a slight systolic murmur at the cardiac apex. Calcium therapy (calcium lactate) was tried from June 27 to August 29, but it has had no obvious effect in regard to the swellings. On October 8 Dr. G. R. Ward found the calcium index (method of Dr. W. Blair Bell) of patient's blood as high as 2·25. Two small intravenous injections of salvarsan (0·2 grm. in each injection) have quite recently been given, but without any decided result as yet.[1]

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[1] Dr. Weber showed the case at the Clinical Society of London on October 22, 1897, and again on January 27, 1905 (*Trans. Clin. Soc. Lond.*, 1898, xxxi, p. 277; 1905, xxxviii, p. 216); see also F. P. Weber, "Localized Flushing and Sweating of the Cheek on Eating," *Medical Press*, London, 1905, cxxxviii, p. 261.

[1] [*Supplementary Note* (November 14, 1912).—A third injection (neo-salvarsan, equivalent to 0·4 grm. of original salvarsan) was given on October 19, and since October 24 the patient has had no "swellings" of any kind (in spite of his doing work).—F. P. W.]

DISCUSSION.

Dr. BRAXTON HICKS asked whether the patient was taking calcium lactate at the time of or previous to the calcium index being estimated. If so, that reading was of no practical value. It was usual to find a low index in these conditions, and the high index in the case was possibly due to treatment and not to the condition.

Dr. PARKES WEBER replied that the patient had been taking calcium lactate before the calcium index was estimated. The exact dates would be inserted in the description of the case.

Multiple Calcification ("Calcinosis") in the Subcutaneous Tissue.

By F. PARKES WEBER, M.D.

THE patient, a German girl, A. G., aged 7, was admitted to the German Hospital on July 30, 1912, on account of the presence of a large number of hard nodules in the subcutaneous tissue of the extremities and the portions of the trunk adjoining the extremities. Most of the nodules are smaller than an average pea, but some of them, especially those on the buttocks and about the knees, are much larger, the larger nodules having apparently arisen by the coalescence of several smaller nodules. The face, head, thorax and abdomen are practically free. On the child's admission to the hospital the skin over one of the nodules was ulcerated, and the skin was inflamed and adherent over one or two others, but the nodules, as a rule, give rise to no pain or tenderness, and seem to have developed without the child being aware of their existence. The lymphatic glands in the groins and right axilla, and some in the neck, are moderately enlarged. The liver and spleen cannot be felt, and the child seems to be free from any visceral disease. The urine contains no albumin or sugar. There is no fever. Brachial systolic blood-pressure, 110 mm. Hg. Ophthalmoscopic examination (right eye) shows nothing abnormal. Blood examination (Dr. G. Leopold and Dr. H. Rosenbusch, October 8, 1912): hæmoglobin, 70 per cent.; red cells, 4,070,000 per cubic millimetre of blood; colour index, 0·9; white cells (after a meal), 11,200 per cubic millimetre of blood. The differential count of the white cells gives: polymorphonuclear neutrophiles, 65 per cent.; small lymphocytes, 27 per cent.; large lymphocytes, 2 per cent.; large mononuclears, 3 per cent.; transitionals, 2 per cent.; eosinophiles, 1 per cent.; mast cells, none in the count. The red blood corpuscles appear normal. The coagulation time (estimated by Sir A. E. Wright's tubes, at 25° C.) was about three minutes. On October 7, 1912, Dr. G. R. Ward kindly estimated the calcium index of the blood by W. Blair Bell's method, and found it to be 1·36, as against a normal average of about 0·9.

According to the history obtained, the presence of hard subcutaneous nodules was first noticed twelve months before the patient's admission to the hospital, and since these were detected many others had appeared. One little nodule near the right knee became very prominent and discharged spontaneously six weeks before admission. In regard to the patient's past history, she is said to have been subject to skin eruptions, but the chief point is that in November, 1909, she was under treatment at a fever hospital for scarlet fever and that from that time to August, 1910, she suffered successively from diphtheria, paresis of the lower extremities (diphtheritic paralysis ?), a skin eruption somewhat resembling lichen ruber, pneumonia, an abscess in the right axilla and an abscess at the back of the neck, a corneal ulcer (marantic ?) of the left eye, and left-sided otorrhœa. The corneal ulcer perforated (giving rise to considerable prolapse of the iris), but ultimately healed up again. During the latter period of this succession of illnesses the patient was in the German Hospital. She left the hospital on August 1, 1910, but was readmitted in September of the same year for temporary fever of uncertain origin. Since that time the child's condition seems to have remained satisfactory until the appearance of the subcutaneous nodules first attracted the parents' attention about August, 1911. In regard to the family history there is little to be said. The father, a traveller, is a healthy-looking man, who has never suffered from any venereal disease. The mother, aged 35, subject to epilepsy, has had no miscarriages or abortions. Of the patient's four brothers and one sister, one brother had rheumatic fever and another had tuberculous disease of the left ankle-joint (the others said to be healthy).

That the subcutaneous nodules in the present case consist largely of calcareous material has been proved both by Röntgen-ray examination and by chemical analysis. The skiagrams (Dr. G. Dorner) show how numerous these calcareous infiltrations are (there must be over a hundred of them in the patient's limbs, *see* figs. 1 to 3), and suggest likewise that the larger nodules are formed by the coalescence of smaller foci. The nodules occasionally become inflamed and softened, the skin over them becomes adherent, and if they were left to themselves thin purulent matter mixed with calcareous debris would doubtless be gradually extruded through a fistulous opening in the skin. Two such softened nodules have been excised and examined (Dr. G. Dorner). The gritty material was found to consist of calcium carbonate and calcium phosphate. (The addition of weak sulphuric acid under the microscope was followed by the separation of bubbles of carbonic acid gas and by the formation of acicular crystals of calcium sulphate; the ammonium molybdate test showed the presence of phosphates.) No tubercle bacilli or other microbes could be detected in the contents of the nodules. Culture tubes of glycerine-agar, glucose-agar, and egg-agar were inoculated, but remained sterile. Another concretion was examined for uric acid by the murexide test, but with negative result. Microscopical sections of a softening nodule (*see* fig. 4) show that the nodules consist of a sponge-like matrix of subcutaneous connective tissue in the interstices of which the granular particles of lime salts are embedded. Thus the accompanying illustration, from a micro-

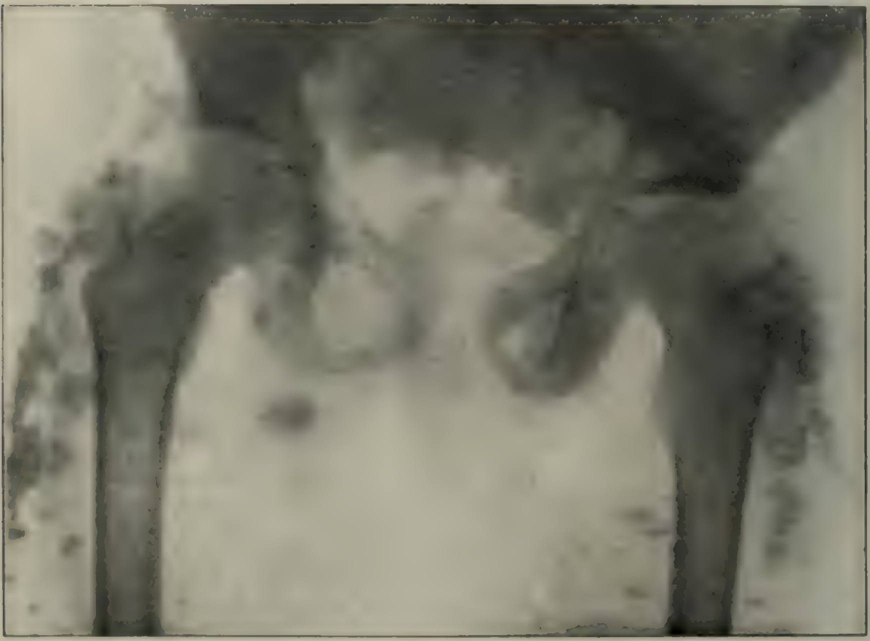

FIG. 1.

Skiagram of the pelvis and upper part of thighs to show the numerous subcutaneous calcareous concretions, especially about the hips.

scopical section stained with hæmatoxylin, shows many structureless islands (necrotic debris), from which the lime salts have been removed (by the weak hydrochloric acid ordinarily used for decalcifying), embedded in a spongy framework of connective tissue, which is undergoing inflammatory small-cell infiltration (inflammatory softening) previous to the process of "breaking down" and discharging. The nodule, in fact, consists of granules of calcareous material held together by a fibrous framework, and there is nothing of the complete (stone-like)

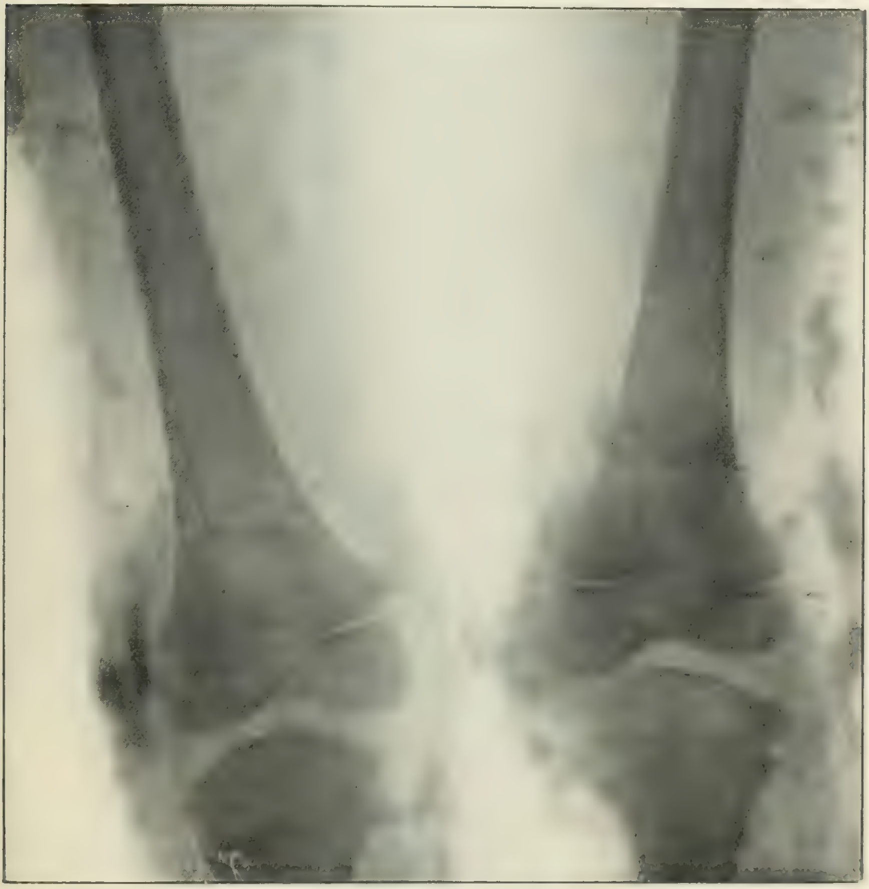

Fig. 2.

Skiagram of lower limbs to show the numerous subcutaneous calcareous concretions, especially about the knees.

calcification met with in calcified tuberculous (caseous) foci and in calcified gummata. The diffuseness of the process of calcification explains why, in this kind of calcification, the concretions cannot be "shelled out" when an attempt is made to remove them.

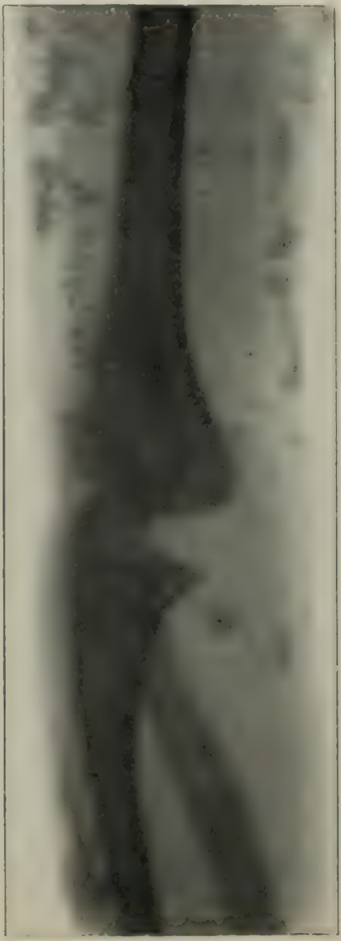

Fig. 3.

Skiagram of upper extremity about the elbow to show subcutaneous calcareous concretions.

Further skiagrams (kindly taken by Dr. Finzi) of the abdomen, thorax and axillæ give no evidence of calcification in the axillary, intra-thoracic, or intra-abdominal lymphatic glands, or in any of the thoracic or abdominal viscera.

In regard to the ætiology of the calcareous nodules, syphilis and tuberculosis may be practically excluded by reason of the various above-mentioned data. Moreover, both Wassermann's reaction for syphilis (tried at the Lister Institute) and von Pirquet's cuti-reaction for tuberculosis gave a negative result ; the complete absence of fever is likewise an important point. The case is not at all like one of myositis ossificans, for the muscles (at all events as yet) seem not to be affected ; there is no abnormal formation of true bone ; and the inflammatory softening of the calcareous nodules is different to anything which occurs in myositis ossificans. Moreover, the child has no shortness (microdactyly) of the great toes, such as has been recorded in several cases of myositis ossificans. The scattered literature of the subject shows, however, that the child's condition can be regarded as one of a definite disease, as

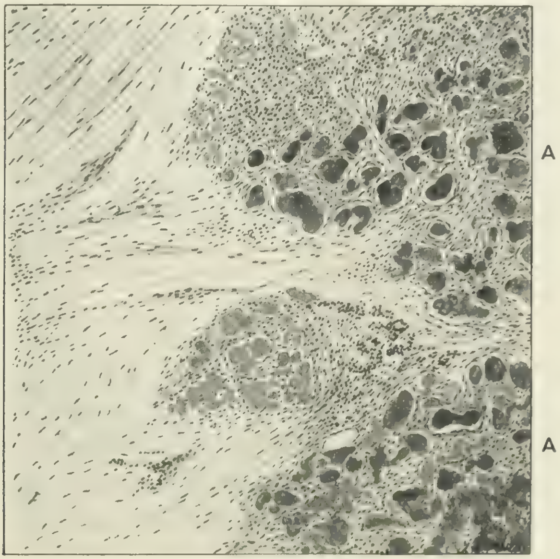

FIG. 4.

Part of the edge of an inflamed calcareous nodule from the right forearm. Drawn from a decalcified microscopical section, stained with hæmatoxylin ; magnified 55 times. On the right of the picture are variously shaped islands (**A**) of structureless material, apparently the necrotic debris marking the site of minute concretions, the lime salts having been extracted by the weak hydrochloric acid used for decalcification. These decalcified particles are contained in a meshwork of connective tissue and inflammatory small-cell infiltration. On the left of the picture is subcutaneous connective tissue.

distinct at least as myositis ossificans and many other "diseases." "Calcinosis" is probably the most convenient term to be used for the disease in question, and more advanced cases have been described under the headings "Calcinosis interstitialis," and "Calcinosis universalis."[1] Apparently the chief danger is the risk of septic infection associated with the "breaking down" and discharge of the calcareous nodules. There is a condition, occurring in rather older subjects, which possibly represents a more chronic and less severe variety of the same disease. In that condition the calcareous deposits occur chiefly in the subcutaneous cushions of the finger-tips and at the elbows, about the olecranon, and sometimes in the toes. Such cases have been demonstrated in England by Scholefield and Weber[2] and by Haldin Davis[3]; and quite recently by W. K. Hunter at the Glasgow Medico-Chirurgical Society (October 4, 1912); the patients are all subject to Raynaud's phenomena and most of them gradually develop a sclerodermatous change, chiefly in the fingers ("sclerodactylia.") Sir William Osler has met with two such cases of sclerodermia associated with subcutaneous calcification.

A Case of a Boy, aged 7, showing (*a*) Double-jointedness, (*b*) Dermatolysis ("Elastic Skin") with great Friability of the Skin and excessive Tendency to Bruising, and (*c*) Multiple Subcutaneous Tumours on the Limbs (? Fibromata, ? Neuromata).

By H. Batty Shaw, M.D., and Percy Hopkins.

B. M. is the elder of two children, his sister being quite normal. Before the birth of this child the mother had had one miscarriage. He was born at the eighth month, and was noticed to be peculiar; his bones seemed very soft and "jelly-like," and he looked "like a little

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[1] See especially the recent paper by Max Versé, "Ueber Calcinosis universalis," *Ziegler's Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1912, liii, p. 212.

[2] R. E. Scholefield and F. Parkes Weber, "A Case of Sclerodactylia with Subcutaneous Calcareous Concretions," *Brit. Journ. Derm.*, Lond., 1911, xxiii, p. 276; also *Proc. Roy. Soc. Med.*, 1912, v (Derm. Sect.), p. 124.

[3] Haldin Davis, "Case of Raynaud's Disease associated with Calcareous Degeneration," *Proc. Roy. Soc. Med.*, 1912, v (Derm. Sect.), p. 99.

Chinaman." It was not thought at first that he was alive, and he was put aside as stillborn—eventually it was found he was living. He was breast-fed for three weeks only, and subsequently was fed on peptonized milk. Meat was first given at the age of 18 months. He began to talk at the usual time, but walking was delayed. Squint has been observed since the age of 3. According to the mother, the anterior fontanelle did not close until the age of 5. The mother's confinement was unattended with any difficulties, but she suffered from "blood-poisoning" shortly afterwards.

Advice was sought particularly with regard to a slight loss of weight, and because of the tendencies to bruise easily and to rupture of the skin on falling.

The boy is very intelligent, but rather old-fashioned—the latter probably being due to the fact that he cannot go to school with other children owing to the friability of the skin. The head is of normal size; there is an alternating internal strabismus and well-marked encanthus; the teeth are a little irregular. Nothing abnormal can be demonstrated in the nervous system. The joints are very loose, the child taking particular pleasure in forming circles by locking the index and middle finger of each hand. He is somewhat pigeon-chested, and there is marked lateral curvature of the dorsal spine, with the convexity to the left. Dr. Stanley Melville showed by radiography that the lateral curvature was independent of any deformity of the vertebræ; the bones generally were found to be more translucent than usual, but the epiphyseal development was quite normal. The skin is soft, and can be drawn readily from the limbs and body, as in the condition known as dermatolysis or "elastic skin." Numerous small subcutaneous nodules can be defined in the limbs, from the size of a small millet-seed to that of half a pea. They are not painful, and are not restricted to the neighbourhood of the joints, nor apparently to the large nerve-trunks. Bruising is very readily produced even by moderate pressure; the mere holding of one upper arm on the occasion of the first visit was followed by several well-marked bruises observed a few days later. The fronts of the knees and the chin bear evidence in the form of scars of the ease with which quite small falls cause the skin to rupture. Beyond rickets, there is little to recognize which would explain the features of his condition. So far as either parent knows, there is no other example of such a condition in any member of their respective ascendants or collateral relatives.

DISCUSSION.

Dr. BATTY SHAW said that what was remarkable was the ease with which the skin over the knees and elbows ruptured on the occasion of a fall, such rupture occurring as a result of the tension developed in the skin over the extensor surfaces of the joint and not from direct injury. He did not know what was the meaning of double-jointedness, but everyone presumed that it was a want of fibrous tissue about the joints, and this case was an extreme example of the results of such deficiency.

Dr. GALLOWAY said that such cases as Dr. Batty Shaw had shown occurred very rarely. The exact nature of the change which occurred in so-called cases of "hyper-elastic" skin—cutis laxa, as it was sometimes named—were not well understood. Very little change of degenerative nature seemed to occur in the elastic tissue fibres—tissues having little to do with the extensibility of the skin—but the white connective tissue fibres appeared to be, in some cases at any rate, less dense than usual. The various structures of the skin, including the muscles, seemed to be more elongated than was normal. Perhaps the chief histological observations in such cases have been carried out in a well-known case of an "elastic-skin man," in whom the skin was so extensile that it could be pulled well out from the body and when freed returned to its normal position with an actual snap. This patient had been exhibited in various parts of the world as a curiosity.[1] Minor degrees of the condition were perhaps not very uncommon. The structure of the numerous little tumours which existed in this boy's skin would be best understood after histological examination, and he hoped that Dr. Batty Shaw might be able to obtain a piece of tissue for this purpose. The occurrence of such tumours in cases of this form of easily stretched skin had been previously noted,[2] but was of very rare occurrence.

Dr. F. PARKES WEBER said that all the little nodules in the present case seemed to be perfectly spherical and elastic (like gelatine capsules), and could be freely moved in the subcutaneous tissue. He hoped Dr. Batty Shaw would examine one of the nodules microscopically. He referred to a similar case shown by Sir Malcolm Morris on May 9, 1900, at a meeting of the old Dermatological Society of London. Sir Malcolm Morris's case was that of a boy, aged 14, with a so-called "elastic skin," and with a number of small spherical bodies, which could be felt to be freely movable in the subcutaneous tissue of his limbs. The boy could over-extend the joints of his fingers, &c. He suffered from a tendency to ecchymoses about the knees. Sir Malcolm Morris exhibited microscopic preparations of the elastic skin and of the minute subcutaneous tumours. The bodies in question seemed to consist of a spherical fibrous outer portion (in which was possibly a zone of calcification) and a fluid interior portion.

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[1] Unna and Walker, "Histo-pathology of the Diseases of the Skin," Edinb., 1896, p. 981.

[2] Malcolm Morris, *Brit. Journ. Derm.*, 1900, xii, p. 209.

Abdomino-perineal Excision of the Rectum; Transverse Colostomy.

By Lawrie H. McGavin, F.R.C.S.

Patient is a man, aged 57, and was admitted with carcinoma of the second part of the rectum and having much pain on defæcation. Growth was a large cauliflower mass, bleeding readily, and very slightly attached to the sacrum. Under chloroform a transverse colostomy was performed, and the abdomen being opened, both internal iliac arteries were ligated. The sigmoid colon was divided between ligatures, the end of the upper section being inverted. The end of the lower portion was carbolized, tied up in gauze, and after the bowel had been stripped into the pelvis, was returned to the pelvis and the abdomen was closed. In the lithotomy position, the anus having been closed, the bowel was removed via the perineum. A tear occurred at the site of the growth, which was here more fixed to the sacrum than had been thought. Some infection of the wound occurred, but the suppuration was confined to the superficial layers. The bowel was opened on the third day. Patient made a good recovery. Operation done eighteen months ago.

Abdomino-perineal Excision of the Rectum; Transverse Colostomy done under Spinal Analgesia and Chloroform.

By Lawrie H. McGavin, F.R.C.S.

Patient, an unmarried woman, aged 67, was admitted for carcinoma of the first and second parts of the rectum with signs of commencing obstruction. Spinal injection of stovaine-glucose solution 6 cgrm. Transverse colostomy performed by Maydl's method. Laparotomy: ligature of both internal iliac arteries. Sigmoid double ligatured, divided, and ends inverted by Lembert's suture. Bowel with mesosigmoid and glands stripped down to pelvis. At this point light chloroform anæsthesia given for half an hour, patient never being fully unconscious. Stripping completed in pelvis. Chloroform stopped and patient placed in lithotomy position. Anus closed by suture, and the whole length of rectum and lower part of sigmoid removed with growth intact. Patient made an excellent recovery, and the colostomy was completed by opening the bowel on the fourth and dividing it on the tenth day. The patient was fully conscious during the last twenty minutes of the operation and left the theatre with a pulse of 85 and no sign of shock. Operation done sixteen months ago.

DISCUSSION.

Mr. DOUGLAS DREW said he would like to refer to one or two points about the operation of abdomino-perineal excision of the rectum. Mr. McGavin was to be much congratulated on the result in this case: probably it was the only case in a patient, aged over 60, in which the operation had proved successful. Mr. Miles, who had a large experience in this class of case, said in a recent paper that in every case in which he had operated and the patient was aged over 60, there was a fatal termination. The operation done by Mr. McGavin in these two cases differed from that he had himself followed in a number of similar cases, in that Mr. McGavin made a separate colotomy opening, and ligatured the internal iliacs. His own experience was that it was unnecessary to tie the internal iliacs, and that doing so materially increased the time occupied by the operation. Moreover, there might be considerable difficulty in finding them. There was not much bleeding from the lower vessels of the rectum. It was the inferior mesenteric artery which it was important to secure in the early stages. With regard to the colotomy, in his own cases he had always divided the bowel and brought the proximal end of the bowel into the abdominal wound: he found this very satisfactory: the patient got a single rosette in the upper end of the wound. And it obviated the disadvantage of having a second abdominal incision so as to make the transverse colotomy.

Mr. MCGAVIN, in reply, said that he knew that the rule was to tie the inferior mesenteric, but the only case of three which he lost was one in which he tied this vessel in place of the iliacs. The cause of that death was, to a great extent, loss of blood. He was convinced that there was a considerable loss of blood from the pelvic vessels in cases of ligature of the inferior mesenteric, and he did not see how that could always be avoided, remembering that the internal iliac arteries give off vessels to the pelvis. The vessels were tied in less than five minutes in both the cases, and he did not see any subsequent harm from it.

Cavernous Angeioma of the Scar of an old Laparotomy in the Site of the Fixation of a Myomectomy Stump.

By Lawrie H. McGavin, F.R.C.S.

Patient is a married woman, aged 56. Twenty years ago myomectomy performed, the stump of the tumour being fixed in the upper angle of the laparotomy wound. Ventral hernia developed soon after operation and has gradually increased, especially owing to three confinements since operation. Repeated abscess formation in the upper part of the cicatrix seven years ago, since when this portion has become affected by increasing vascularity, and is now converted into angeiomatous tissue. The colour varies in different parts from time to time, pink and blanched areas alternating with patches of cyanosis, especially near the circumference. The tumour is tender and becomes much enlarged at times. The uterus is enlarged and can be felt to be adherent to the right side of the tumour.

Bilateral Congenital Displacement of the Upper Ends of the Radius and Ulna.

By Lawrie H. McGavin, F.R.C.S.

Patient is a female child, aged 8 months. First child, transverse presentation, mother noticed the deformity a fortnight after birth. Both arms are rotated internally, so that the thumbs point backwards, and the palmar surfaces of the hands face outward. Supination only possible through quarter of a circle with the elbows flexed. Skiagram shows transposition of the heads of the radius and ulna; the radius articulating with the trochlea and the ulna with the capitellum. No history of deformity in any of the other members of the family.

Torticollis following "Mumps" and associated with Bilateral Cervical Ribs.

By Lawrie H. McGavin, F.R.C.S.

Patient is a girl, aged 11. Three months ago she had an attack of "mumps," which her mother states lasted a fortnight and caused much swelling, not only over both parotids but "round the back of the neck." During resolution patient's head began to assume its present position, the face being tilted upwards and rotated to the left. There is no history of injury at birth; the patient has never suffered from any serious illness. There is no history of rheumatism, phthisis, &c., in the family. Mother says the child has curious "trembling fits" and is very nervous at times. Some shotty glands are felt in both sides of the neck. Skiagram shows well-marked but short cervical rib on both sides; these, however, give rise to no symptoms.

Oblique Fracture of Femur into the Knee-joint, and of both Tibiæ and Fibulæ; Arthrotomy and Union of Condyles of Femur by Screws.

By Lawrie H. McGavin, F.R.C.S.

Patient is a man, aged 48, who was struck by a swinging balk of timber. On admission he was found to have a fracture running from the centre of the right femur downwards and outwards through the outer third of the cartilaginous surface of the external condyle. The right tibia was completely comminuted in the middle third, and in the lower third of the left tibia there was an oblique fracture, slightly comminuted, with 1½ in. of shortening. The displacement of the femoral fragments was so bad that there was about 3 in. of shortening, and the lower end of the upper fragment threatened to come through the skin. The joint was tensely distended with blood. The joint was freely opened and the fracture reduced with difficulty, the condyles being united by two long screws. No drain was used. The right tibia was simply placed in a Croft's splint and the left was plated. There was some necrosis of the comminuted fragments in the latter, but there is good union. It will be noticed that the lower screw presents slightly into the joint. This, however, is causing no trouble, and will be removed before flexion of the joint.

A Case in which a Serous Cyst was evacuated from the Ramus of the Mandible.

By Lawrie H. McGavin, F.R.C.S.

Patient is a man, aged 30. Three years ago a swelling developed in his left parotid region; as it steadily increased in size it was incised in the West Indies. Patient states that nothing but blood was evacuated. It was not painful, and after operation it again increased in size. On examination the tumour was of about the size of a small tangerine orange, it was firm in consistence, and evidently deep to the masseter muscle. There was no facial paresis, although it lay in the line of the facial nerve. In the centre fluctuation could be felt; no bone crackling was detected. All teeth of upper jaw except the wisdom tooth were present; this latter had been removed a year previously. Under chloroform an incision, following the angle of the jaw, was made, and a flap containing the masseter was turned up. The mass proved to be a cyst in the upper part of the ramus of the jaw, which was expanded to a thin shell, and contained clear yellow serous fluid. The lining membrane was removed. Microscopical examination gave no indication as to the nature of the cyst.

Apparatus devised for Exerting Extension on the Fragments in Cases of Fractures in the Length of the Tibia.

By Lawrie H. McGavin, F.R.C.S.

This apparatus can be placed on the operating table, the operation of plating and screwing being carried out upon it, when, by exercise of tension obtained by the screw, the fragments have been brought into line. By its use the operation, especially in cases of old oblique fractures, is greatly facilitated, and the time of operation much reduced.

DISCUSSION.

Mr. C. H. Fagge said he was not quite sure how Mr. McGavin made use of the boot which he had shown. The commonest fracture of the lower limb was an oblique or spiral fracture at the junction of the upper two-thirds and the lower third of the tibia, and in this type there was often much overlapping. A long incision with free exposure of bone made every step of a plating operation easier and the top of the boot shown would considerably overlap the lower end of the required incision which, as a rule, he carried down to the malleolus. Personally, he had never felt the necessity of any such apparatus. The worst case of an old fracture in this position he had operated upon was in a man aged 21, with $1\frac{3}{4}$ in. overlapping. He was seen and operated on fourteen weeks after the accident and the bones placed end-to-end, so that no shortening resulted. Much of the difficulty and labour necessitating forcible traction would be done away with if operators would follow the method advocated by Mr. Arbuthnot Lane, of carrying the foot and lower fragment outwards at right angles so that the two fragments impinged one upon the other; the foot was then brought into a straight line. Old fractures of the femur in muscular men presented similar difficulties. The secret of the whole question was to educate doctors and patients to submit to operation within a few days of the accident.

Mr. McGAVIN, in reply, admitted that it might sometimes get in the way of the operation area in very low fractures, but with the alteration he had made, with straps to go over the malleoli, the advantage was enormous, and especially when the opportunities of sepsis occurring from pulling about and the displacement of towels was considered. He suggested that the reduction of a fracture in a seaman, aged 50, who had been at sea all his life and was a muscular person, was a very different matter from that in a slight and not very muscular youth. With regard to Mr. Lane's method of reducing fractures, mentioned by Mr. Fagge, in some cases where the fracture was very oblique it was impossible to obtain leverage by setting one broken end against the other, the ends persistently slipping. The apparatus exhibited did away at once with this difficulty.

Case of Carcinoma of Intestine; Resection and End-to-end Anastomosis.

By LAWRIE H. McGAVIN, F.R.C.S.

J. S., AGED 65, boiler-maker, admitted into hospital on February 3, 1910, with tumour in left lumbar region, freely movable. Patient noticed the lump three weeks before admission, when he had some abdominal pain. Three days before admission he had violent pain and vomiting, and was recommended to come into hospital. Bowels regular. Always healthy. Prolapse of rectum for forty years. Practice of going to stool at night. No history of hæmorrhage from bowel or stomach. Family history good. Condition on examination: A roundish, lobulated tumour about the size of a large orange in left lumbar region. Freely movable; alters in position with movements of patient. *Per rectum*, nothing abnormal except prolapse.

Operation (February 7, 1910). Incision made to left of mid-line above umbilicus. Tumour about the size of a coco-nut at lower border of transverse colon (near sigmoid flexure). The small intestines and the omentum were all bound up together, and it was impossible to separate them. The small intestine was resected and an end-to-end anastomosis done. The transverse colon, with the growth, was brought up through the incision and anchored with a glass rod. The upper and lower ends of incision were closed and tumour covered with dressings.

Five days later the upper end of the gut was opened. Six days afterwards the whole mass was removed with the loop of bowel. Patient was discharged on April 18, 1910, and returned to see Mr. McGavin on October 11, 1912, in perfectly good health, with no signs of fresh growth and no trouble with colotomy.

Traumatic Myositis Ossificans.

By PAUL B. ROTH, F.R.C.S.

A CARMAN, aged 54, attended hospital on September 3, 1912, stating that eleven weeks before he was kicked on the right thigh by a van horse. After the injury he was treated at a hospital for a fortnight, being given a liniment. He had done no work since. He complained of weakness in the right thigh and knee. On examination he walked with a moderate limp, and the right thigh was seen to be obviously enlarged. Occupying the middle third of the front of the right thigh was a hard, irregular, roughly rounded, flattened mass, the size of one's palm. It appeared to lie in the substance of the quadriceps extensor muscle. The skiagram taken the following day showed the presence of an ossified mass, 4½ in. long and ½ in. broad at its widest part, lying close to the femur, but in front of it, and to its outer side. The upper end of this mass appeared well defined and attached to the femur; the lower part was less definite, was in several shreds, and seemed to be lying free in the muscle. The appearances were very suggestive of a traumatic detachment of periosteum, with subsequent ossification.

As operative interference in these cases has fallen into disfavour, it was decided to treat him entirely by exercises; he now does "wing curtsey standing" and "alternate knee flexion and extension" ten times each, before and after every meal. Even in the short time he has been under treatment his condition has much improved, and now (October 11) the swelling is but a quarter of its original size.

Case of Tremor. ? Nature.

By F. E. BATTEN, M.D.

P. W., MALE, married, aged 43. Family and past history entirely negative. Venereal disease denied. In December, 1911, patient had an attack of gradual onset of swelling, pain, and redness, involving chiefly the hands, to a much less extent the feet, lasting about a month. The symptoms were constant, but with exacerbations lasting one to two days at a time. He then felt perfectly well up to August, 1912, when he had a second similar attack of about a month's duration.

Shortly after the second attack his arms and legs began to shake slightly, both at rest and on movement. The shaking gradually increased, especially on the right side, and reached its height about a month from onset. He was then unable to hold anything in his right hand, and his right leg appeared to drag. Since then the shaking has lessened somewhat, especially on the left side. He complains also of occasional painful cramps on the dorsum of the right foot.

The patient's condition on October 3, 1912, was as follows: His emotional state is decidedly exalted. He is almost constantly smiling, and takes nothing seriously. His eyes are a trifle prominent, and show slight irregular jerkings on lateral deviation to right and left. The right side of his palate hangs a little lower than the left, but both sides move equally well. The optic disks are normal. His abdomen is full and tense, and the reflexes cannot be obtained. Otherwise examination shows nothing noteworthy, except for a tremor involving all four extremities. This is very much more marked on the right, and the arm is more affected than the leg. The tremor persists during rest, and is increased by stated conditions and movement, and when the patient feels himself observed. The tremor of the right hand when at rest is coarse, wide, rapid, consisting chiefly of rhythmic lateral movements at the wrist, with accessory accompanying movements at the elbow and shoulder. The static oscillations are chiefly in a vertical axis, and may become so violent and rapid as to obscure altogether the outline of the hand. The movement tremor resembles the intention type in that the oscillations become more violent, and of wider range towards the close of the act. Reflexes: The deep reflexes are all present, and not exaggerated; the plantar reflexes have a flexor tendency.

Clinical Section.

November 8, 1912.

Sir WILLIAM OSLER, Bt., F.R.S., President of the Section, in the Chair.

A Case of Rheumatoid Arthritis.

By M. A. CASSIDY, M.D.

THIS patient, a dressmaker, aged 26, was admitted to Dr. Hector Mackenzie's ward at St. Thomas's Hospital in May, 1909, three years after the onset of rheumatoid arthritis. She had been bedridden and unable to stand for more than a year; both legs were acutely flexed on the thighs; the left knee-joint was fixed and the right allowed only a few degrees of movement. The left shoulder-joint was firmly ankylosed, and the hands presented the appearances characteristic of advanced rheumatoid arthritis.

Treatment: Radiant heat, massage, passive movements and extensions produced very little improvement, though persevered with for four months. On September 21, 1909, both knees were moved under an anæsthetic and put up in plaster. On September 30 a double-hinged splint was fitted by Mr. Hoefftcke. On December 3 she was able to walk with the help of sticks, and on January 1, 1910, she could walk without assistance. Now (November, 1912) she can run, and her walking gait would not attract attention.

DISCUSSION.

Dr. CASSIDY said he thought many similar cases drifted into infirmaries untreated when this splint might be applied with as satisfactory a result as in the case now shown. The patient was still wearing the elastic extensions. It was hoped to try her soon without the splint.

Dr. F. J. POYNTON asked if there was any difficulty in getting the limb free under an anæsthetic; there was always anxiety about such cases, particularly in old persons, because of the thinning at the ends of bones.

Dr. CASSIDY replied that the limb could not be got into anything like a good position under an anæsthetic.

Advanced Carcinoma of Epiglottis, with Involvement of Glands, treated by Operation without Laryngectomy; Operation in November, 1910; no Recurrence.

By Wilfred Trotter, M.S.

J. C., aged 49. Admitted to University College Hospital on November 3, 1910. Difficulty in breathing and swallowing had been present for nine months. There was well-marked stridor. The epiglottis was greatly enlarged by an obviously malignant growth. The upper opening of the larynx was completely obscured by the tumour, which also caused considerable obstruction of the pharynx. The growth was more extensive on the right side than on the left. In the right anterior triangle a glandular mass 1½ to 2 in. in diameter was present. It was very hard and fairly well defined, but was obviously fixed to the carotid sheath.

Operation on glands, November 5, 1910: Right side of neck dissected. Internal jugular vein and sternomastoid muscle removed with contents of triangles. One gland was adherent to the vein. During the operation laryngeal obstruction supervened and a tracheotomy was done.

Operation on primary growth, November 21, 1910: Exposure of tumour by longitudinal "transthyroid" pharyngotomy. Local excision, including epiglottis, part of the tongue and the anterior and lateral walls of larynx as far down as the vocal cords. The tongue and remains of larynx were drawn together by strong mattress sutures, the gap left by removal of the tumour being completely closed. The longitudinal wound in the pharynx was then closed. Healing was fairly rapid, and there was very little leakage from the pharynx.

In March, 1912, a few glands were removed from the left side of the neck. No evidence of disease was found in them. The patient has thus been free of recurrence for two years.

The case is an instance of the curability of advanced carcinoma of the upper opening of the larynx and shows that cure can be effected without laryngectomy.

Mr. Trotter said the patient was now in good health, and, apart from some weakness of voice, no definite abnormality remained. The points on which he would lay stress were: First, that the patient was now free from disease two years after being operated upon for carcinoma of the epiglottis, which was generally regarded as a very formidable disease, and in spite of the fact that the disease had, in this patient, reached an advanced degree. The glands in the neck were of the size of a tangerine orange, and were fixed to the carotid sheath. The primary growth was so large that during the operation upon the glands, which was undertaken first, the patient became asphyxiated, and tracheotomy was necessary. Secondly, the case showed that it was possible to cure advanced cancer of the epiglottis without removing the larynx; if, as was often agreed, two years' freedom from recurrence could be called cure. The operation for this condition had, in the past, generally included total laryngectomy, making it one of the most mutilating procedures in surgery; if it was possible to cure the disease without laryngectomy there could be no doubt that the advantage to the patient was very great. If operated upon fairly early, he thought all tumours at the upper opening of the larynx could be cured without removal of the larynx, and consequently without destroying the voice. Thirdly, it was generally supposed that if a case of epithelioma of the mouth or pharynx showed considerable disease of glands it might be looked upon as hopeless with regard to cure by operation. But that did not correspond with his experience; success or failure depended far more on the nature of the enlargement of glands than on the extent of that involvement. Even if many glands were enlarged, and they remained hard and well defined, the disease could almost always be cured by operation, even though the enlarged glands might reach nearly to the clavicle. On the other hand, if only a few glands were enlarged, but were soft and ill defined, or were surrounded by a brawny induration, he believed no operation could cure. In this case the glands were hard and well defined, although fixed to the carotid sheath. One point of pathological interest, which this patient also illustrated, was the false alarm of recurring growth in the glands which one sometimes experienced. After doing an extensive dissection of glands in the neck one found that outlying lymphatic glands on the periphery of the area operated upon were apt to become enlarged, and one was naturally inclined to consider that these were necessarily the seat of disease. He had operated upon many of these cases, however, and had been unable to find microscopical evidence of recurrence of the disease in them. Therefore he imagined that the enlarged glands were the seat of some hypertrophy.

Case of Acholuric Jaundice (Hæmolytic Anæmia).

By W. Essex Wynter, M.D.

M. W., female, aged 22, has one half-brother, aged 7, who is healthy. Her mother has had no other children, so the history does not point to congenital syphilis. She has been jaundiced from birth, the intensity varying, but being always more decided at the menstrual periods. During exacerbations the linen becomes stained and the urine very dark brown. Sometimes there is itching of the skin, and she often complains of dragging pain in the left hypochondrium, but has no vomiting. The motions are fully coloured and the urine contains urobilinogen but no bilirubin. The spleen is much enlarged and hard. The liver lies within the costal margin. Wassermann's reaction is negative. Bilirubin is present in blood serum.

Blood count: Red cells, 2,710,000; white cells, 13,000; hæmoglobin, 52 per cent.; hæmoglobin index, 0·86; nucleated red cells, 78 per cubic millimetre.

Differential white cell count: Lymphocytes, 35·4 per cent.; hyaline and transitional, 3·6 per cent.; polymorphonuclears, 59·2 per cent.; eosinophiles, 1·8 per cent.; mast cells, 0.

Fragility of red cells is represented by laking in saline solution, 0·55 per cent. as against 0·45 per cent. control. The laboratory investigation has been done by Mr. W. T. Hillier.

DISCUSSION.

Dr. ESSEX WYNTER added that the patient was sent to the hospital for splenectomy, but he was anxious to ascertain from Fellows what benefit one might expect before undertaking such a serious operation. So far he had not felt greatly encouraged to recommend it. He would also like to hear of what prospect there might be of beneficial treatment by drugs.

Dr. F. PARKES WEBER referred to a recent paper by Banti in regard to good results obtained by the help of splenectomy in cases of what Banti termed "hæmolytic splenomegaly." [1] In that paper Banti referred to various cases of anæmia and splenomegaly associated (at first or later on) with acholuric jaundice. At the meeting of the Section for the Study of Disease in Children, on October 25, 1912, Dr. C. R. Box brought forward the case of a child with chronic acholuric jaundice, anæmia, and splenomegaly, in which the anæmia was lessened after the operation of splenectomy. In Dr. Wynter's case the jaundice was said to be more decided at the menstrual periods, and in a woman whose case had come under his (Dr. Weber's) notice [2] the jaundice had been increased during every pregnancy. In one of Dr. Claude Wilson's cases death took place during pregnancy with an exacerbation of the jaundice.[3]

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[1] Guido Banti, "La Splénomégalie hémolytique," *Semaine Méd.*, Par., 1912, xxxii, p. 265.

[2] F. P. Weber and G. Dorner, "Four Cases of Congenital Acholuric, so-called Hæmolytic. Jaundice in One Family," *Lancet*, 1910, i, p. 227. (The patient in question was the one alluded to as C 1.)

[3] Claude Wilson, *Trans. Clin. Soc. Lond.*, 1890, xxiii, p. 169, and 1893, xxvi, p. 163.

Dr. GOSSAGE asked if, in the cases referred to by Dr. Weber, the hæmolytic anæmia showed a special prevalence in families, in the same way as did cases of acholuric jaundice.

The PRESIDENT (Sir Wm. Osler, Bt., F.R.S.) remarked that the majority of the subjects of acholuric jaundice had very good general health. As in Murkowski's original series, the present patient illustrated a feature in the disease which was serious—namely, recurring attacks of anæmia; and if it should turn out that removal of the spleen really cured the disease, this was just the type of case in which that operation should be practised. Professor Bastianelli told him last spring that a case of removal of the spleen in acholuric jaundice had been successfully performed in Rome.

Dr. PARKES WEBER said it would be necessary for him to refer to Banti's paper in order to answer Dr. Gossage correctly. Cases of "hæmolytic" jaundice and splenomegaly were divided into congenital and acquired cases on the Continent, by French authors. Amongst the acquired cases were some which might be of a quite different nature, in which the symptoms for a time resembled those of pernicious anæmia.[1]

Resection of Cæcum, Appendix, Ileocæcal Valve, and 10 in. of Ileum for Chronic Appendicitis.

By LAWRIE MCGAVIN, F.R.C.S.

PATIENT is an unmarried woman, aged 52. Four years ago she was admitted to the Hospital for Women for pain in the region of the appendix, with occasional vomiting of five weeks' duration. There was no definite history of recurrent attacks. Patient looked sallow and ill; temperature 99·5° F. In the right iliac fossa a firm, prominent mass was felt, lobulated on the surface and dull to percussion. The mass was increasing in size and there was a leucocytosis of 10,000. On opening the abdomen the lower portion of the ileum, with the cæcum and appendix, was found to be involved in a dense mass of fibro-plastic material surrounded by many adhesions. The whole mass was isolated and resected, and a lateral anastomosis performed by direct suture. Patient subsequently developed a fæcal fistula, but this ultimately closed, since when she has been in good health.

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[1] *See* F. P. Weber, "Acquired Chronic Acholuric Jaundice with a Blood Picture at one Time resembling that of Pernicious Anæmia," *Amer. Journ. Med. Sci.*, Philad., 1909, cxxxviii, p. 24.

Resection of Cæcum, Ileocæcal Valve, Appendix, and 10 in. of Ileum for Obstruction following an Entero-anastomosis; subsequent Ventral Hernia cured by Filigree Implantation.

By LAWRIE MCGAVIN, F.R.C.S.

PATIENT is a married woman, aged 49. She was admitted to the Hospital for Women in 1908 for the cure of an umbilical hernia. Eighteen months previously she had been admitted to St. Bartholomew's Hospital for the cure of a femoral hernia which was recurrent and strangulated, an enterectomy being performed.

While waiting for operation she developed signs of obstruction. At operation a mass of bowel was found adherent to the middle line above the pubes, the bladder, and the iliac fossa. On separation it was found to consist of the cæcum, appendix, and 9 or 10 in. of ileum and some omentum. At the point of attachment of the ileum to the abdominal wall there was a perforation communicating with a fæcal abscess; the lumen of the gut was here reduced to the size of a lead pencil, the stenosis having occurred at the site of the old anastomosis. The whole mass was excised and a lateral anastomosis was performed, the abscess cavity being drained. A ventral hernia subsequently appeared and became rapidly larger; eight months later this was cured by the implantation of a 6-in. filigree, since when the patient has been in good health.

Resection of Cæcum, Ileocæcal Valve, Appendix, and 5 in. of Ileum for Sarcoma; Anastomosis by Murphy's Button; Button retained for Four Years.

By LAWRIE MCGAVIN, F.R.C.S.

PATIENT is a man, aged 24. In 1908 he was taken suddenly ill with pain in the region of the appendix, set up by lifting a heavy girder. This lasted three weeks, when he commenced to vomit, and was admitted to the Seamen's Hospital. His bowels acted normally and he had no pyrexia. He stated that he had had a similar attack six months previously.

On admission he looked very ill, but his pulse, like his temperature, was normal. In the right iliac fossa was a mass large enough to produce an obvious bulging of the abdominal wall; it was firm, fixed, lobulated, and devoid of tenderness. There was neither hyperæsthesia nor rigidity, and the respiratory movements were good. Nothing was felt *per rectum;* no other lumps in the abdomen, and hepatic dullness was normal. Patient's tongue was foul, and he showed a leucocytosis of 12,000.

Under spinal analgesia the abdomen was opened, and the mass was found to involve the organs above mentioned: no glands were felt in the mesentery or portal fissure; the whole mass was therefore resected with the corresponding iliac fascia and mesentery, and a lateral anastomosis performed. Owing to the threatening condition of the patient, this was rapidly accomplished by means of a Murphy's button. Patient recovered well, but up to date he has not passed the button, which has remained for over four years in the blind end of the ascending colon without causing patient the least inconvenience. For this reason he refuses to have it removed.

Pathological Report (Pathological Laboratory, London School of Clinical Medicine).—"Microscopically the mass is a myxo-sarcoma apparently arising in the cæcum at or about the base of the appendix. There is much inflammatory infiltration about the latter, which is, however, irrespective of the tumour."

Gastro-enterostomy for Hæmatemesis followed by Ileus and Fæcal Vomiting; Cæcostomy; subsequent Ventral Hernia; cure by Implantation of 6-in. Filigree.

By Lawrie H. McGavin, F.R.C.S.

Patient is a man, aged 44. In 1909 he suffered from gastric ulcer and was admitted to the Seamen's Hospital with severe hæmatemesis for which gastro-enterostomy was performed. The hæmorrhage ceased, but forty-eight hours later vomiting, becoming rapidly fæcal, set in. The abdomen was re-opened and nothing was found except a general condition of paralytic ileus. The cæcum was therefore opened and the ileum intubated. The vomiting ceased and the patient made a good recovery. Later the cæcum was replaced, but a ventral hernia soon developed at this point, and four months later a 6-in. filigree was implanted under spinal analgesia. Patient has never had the least discomfort from his gastro-enterostomy, his cæcostomy, or his implantation.

DISCUSSION.

Mr. Lawrie H. McGavin added that in the first case the mass was so adherent that the pelvic fascia had to be stripped from the muscle. In doing this the cæcum was opened and a fæcal infection of the wound occurred. The resulting fistula, however, ultimately closed and the patient made a good recovery.

In the next case the whole condition was produced not by chronic appendicitis but by the fact that she had had an enterectomy done at another hospital for strangulation of a femoral hernia, and there was marked stenosis of the ileum at the site of the end-to-end anastomosis, involving the abdominal wall in an abscess; the appendix, cæcum, and part of the ileum were buried in a huge plastic mass. In the third case, he was not satisfied that it was due to sarcoma, in spite of the fact that the pathological report was positive on the point. The fact that the man had been well for four years aroused a doubt as to whether it could have been malignant. He had brought the section for examination. Preferring direct suture, this was, with one exception, the only case in which he had used a Murphy button. The patient was quite comfortable, and the button, which was lying in the blind end of the ascending colon, had not even turned over, as shown by occasional X-ray photographs. In the fourth case there was what he considered to be true fæcal vomiting—that is to say, solid particles from the large intestine were brought up. This was the only case of true fæcal vomiting which he had seen get well after secondary laparotomy. The patient had had altogether four operations, and had come through them extremely well. The last case and the second one were examples of filigree implantations.

Dr. F. Parkes Weber said that the vomiting of lumps of solid fæces in Mr. McGavin's last-mentioned case (the man, aged 44) was a very interesting point. It had been supposed that vomiting of solid fæces (actual scybala), apart from cases of gastro-colic fistula, occurred practically only in certain cases of functional nervous disease.[1]

Dr. William Ewart asked if, in the patient who vomited solid fæces, there was any obstructive condition at the level of the ileocæcal junction, where the fæces ought to have been fluid. Normally there were no solid fæces until that valve had been passed. Possibly in this instance an obstruction might have influenced a solidification of the fæces at a higher level than normal.

Mr. McGavin replied that the patient was a virile type of man, and not one likely to be neurotic; yet there was no question that he brought up fæces in his vomit. The case was the first he had had of ileus following a simple gastro-enterostomy; he was certain it was an ileus, and not obstruction. He examined the small intestine from end to end, and there was nothing to suggest obstruction; therefore, he opened the cæcum and passed his finger into the ileum, and intubated it. Four hours after a subcutaneous injection of eserine salicylate the patient passed an enormous quantity of flatus and fæces, and the condition cleared up.

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[1] *See* F. P. Weber, "Fæcal Vomiting in Functional Nervous Disease," *Brain*, 1904, xxvii, p. 170; also, "Two Strange Cases of Functional Disorder," *International Clinics*, Philad., 22nd Ser., 1912, i, pp. 125-38.

Celluloid Splints in the Treatment of Acute Poliomyelitis, illustrated by Two Cases.

By F. E. Batten, M.D.

The importance of the use of splints in the treatment of the early stages of acute poliomyelitis in order to prevent deformity and hasten recovery is fully recognized, the paralysed muscles being placed in a position of relaxation. It is difficult to obtain one splint suitable for all purposes. It is important to have a splint which is comfortable for the child, out of which it cannot wriggle, which is easily removed, which keeps the leg in a good position, and which can be worn day and night, whether the child is up or in bed. The celluloid splint answers all these requirements, and is especially suitable for this purpose—it is easily made, it is extremely light, it fits the leg accurately, and it can be applied within the first few weeks of the onset of the disease, and is not expensive. It can be worn not only during the earlier but also during the later stages of the disease.

There are three processes involved in making these splints: (1) the taking of the cast of the patient's limb, (2) the making of the positive from the cast, and (3) the moulding of the splint on to the positive. It is the first, the taking of the cast of the leg, which is most important, for it is essential to keep the limb in a good position whilst the cast is being made.

These splints were first made by Calot, of Berck-sur-Mer, and introduced into this country by Gauvain, who has used them extensively in the treatment of tuberculous disease of bones and joints.

Case I.—L. J., a boy, aged 2, was taken ill suddenly on September 9, 1912, with paralysis of both legs. On admission to the hospital on September 26 he had flaccid paralysis of both legs, with a tendency to extension of left foot. Knee-jerks and ankle-jerks were absent. Celluloid splints were made for both legs, and by October 21 the boy was walking about the ward in the splints with a little assistance. The splints tend to prevent the development of deformity and allow the boy to walk about.

Case II.—R. M., a boy, aged 6, taken ill on October 23, 1911. On the following morning weakness of both legs and right arm was present. He had severe pain for fourteen days. On admission to the hospital in August, 1912, he had a flaccid paralysis of both legs, with marked foot-drop and contraction of the tendo Achillis. The knee-jerks and ankle-jerks were absent. In September tenotomy was performed by Mr. Sargent and the feet put up in plaster. Celluloid splints were made, and the boy can now walk about in these splints with the aid of a stick.

DISCUSSION.

Mr. McGAVIN asked if any steps had been taken to render the celluloid non-inflammable; otherwise, in such cases as the application of strapping with the aid of a spirit lamp there might be danger of firing the splint.

Dr. BATTEN replied that celluloid splints as at present made were not inflammable. He demonstrated this by applying a lighted match to a portion of a splint, which would not burn unless the flame was kept constantly in contact with the splint.

A Case of Congenital Syphilitic Disease of the Knee-joint.

By E. C. HUGHES, F.R.C.S.

H. T., AGED 12. In 1908 the left knee was noticed to be swollen. In 1909 fluid was aspirated from the joint for bacteriological examination; no organisms were found. In 1912 the knee was much swollen, and there was considerable hypertrophy of the synovial fringes. Fluid from the joint was injected into a guinea-pig, with no result. Wassermann reaction positive. In spite of the marked swelling of the joint its functions are not much interfered with.

Mr. HUGHES added that there was a history of many miscarriages in the boy's mother. The joint at present resembled in appearance a Charcot's joint, but the jerks and reflexes were normal, and there were no signs of tabes. X rays showed no bony abnormality. The Wassermann reaction was positive, and congenital syphilitic disease appeared to be the only diagnosis possible.

Chronic Circumscribed Inflammation of the Corpora Cavernosa.

By Donald Armour, F.R.C.S.

A. G., aged 56, noticed a "growth" on his penis twelve months ago. This has gradually grown larger, spreading upwards towards the root of the penis. It is painless. On erection the penis becomes dorsiflexed at the site of the inflammatory plaque. He had gonorrhœa when a boy, but denies syphilis. Urine is normal.

DISCUSSION.

Mr. Donald Armour said he wished to elicit the views of the meeting as to ætiology and treatment. Gout, diabetes, and syphilis had all been advanced as causes of this condition, but they could be excluded here. Iodide of potassium and inunctions of mercury had been tried.

Mr. McGavin suggested that it might be a gummatous condition which had been overlooked. Not long ago there was a case at the Seamen's Hospital, in which there was no history or sign of syphilis, and it was denied by the man. There was dorsiflexion just as in this case, when the man had an erection. Chiefly because of the lack of evidence of syphilis it was thought to be gonorrhœal. But six months later the man returned with a definite breaking-down gumma in the fibrous septum of the penis, which cleared up under very large doses of iodide of potassium.

Mr. Armour replied that he agreed that as the man had had gonorrhœa one could not positively exclude syphilis, but he had been on potassium iodide and mercury for some time without any benefit: in fact, the plaques had increased during the administration of these drugs.

Mr. McGavin said he had a case recently in which a man had a deep sinus in the trochanter of the femur, and no diagnosis was arrived at. On the supposition that it might be syphilis he was given moderate doses of iodide of potassium, but nothing happened, the sinus refusing to heal. It was then thought to be tubercle, but von Pirquet's reaction was negative. The sinus was scraped, and even gouged out, but got no better. It was then decided to give him iodide of potassium until he could not take any more. Finally, when he was taking 128 gr. three times a day the sinus healed rapidly, and he got perfectly well.

Case of Tuberculous Peritonitis.

By JAMES GALLOWAY, M.D.

THE patient, a boy, aged 10, was sent to hospital with the diagnosis of splenomegaly. The abdominal tumour had been noticed from the beginning of August, but the patient had been ailing for some months before that date. An indurated area may be readily felt occupying the upper portion of the anterior and lateral aspects of the abdomen. Its lowest position is in the central line, where its edge may be readily defined two finger-breadths below the umbilicus. The indurated mass is very superficial, giving the impression of being incorporated with the abdominal wall, especially in the umbilical area. The skin at the umbilicus and the immediate neighbourhood appears to be directly adherent to the underlying indurated mass. There is evidence of a small amount of fluid in the abdominal cavity. The glands in the groin are enlarged.

The examination of the blood on October 16 gave the following result: Rouleaux formation and fibrin formation very poor. Red cells, 4,300,000 per cubic millimetre; hæmoglobin, 76 per cent.; colour index, 0·8; white cells, 1,540 per cubic millimetre; polymorphonuclear cells, 61·2 per cent.; small lymphocytes, 29·2 per cent.; large lymphocytes, 4 per cent.; eosinophiles, 3 per cent.; large hyaline, 2·2 per cent.; basophiles, 0·4 per cent. A tuberculin cutaneous reaction gives a very doubtful result. There is a slight degree of fever, his temperature varying between 97·4° and 100° F. There is no evidence of pulmonary tuberculosis.

One of the glands was removed from the groin recently and shows characteristic tuberculous structure. The case is brought forward on account of the early and probably extensive tuberculous infiltration of the omentum with adhesions to the anterior abdominal wall and the special involvement of the superficial structures in the neighbourhood of the umbilicus.

DISCUSSION.

Dr. GALLOWAY said that he had no doubt as to the diagnosis, and he would be glad if members present would give him the benefit of their experience—first as to the local application of mercury in the form of inunctions, or of the liniment; secondly, of surgical methods; and thirdly, especially of any favourable result obtained by the use of tuberculin in such a case as this.

Dr. WARRINGTON said that some time ago he had a case of ascites, supposed to be due to tuberculous peritonitis, and it cleared up with inunctions of mercury. The patient was a girl, aged 18, and she left the hospital apparently well. But recently she came back with a painful and indurated lump in the abdomen similar to that in this case. She passed an extraordinary stool, containing much liquid mucus. After this the pain subsided, and she was again having mercurial treatment, under which the swelling was again diminishing. A diagnostic subcutaneous injection of tuberculin was made which sent the temperature up a degree.

Mr. NORBURY mentioned a somewhat similar case, acute, not chronic, which came under his care some time ago. It was that of a girl who came to hospital with apparent signs of acute peritonitis, and she had an enormous plaque across the abdomen which puzzled those who saw her. As she had an acute lesion, it was thought well to explore. The whole peritoneum was found to be studded with tubercles, and the plaque was the omentum, which resembled a piece of leather, and was studded with tubercles. Nothing beyond closing the abdomen was done, but she got well from that date, and the swelling disappeared.

Sclerema Cutis (Adultorum).

By JAMES GALLOWAY, M.D.

THE patient, J. M., attended the meeting of the Section on May 31, 1912.[1] The thickening and induration of the skin at that time affected the face, neck, shoulders, and trunk to about the level of the loins, and with scattered areas on the extremities. Movements of the arms, neck, and face were very difficult. There is now a great improvement in his condition, large areas of the skin having returned to the normal state.

The treatment consisted of vigorous massage during May, June, and July, and the early part of August. He has had no special treatment since that time, but has now recommenced treatment by massage. The hardening and thickening of the skin is now noticeable only on the face and neck, and slightly on the trunk. The areas still affected are not nearly so firm as when previously exhibited.

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[1] *Proceedings*, 1912, v, p. 203.

DISCUSSION.

Dr. GALLOWAY said that he brought the case forward, as the opinion had been expressed that in all probability the future progress of the case would not be a satisfactory one. The patient, however, was now obviously in much better condition than he was in the early summer. He thought that in such cases as the patient under consideration, in which the hardening of the skin extended deeply in the tissues, and was more of the nature of a firm œdema, the prognosis was much more favourable than when the hardening was more superficial, and led to the ivory-firm trophic patches more usually associated with the name sclerodermia. He had purposely avoided the administration of thyroid substance up to the present, waiting to see the full result of the treatment by massage.

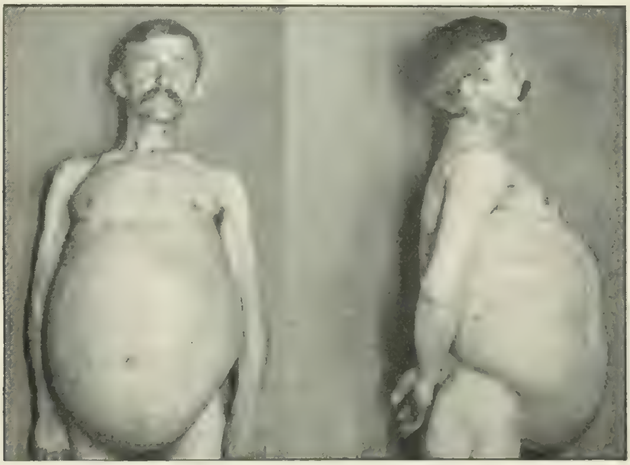
The PRESIDENT remarked that the man still had on the "shirt of Nessus," and his experience of the course in this type was the opposite of Dr. Galloway's. It had been a serious and progressive condition in his cases.

Case of Hirschsprung's Disease.

By R. HUTCHISON, M.D.

R. B., AGED 39, mill-worker. Obstinate constipation and prominent abdomen since birth. As a boy, bowels were usually open one to three times a week. Once he went ten weeks without an action. At the age of 24 he had a severe attack of constipation in which the bowels were not open for eight weeks, and the abdomen became huge. This attack was followed by a week of diarrhœa with some vomiting (not fæcal), after which he was much better for a long time. About the age of 27 he improved, and the bowels acted daily without aperients; the abdomen became smaller, but he was still much troubled with wind. Since then the bowels have been opened every few days, and the abdomen, though always swollen, varies in size. For the last two months the constipation has been very pronounced again. Ever since he can remember he has become swollen and uncomfortable if he remains long in one position, but can get relief by passing flatus. No similar case in the family. No other illness of importance.

Present state: Looks fairly well. General nutrition good. Abdomen enormously distended (*see* photographs), with large loops of distended gut in which active peristalsis can often be seen. One loop runs upwards obliquely from left to right, another transversely across the upper abdomen. In the right loin a large hard tumour can be felt (? fæcal). *Per rectum*, a transverse fold of mucous membrane projects from the anterior wall, and there is apparently a relative stenosis as far as the finger can reach. The other organs are normal, but the heart is pushed up, the apex beat being in the third interspace.



Case of Hirschsprung's disease.

DISCUSSION.

Dr. HUTCHISON said he brought the case because it was uncommon to find the condition in a person of this age, but chiefly in order to invite suggestions as to treatment. What was the best operation, if any, to perform? The literature revealed a difference of opinion as to the best way of dealing with it.

Mr. MCGAVIN asked if Dr. Hutchison had had experience of appendicostomy in this condition. He had not himself had such experience, but he was interested in the case, as he had recently operated on a girl aged 20 for the most intractable constipation with marked abdominal distension, the result being most striking, and he did not see why it should not be of considerable assistance in clearing out the bowel in cases of Hirschsprung's disease.

The PRESIDENT said the case reminded him of the famous case, recorded by Dr. Forman, who exhibited himself at the various medical clinics throughout the United States, and in the side-shows and circuses, as the "Balloon Man." He was even more distended than this man. His large intestine contained, post mortem, 47 lb. of fæces. It was exceptional for the subject of the condition to reach the stage of life which Dr. Hutchison's patient had—namely, 39. He had been interested in the possibility of cure of early cases by medical means, diet, and irrigation. A few cases improved.

Dr. GORDON WARD said he had seen three such cases treated by appendicostomy by Mr. Spencer at Westminster Hospital. One was in an adult, about the same age as Dr. Hutchison's patient, but not so advanced, and the appendicostomy was of the greatest advantage, for while in hospital he was comfortable and had regular motions, and the improvement persisted when seen three months later. One was in an older man, who had general peritonitis at the time owing to a kink, and there was ulceration. He died shortly afterwards. The other case was that of a child who was almost dead on the operating table, and died shortly afterwards.

The PRESIDENT added that he understood that a patient in whom the entire colon was removed by Sir Frederick Treves some years ago was still alive.

Dr. HUTCHISON replied that in the case of a child with this condition he had appendicostomy done, but the child died: he did not know why. But, speaking medically, he would have thought there would have been great mechanical difficulties in doing appendicostomy here. The whole colon was enormously enlarged, and all the normal relations of the parts were disturbed, and so it might be difficult to bring the appendix to the surface. His surgical colleague suggested doing an appendicostomy along with ileo-sigmoidostomy. He had one child with the condition which did very well by keeping the bowel empty. It was difficult to do anything effectual in cases that had been going on for years.

Clinical Section.

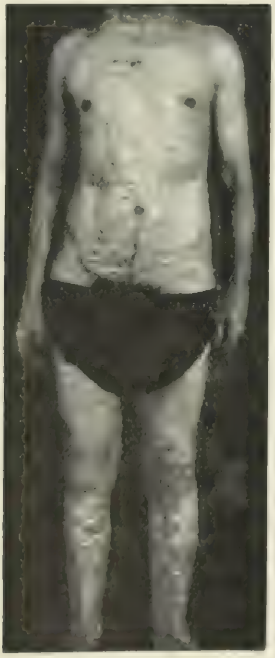
December 13, 1912.

Sir Wm. Osler, Bt., F.R.S., President of the Section, in the Chair.

Case of Obstruction of the Superior Vena Cava.

By A. M. Gossage, M.D.

A man, aged 74, was admitted into the hospital for general malaise. He had had syphilis in his youth, and later had had a gumma on the left upper arm, where there is a scar adherent to the bone. The enlargement of the veins of the trunk had been noticed for the past twenty years.



Case of obstruction of the superior vena cava.

Except that he is rather feeble there seems no impairment of his present health, and he does not suffer from dyspnœa on exertion. On examination there was no enlargement of the heart and no murmurs. The radial arteries are not materially thickened, being remarkably good for his age. The maximum blood-pressure in the brachial artery was 140 mm. Hg. Over the front of the thorax and abdomen are several chains of enormously enlarged and tortuous veins, extending as high as the second ribs, the largest being on the right side. The blood-flow in these veins is from above downwards, and they disappear at the groins. There are no veins at the back of the thorax. The veins of the lower limbs are varicose, and here, of course, the flow is upwards. There is no swelling or congestion of the face or upper limbs, though the brachial veins are rather prominent. X-ray examination shows no abnormal shadow in the thorax.

The case seems to be one of occlusion of the superior vena cava, probably due to cicatricial contraction of a gumma in the upper part of the thorax. The enormous enlargement of the veins evidently allows a free return of the blood from the upper part of the body by way of the inferior vena cava and explains the absence of the usual congestion of the face and arms when the superior vena cava is blocked.

DISCUSSION.

Dr. GOSSAGE added that he had seen two other cases of obstruction of the superior vena cava. The first of these, a man aged about 50, had also had syphilis. Besides the large venous anastomosis over the trunk he had great swelling and congestion of the face and upper limbs, a hoarse voice, and some dyspnœa on exertion. The second case was that of a boy, aged 16, who came into hospital two months ago with right pleurisy and effusion. This was followed by signs of much thickening of the pleura on that side, and of some fibrosis of the upper lobe of the right lung. Gradually the eyes were becoming more prominent, the face swelling and also the lower part of the neck, while the veins in the neck were becoming much enlarged. The patient was very drowsy. That day, for the first time, an enlarged vein stretching from the epigastrium to the umbilicus, in which the blood-flow was downwards, had been found. Thus this case afforded an example of the gradual development of obstruction of the superior vena cava under observation, due presumably to a fibrosis of the mediastinum secondary to a pleurisy, the whole probably of tuberculous origin. The patient, he might add, had a hectic temperature.

Dr. DE HAVILLAND HALL said these cases of obstruction to the venous circulation were always of great interest, but were seldom seen post mortem.

At present he had a patient with considerable obstruction of his superior vena cava. He weighed about 17 st. When he first came under notice, eighteen months ago, there was no œdema of the eyelids and face, and his neck was very thick. Under the influence of iodide of potassium and thyroid extract there had been considerable improvement, and the neck circumference had decreased by 1½ in. But the patient was very drowsy, probably on account of the congestion of the cerebral circulation.

Dr. ESSEX WYNTER said the patient was fairly well known in medical circles—he had been at the Examination Hall several times. The case had always passed as one of obstruction of the inferior vena cava. Dr. Gossage's interpretation was a new one, and he (Dr. Wynter) would suggest a third. He considered that the veins were of small volume compared with the volume of the vena cava, and this man showed no evidence of interference with the circulation of the neck and arms. He therefore put forward the view that it was obstruction of the azygos major, and that the blood which could be seen returning on the chest was only from the walls of the thorax, not from the head and arms.

The PRESIDENT (Sir Wm. Osler, Bt., F.R.S.) had seen two groups of these cases of complete obstruction of one or other vena cava; one in which there were no symptoms whatever. The patients were comfortable and able to do everything, just as this man was. In one such case, which he had published with drawings, there was little or no distension of the vessels of the head. In such cases it had been usually a slow sclerotic process, with complete fibrous transformation of the vein. In the second class, the more common, there was obliteration—from the pressure of an aneurysm or from pressure exerted by lymphadenoma. There was great distress, and very much greater distension of the veins. Patients of this group did not last long. But the literature contained numerous cases with practically no symptoms, but with long-standing fibrous obliteration of one or other vena cava.

Two Cases of Prostatic Calculi.

By RALPH THOMPSON, F.R.C.S.

Case I.—R. P., AGED 67 (case shown). First attended at St. Paul's Hospital in September, 1910, for "thick water," which cleared up with drug treatment. Dr. Allport examined patient and found prostatic calculus. Patient was sent on to Guy's Hospital at the end of November, 1911. Examination with sound revealed stone; calculus apparently felt with shaft of instrument, the beak of which was free in bladder. Rectal examination showed stone in region of prostate. Operation by perineal route in front of transversus perinei, December, 1911; capsule of prostate incised and stone removed; wound healed well; weight of stone, 32·5 grm. Composition of stone: one large and three small facetted pieces. Skiagram important as showing surface markings of prostate, and position of prostatic stone. Patient appears now perfectly well.

Case II.—J. T. P., aged 73 (specimen only shown). Patient admitted for difficulty in passing water on October 30, 1912. Catheters passed with some difficulty. Suprapubic cystotomy under spinal anæsthesia. After operation, some difficulty in passing catheters occasionally, not always. November 10: Stone felt in region of prostate with conic catheter and sound. Skiagram reveals three prostatic calculi lying behind pubes. Nothing felt *per rectum* except a very hard prostate. November 20: Death. Post mortem: Double aortic aneurysm; bladder, prostate and urethra removed for examination and exhibition.

DISCUSSION.

Mr. W. G. SPENCER said he missed in the notes any reference to the presence of carbonates in the calculi, so that he did not see how Mr. Thompson could distinguish a prostatic calculus from a phosphatic calculus lodged in a dilatation at the neck of the bladder. Some years ago, in a case under his care, there had previously been a calculus in the bladder, following upon long-continued stricture of the urethra; then there were two or three operations performed through the perineum, and the removal of small facetted calculi, which contained carbonates. The man died eventually from kidney trouble, after which it was found that these calculi had been removed from within the capsule of the prostate.

Mr. RALPH THOMPSON, in reply, said he did not think the presence of carbonates was important when the calculus was in the prostate itself. The calculi he exhibited were clearly in the prostate. He had to incise the prostatic capsule to get at the stones. Dr. Ryffel reported that the composition of the stones was mainly triple phosphate; he did mention carbonates.

Family Cerebellar Ataxia in Two Half-sisters.

By F. PARKES WEBER, M.D.

Case I.—E. E., a well-grown but somewhat mentally deficient girl, aged 15. She was backward in learning to walk, and her mother says that she never was able to speak distinctly. At the age of 6 she could not walk and run as well as other children of the same age, and tended to fall forwards when excited or if she tried to go too fast. When aged 7 she had scarlet fever, which was followed by right-sided otorrhœa. About Christmas, 1908, increasing tendency to fall was observed, and she sometimes had to support herself with her hands when standing. She often complained of headache, and sometimes vomited. In May, 1909, when she first came under Dr. Weber's observation at the hospital, there was decided unsteadiness in gait, especially noticeable when she tried to walk along a marked-out line and when she turned round suddenly. Occasionally there was tremulousness in the limbs and head. No definite Romberg's symptom. Her mother thinks that her gait afterwards improved somewhat. At present (November, 1912) she walks slowly and somewhat unsteadily, placing her feet rather too far out sideways. She tends to "totter" when turning round quickly. Romberg's sign is negative. Patellar, Achilles, plantar, abdominal, and pupillary reflexes are normal. There is occasionally fine horizontal nystagmus on looking (with strain) to right or left. No muscular wasting; no anæsthesia; no "pes cavus"; no deformity of the vertebral column. Her speech is slow and monotonous. She is clean in her habits. Nothing abnormal in the thoracic or abdominal organs, or in the urine. The Wassermann reaction for syphilis (Lister Institute) is negative with the patient's blood serum. There is scarring from old mastoid operations on the right side. The hearing is good. Nothing abnormal by ophthalmoscopic examination.

Case II.—P. J., a well-nourished but somewhat mentally defective girl, aged 4, half-sister (by her mother) of the first patient, E. E. She is said to have begun to speak when aged 2½. She used to get about, her mother thinks, like other children of the same age. In November, 1911, she had a sore throat, and in January, 1912, she was brought to the German Hospital out-patient department because she had almost lost the power of walking. At that time her knee-jerks were found to be normal. In February, 1912, she could walk about, but with a paretic-spastic gait, and the knee-jerks were excessive. After that improvement is said to have occurred, but in July the child had an attack of follicular tonsillitis with fever, and the gait became worse again. When admitted to the German Hospital (July 15, 1912) the child could not walk or stand without support. Her gait was of an unsteady, paretic-spastic type, and she preferred to place her feet a good deal apart when standing up. There seemed evidently to be some deficiency in the balancing power. Marked tremulousness in the upper and lower extremities when walking; the movements of the upper extremities were rather slow and showed no marked ataxia. The knee-jerks were readily obtainable, and were, if anything, rather excessive. The plantar reflexes were of the extensor type in both feet. The pupillary reflexes were normal, and there was no nystagmus. Ophthalmoscopic appearances were normal. The child spoke very little, and only in a slow, monotonous way. No muscular atrophy; no definite signs of rickets. Nothing abnormal in the thoracic or abdominal viscera. Since July there has been some improvement, and the plantar reflexes were found to be of the normal flexor type, when tested at the end of September. At present the child can stand without support, and can walk if someone holds one of her hands, though the gait is very unsteady and somewhat spastic.

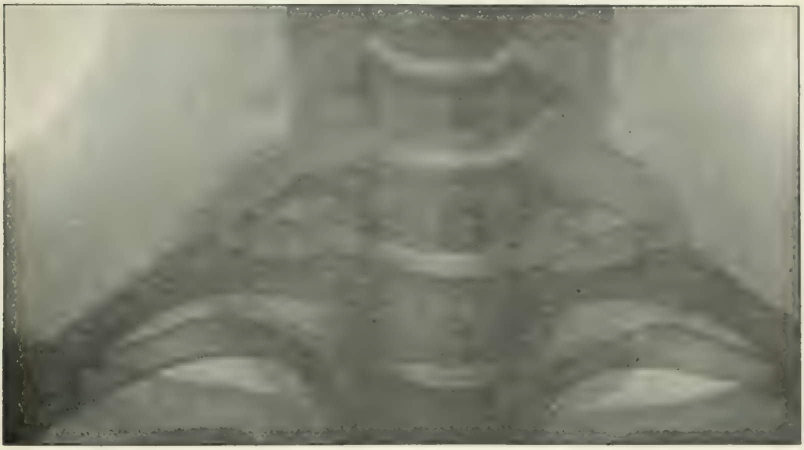
In both of these cases the ataxia is very slight in the upper extremities, and only in the second case is it considerable in the lower extremities. In the eldest patient the chief symptom is perhaps, at present, the tendency to "totter" on walking and turning round sharply. In the second case the spasticity of the gait and the presence of Babinski's phenomenon in July pointed to involvement of the cerebral motor cortex as well as of the cerebellum, the symptoms being those of what one might term a mild cerebro-cerebellar diplegia.

No history of nervous disease in other members of the family can be obtained, but the subject is rather difficult to investigate, as the mother (herself one of a family of sixteen children), a healthy-looking and apparently mentally normal woman, aged 41, has had children by three different men.

Bilateral Cervical Ribs with Unilateral (Right-sided) Atrophy of Hand Muscles.

By F. Parkes Weber, M.D.

The patient, D. W., aged 21, is a well-built young woman. Four years ago she fell on her right side; she noticed nothing especially wrong till a year later (that is, three years ago), when she began to suffer from pain in the right upper extremity and there was some wasting in the right hand. The pain was of a "burning character," passing from the right shoulder along the inner back part of the arm to the elbow and down the ulnar side of the forearm to the wrist and ulnar side of the hand. This pain, though not always present, has troubled her on and off since then, and lately she has likewise had pain of a more biting character on the ulnar side of the affected hand. The wasting in the right hand has somewhat increased since it was first observed (three years ago). When she is exposed to cold weather her right hand is more numbed and bluer and feels colder than her left hand, but she does not think her right hand becomes definitely weaker in cold weather. At present there is marked atrophy of the intrinsic muscles of the right hand; this atrophy is shown in front in the thenar and hypothenar eminences and at the back in the region of the interossei muscles between the metacarpal bones. When she holds her right hand open with the thumb stretched out, the transverse thenar ridge, formed by the outer part of the adductor pollicis muscle, is very well marked, as pointed out by Kinnier Wilson in cervical rib cases. The distal part of the right forearm appears slightly smaller than the corresponding part of the left upper extremity. The dynamometer grasp is $7\frac{1}{2}$ in the right hand against $15\frac{1}{2}$ in the left hand (the normal grasp by the dynamometer in question would be about 15 to 20). Electrical examination with the galvanic current shows that ACC is greater than KCC in the muscles of the thenar eminence of the right hand, whereas in the corresponding muscles of the left hand KCC is greater than ACC. There is no anæsthesia or hypo-æsthesia in the hand and forearm of either side. Skiagrams, kindly taken by Dr. N. S. Finzi (*see* figure), show that the patient has a small seventh cervical rib on each side, though these cannot be felt by palpation in the neck. Nothing else abnormal has been detected except that she has moderate retinitis



Skiagram to show seventh cervical ribs in the case of D. W. (December, 1912.)

pigmentosa (Dr. R. Gruber) and decided internal-ear deafness (Mr. G. J. Jenkins), and that from childhood she has had slight "dysarthria" in regard to the pronunciation of certain consonants. The brachial systolic blood-pressure is, if anything, slightly greater on the affected side (120 mm. Hg.) than on the unaffected, that is to say, the left side (110 mm. Hg.). There is no scoliosis and no abnormality in regard to sweating, pupillary reactions, or tendon reflexes, and, when she is not exposed to cold weather, there is no difference in colour and temperature between the two hands.

Symptoms due to seventh cervical ribs are commoner in females than in males, and usually first show themselves about the time of puberty, as they did in the present patient (in whom menstruation commenced at the age of 17). When, in cases of bilateral cervical ribs, the symptoms are only unilateral, Dr. Weber believes that they are usually (as in the present case) on the right side. The slight dysarthria (defect in the pronunciation of certain consonants) observed in the present case represents a faulty development in an important function, which may in a kind of way be compared with faulty developments in structures of the body (such as the development of cervical ribs), since they both belong to the class of (unimportant or important) abnormalities which have sometimes been included as "stigmata of degeneration." An occasional association has been claimed for cervical ribs with other minor malformations (Oppenheim), such as the presence of medullated nerve-fibres in the retina, and also with various "degenerative" nervous diseases, amongst which may possibly be classed Graves's disease and syringomyelia. On the other hand, cervical ribs are not very rare, they often give rise to no symptoms (so that their presence is not sought for by Röntgen-ray examination), and the association may be a chance one. Moreover, symptoms connected with cervical ribs may occasionally have been supposed to indicate the presence of syringomyelia, when the latter disease was not really present.

The retinitis pigmentosa and internal-ear deafness are interesting in the present case. Retinitis pigmentosa is well known to be sometimes associated with deaf-mutism or internal-ear deafness, but amongst the various congenital or developmental abnormalities which occasionally accompany retinitis pigmentosa, cervical ribs are not mentioned by Wilbrand and Saenger.[1] No relatives of the patient are known to have cervical ribs or retinitis pigmentosa.

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[1] H. Wilbrand and A. Saenger, "Die Neurologie des Auges," Wiesb., vol. iv, part i (1909), pp. 97-101.

Cervical Ribs with Atrophy of Hand Muscles.

By F. Parkes Weber, M.D.

The patient, E. B., aged 17, is a well-nourished girl, who first came under Dr. Weber's attention when she was aged 13, for symptoms suggesting the presence of seventh cervical ribs. The symptoms were almost entirely confined to the right upper extremity. There was decided wasting of the thenar, hypothenar, and inter-metacarpal regions of the right hand. The right hand was weaker and usually felt colder than the left hand. Exposure to cold made it still weaker, and it more readily became numbed than the right hand. The dynamometer grasp in the right hand was 5, and in the left hand 15 (the normal grasp by the dynamometer in question would be about 15 to 20). Electrical examination by galvanism showed reaction of degeneration in the muscles of the thenar and hypothenar regions of the right hand. There was decided hypo-æsthesia on the ulnar side of the right upper extremity, notably in the hand. A skiagram (fig. 1) by Dr. N. S. Finzi, showed the presence of a small seventh cervical rib on each side, but the one

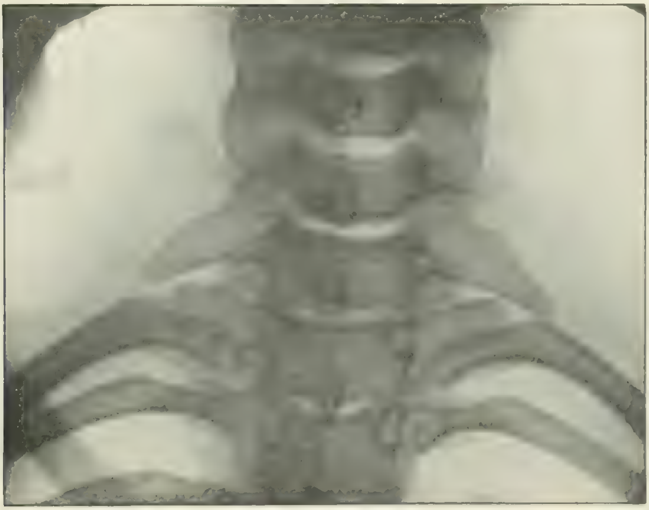

Fig. 1.

Skiagram to show seventh cervical ribs in the case of E. B., in April, 1909, before operation.

on the right side was the bigger of the two. Neither of them could be detected by ordinary palpation. The wasting in the hand muscles had been observed during the previous two or three months only, but pain of a sharp, shooting character in the right upper extremity had been occasionally complained of for the last two years. The knee-jerks and Achilles-jerks were normal. In this case the symptoms due to cervical ribs first attracted attention, as usual, about the period of puberty, for menstruation commenced when she was aged 13.

The right seventh cervical rib was removed by Dr. E. Michels on May 14, 1909, and since then the patient has never had the peculiar pain she complained of in the right upper extremity. After the operation, however, she at first lost power in her right hand. This gradually returned, and at present the dynamometer grasp (the same dynamometer used as previously) with her right hand is 11, against 26 with her left hand. There is still much wasting in the intrinsic muscles of the right hand, and her weakness causes difficulty in writing, &c., for she has not become left-handed. In the wasted hand there is very slight flexion of the fourth and fifth fingers, but no definite contracture. The electrical examination with galvanism shows that ACC is greater than KCC in the muscles of the thenar and hypothenar regions of the wasted hand. During exposure to cold the right hand still becomes more readily numbed than the left hand, and there is still decided hypo-æsthesia over the ulnar portion of the right wrist and hand,

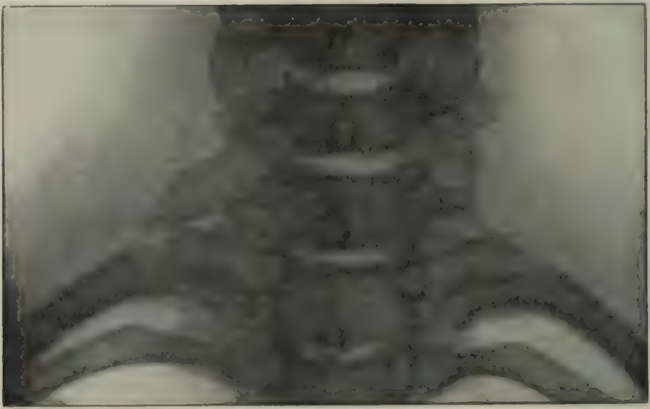

Fig. 2.

Skiagram of the neck in the case of E. B., taken in 1910, to show appearance after the removal of the right seventh cervical rib.

including the fourth and fifth fingers. Before the operation the patient was shown at the meeting of the Medical Society of London, on April 26, 1909.[1] The skiagram of the neck, taken by Dr. Finzi in 1910, shows the appearance after the operation (fig. 2). A brother and a sister of the patient have been found by Röntgen-ray examination (Dr. Finzi) to have bilateral cervical ribs, but they are both younger and have not, as yet, complained of any symptoms.

DISCUSSION.

The PRESIDENT suggested that the surgeons should bring before them the results of removal of cervical ribs. The cases were very serious and difficult to treat. One was at a loss what to advise a patient with an atrophied and painful hand, due to a cervical rib. The operation was a serious one, and in a certain number of cases it was said not to have been successful. The day before he had seen a girl sent in for the M.B. examination, who, four or five months after operation, was certainly worse, except in one point the pain was better, but she had a useless hand.

Dr. HINDS HOWELL said operations had been performed in four of his cases. With regard to the after-effects, one of these four patients had complete loss of power in the whole arm, which persisted for a month, and then gradually improved. Eventually the power in the other muscles, not affected before the operation, became normal again, and there was improvement, but not complete cure, of the atrophied muscles. Another case was that of a woman, whom he saw again three years after the operation for removal of the rib, and her muscles were practically normal: she had been operated upon soon after the muscular atrophy began to appear. In the third case subjective sensory disturbance was very marked, and the muscular atrophy comparatively slight. After operation for removal of the rib there was a very severe pain in the neck, at the site of the scar: this pain persisted for three years after the operation, and had been even worse than the pain originally complained of. It was slowly improving, and the power of the hand was much improved. The fourth case was that of a man in whom there was very extensive hand atrophy. Following removal of the rib there was paralysis of the serratus magnus, an accident which had happened on more than one occasion, because the posterior thoracic nerve ran considerable danger of being injured at operation if the rib was disarticulated. Neither the serratus magnus paralysis nor the hand atrophy was recovered from when the man was lost sight of, but the vasomotor condition of the hand was improved. In well-defined cases he recommended early operation.

Dr. PARKES WEBER remarked that when the collection of cases was arranged, it should include late cases which had not yet been operated upon, so as, if possible, for purposes of comparison, to illustrate the progress of the hand changes when the cervical ribs were not removed.

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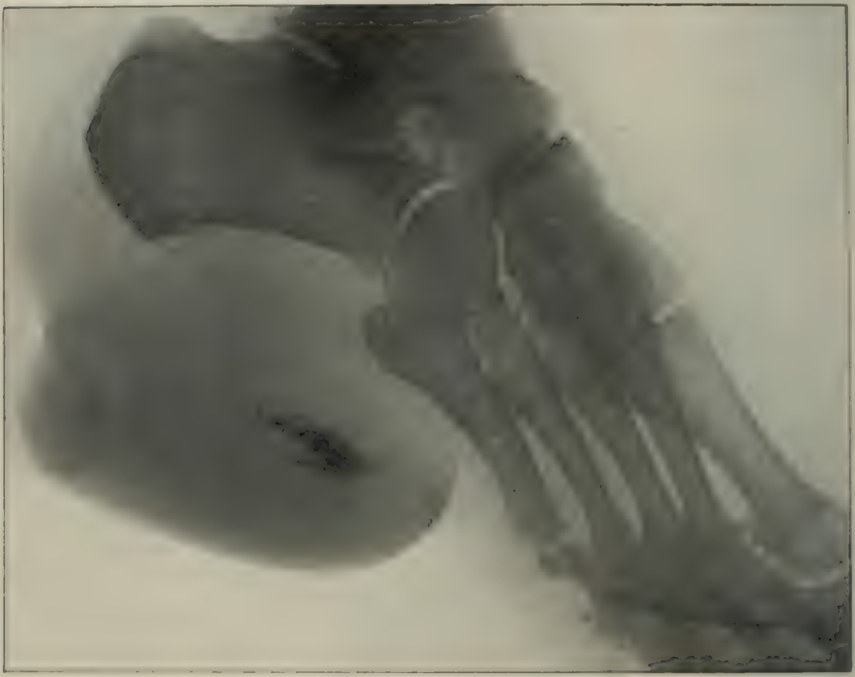
[1] F. P. Weber, *Trans. Med. Soc. Lond.*, 1909, xxxii, p. 304.

Sarcoma of Foot.

By Philip Turner, M.S.

J. H., aged 72, first noticed a swelling of his right foot thirty-four years ago. He says that it appeared after an attack of rheumatism. Though slowly increasing in size, it gave him no particular trouble till recently, when the enlargement has been more rapid. Two months ago the skin at the posterior part gave way and the resulting ulcer has never healed. There is now a large tumour, the maximum length of which is 4 in., situated at the posterior part of the sole and outer side of the right foot. Posteriorly there is an ulcerated surface the size of half-a-crown from which a fungating mass protrudes. The tumour is elastic in consistency, and appears to be adherent to the os calcis. Radiographic examination, however, shows this and the other tarsal and metatarsal bones to be unaltered. There is a small area of ossification near the centre of the tumour.



Sarcoma of foot.

Case of Subcutaneous Drainage for Ascites.

By W. ESSEX WYNTER, M.D., and JOHN MURRAY, F.R.C.S.

E. C., MARRIED, two children, was admitted on August 30, with a history of peritonitis and colic nine years before, followed by enlargement of the abdomen. She had attended the out-patient department for three months on this account, increase in the size of the liver and spleen being noted. She had suffered with symptoms referred to the liver for three years, with occasional diarrhœa, vomiting, and hæmatemesis, and had been losing weight. There had also been complaints of numbness and loss of power in the hands, with shooting pains in the legs. She is now thin, with earthy complexion and stigmata on face, the knee-jerks are feeble, and there is some arterial degeneration. On September 17, the abdomen being very tense and respiration hampered, 9 pints of fluid were withdrawn; measurement, 34¼ in.; the enlargement of the liver and spleen being then obvious. Fluid rapidly re-accumulated, and by October 10 she was tenser than before; measurement, 35½ in. On October 18 an incision was made in the mid-line below the ensiform cartilage, and a decalcified bone tube inserted into the peritoneal cavity, its free end being buried in the parietes. The skin was then sutured over it. The hobnailed surface of the liver was felt, leaving no doubt as to cirrhosis, some fluid escaped, and considerable leakage occurred afterwards. Owing to delay in healing, and the occurrence of some suppuration, the bone tube was withdrawn fourteen days later. Measurement, 26½ in. Temperature throughout ranged from 97 to 100° F. Fluid has not re-accumulated.

DISCUSSION.

Dr. ESSEX WYNTER added that since sending in the notes some fluid had collected in the abdomen, although much of the protrusion was due to the flatulent distension. But he thought the result was satisfactory, inasmuch as on first admission the abdomen was very tense, refilled within a week, and soon afterwards had to be emptied again; but now the patient had gone on for two months practically without re-accumulation.

Dr. DE HAVILLAND HALL asked whether Dr. Wynter brought this method forward as an improvement on his femoral drainage. He did not understand how the fluid would drain from the peritoneal cavity into the cellular tissue of the chest. Femoral drainage seemed quite reasonable, and as he had stated before, Mr. Spencer had operated upon a patient of his with some benefit, but not with complete success, as the patient had had to be tapped forty times since.

Sir JOHN BROADBENT said he had a patient who was subject to recurrent attacks of ascites for which she was tapped every month for about twelve months, but she did not seem to lose flesh or become seriously run down in health. When she came to hospital she had a temperature of 102° F., and her chart was irregular. The liver could be felt 2 in. below the costal margin. As a last resort he had the operation of omentopexy done by Mr. Jackson Clarke, and she entirely recovered, and had not had a recurrence, though it was done nine months ago. The liver was found to be rough, and there was much lymph on its surface. In cirrhosis of the liver there was a hobnail fibrous condition, there was very little liver surface left. As Dr. Hale White had said, the real danger there was due to the absence of liver substance, and not merely the occurrence of the ascites, which was often a terminal phase. When the liver was enlarged and there was perihepatitis, the operation stood a good chance of being successful. Previously to that case he had had little faith in operation for cirrhosis of the liver, for he had seen one or two cases which did not do well. The case he had described was not tuberculous.

Mr. W. GIFFORD NASH said he had performed the femoral operation in two cases, as advised by Dr. Wynter, and both showed some peculiar features. The first was a woman, aged 48, who was a servant in a public-house. In August, 1908, at St. Bartholomew's Hospital, 14 pints of fluid were drawn off. In January, 1909, she was again tapped and 20 pints drawn off. The following March spontaneous rupture occurred at the umbilicus, and 30 pints escaped. Later she attended Bedford Hospital, and he drew off 41 pints on May 2; 32 pints on May 23; 32 pints on June 13; and two days later he performed omentopexy. He drew off 16 pints on July 4; 19 pints on July 25; 18 pints on August 15; 8 pints on September 5; 13 pints on September 26; and 13 pints on October 28; after which she went four months to February 18, 1910, and 11 pints were then evacuated. A week later he put a bone bobbin into the femoral canal, and there had been no return of the fluid since. On admission she had a gumma over her ribs, and throughout her stay in that hospital she had iodide of potassium and mercury. At the first operation he found she had gummata of both liver and spleen. Though she had an alcoholic history, he felt that the ascites was largely due to the syphilis. The second case was that of a barman, aged 43, who was in St. George's Hospital at the beginning of this year, and 5 pints were drawn off on January 23; 14 pints on February 6; 16 pints on February 20; 12 pints on March 4; and 15 pints on March 15, when he was discharged to go to the infirmary. But instead of this he went into the country and came to Bedford Hospital. Thirty pints were drawn off on March 27; 17 pints on April 11; and on the following day he inserted a bone bobbin into the femoral canal. In order to avoid tension on the stitches he drew off 11 pints on April 21; 11 pints on May 3; and 9 pints on May 12. In July he left the hospital, and had not had a recurrence by October. There was no history of syphilis in his case, but being a barman, and an alcoholic, he gave him iodide of potassium and mercury. He was now well except for his enlarged liver and spleen. In Osler's "System of Medicine," and in Rolleston's "Diseases of the Liver," it was stated that cases of cirrhosis of the liver rarely survived a second tapping. This had also been his experience in purely alcoholic cases, the patients dying of cholæmia. He doubted, therefore, whether the cases which recovered after repeated tappings were purely alcoholic; they were likely to be due to chronic peritonitis, often syphilitic.

Mr. W. G. SPENCER said that three years after he had performed omentopexy he had shown, at the Clinical Society,[1] a woman who was a drinker, and who said she continued to drink as much as she could get for three years. She had no re-accumulation of fluid. Later she died at Westminster Hospital, with evidences of alcoholic cirrhosis of the liver, without any signs of syphilis. She was under Dr. Hall, and had been tapped ten times before Mr. Spencer operated. Her liver was large. He had not been fortunate with cases of atrophic cirrhosis of the liver, but no harm followed if the operation was aseptic.

Dr. ESSEX WYNTER, in reply, said this woman's condition was such that it was thought she could not stand the double incision for the femoral opening. One patient developed a femoral hernia, and it was to avoid that risk, and also to suit the measure to the patient, that he suggested this operation. He saw, a fortnight ago, the man who was the first subject of femoral drainage, and during the five years he had remained free from ascites, although he had been drinking steadily the whole of the time.

Case of Hirschsprung's Disease.

By R. HUTCHISON, M.D.

THIS patient was shown at the last meeting,[2] and is now exhibited again to show the result of treatment by brine enemata. The abdomen has become quite flat.

DISCUSSION.

Dr. HUTCHISON added that the patient had lost 2 st. in weight under the treatment, and his general condition had improved. Probably most of the weight lost represented fæcal matter. He was still having daily brine enemata.

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[1] *Trans. Clin. Soc. Lond.*, 1906, xxxix, p. 239.

[2] See *Proceedings*, p. 44.

Dr. JAMES GALLOWAY said that the improvement in this patient's condition was very remarkable, and wished to know if any special method had been adopted by Dr. Hutchison in prescribing or administering the brine enemata.

The PRESIDENT remarked that notwithstanding the immediate good result of the brine enemata the outlook for that man, in his view, was hopeless unless he had surgical intervention. A procedure successful in some cases was to make an artificial anus and then, in a double operation, to do lateral anastomosis and afterwards remove the colon, or whatever part of the tract was at fault.

Dr. HERTZ said he considered the outlook of surgical influence in Hirschsprung's disease was very far from bright He had had three cases of the condition operated upon and had seen a fourth, and all four died. A fifth case of his had been similarly treated against his advice; the whole colon was successfully removed, but the patient returned three months later with the abdomen as distended as ever, probably because the small intestine was now also involved.

The PRESIDENT said that the general results of surgical interference in cases which had reached this grade were better than from medical treatment. Sir Frederick Treves's case, which was operated upon some eleven years ago, was, he believed, still alive. Several of the cases operated upon at Johns Hopkins Hospital had done well.

Mr. SIDNEY BOYD desired to refer to a case which he had a few years ago, not of ordinary Hirschsprung's disease, as there was a congenital stricture of the rectum just above the anal canal. The patient was a boy, aged 8. He had an enormous abdomen. X-rays and a bismuth enema showed the colon to be normal as far as the pelvic brim on the left side; it then turned up to the gall-bladder, and went down the pelvis on the right side. By dilating the stricture and treating him with enemata the bowel became clear. He went on for over a year having treatment by massage and drugs and enemata, but could not be left alone for long. Mr. Boyd explained to the mother that the boy would probably not get any better, and she then agreed to operation. By giving a test meal he found that the real hindrance to the passage of faeces began beyond the iliac colon, and he removed the colon beyond that point, as low as possible, and made an anastomosis with the upper end of the rectum. The patient made a good recovery, and though that was a year ago, no drugs or enemata had since been required, and the bowels had acted every day.

Case of Organic Hemiplegia following Typhoid Fever, in which the Plantar Reflex is Flexor, but Babinski's "Second Sign"—Combined Movement of the Trunk and Pelvis—is Present.

By A. F. Hertz, M.D.

T. J., aged 31, had typhoid fever in 1902. At the end of the second week of his illness he woke one morning to find that the left side of his face and his left arm and left leg were paralysed. The paralysis diminished for a time, but after some months no further improvement occurred.

There is now very little evidence of facial paralysis and the arm has recovered most of its power but still shows associated movements. The left leg is weak and spastic, the knee-jerk is increased and ankle clonus is present. The evidence, so far, is strongly in favour of the case being one of organic hemiplegia, due probably to cerebral softening following thrombosis occurring during typhoid fever. It was therefore expected that the left plantar reflex would be extensor, but it is quite definitely flexor. The organic nature of the hemiplegia is, however, proved by the presence of the "combined movement of the trunk and pelvis," described by Babinski, which I have called for convenience "Babinski's second sign." The patient, lying flat upon his back, with his arms folded across his chest and his legs widely separated, is told to rise to the sitting position without using his arms. At each attempt to do so the paralysed leg rises, the other leg remaining on the floor or rising considerably less high. The same thing happens to a less marked degree when the patient falls back from the sitting to the dorsal position.

Babinski was the first to point out that the paralysed leg remains flat on the floor in hysterical hemiplegia, whereas in organic hemiplegia it always rises higher than its fellow. In my experience the sign is of great value, as I have several times obtained it when the plantar reflex was unobtainable, and also in children under the age of 3 in whom the plantar reflex is normally extensor.

A Case of Pneumonotomy for Foreign Body.

By Thomas H. Kellock, M.C.

The fortunate rarity of the necessity for such a serious operation as that of cutting into the substance of a lung for the removal of a foreign body, and the paucity of records of such where the condition was not associated with the presence of empyema, make me think that the history and records of the case I am showing this evening cannot fail to be of interest. In this case, as will be seen, every effort had been made to remove the pin through the trachea. These not having been successful, there seemed to be only two courses open: One might have awaited the formation of abscess and empyema with the hope that the pin would have found its way into the pleura and have been removed when the empyema was opened; the alternative being direct incision into the lung through an opening in the chest wall. Realizing what great danger the child would run by adopting the former of these courses, and remembering how seriously and permanently the lung in such cases is damaged, even if the patient escapes with his life, I thought it best, after advising the parents of the severity of its performance, to make an effort to remove the pin by a direct incision.

The patient is a little boy, who at the time of the accident was aged 1½. He was brought to the Middlesex Hospital on June 3 of this year from Lincolnshire, to be under the care of my colleague, Mr. Somerville Hastings. The history given was that four days previously he had swallowed (as it was termed) a shawl pin about 2 in. long. When admitted he appeared to be in good health, and there were practically no abnormal signs to be found in the lungs. A skiagram showed the shadow of a pin at the level of the third rib on the right side, apparently in the right bronchus, with the point upwards. The day after admission Mr. Hastings tried to remove the pin through a bronchoscope. It was seen and grasped several times with forceps, but efforts to extract it were unsuccessful. This operation was followed by a good deal of rise of temperature and some consolidation at the base of the right lung. On June 12 a low tracheotomy was performed, and an endeavour made to remove the pin by means of the bronchoscope passed through the wound and also through the larynx, and on June 15 and on June 19 this was again attempted, but without success.

As Mr. Hastings was leaving London, he kindly transferred the child to my care on June 22. The tracheotomy wound was then closing, but there was a good deal of emphysema about the neck and thorax. The child's general condition was quite good, but he had a slight cough, and the breath was rather offensive. A skiagram taken at this time showed the pin to be lying further down towards the diaphragm than on the previous occasion.

On June 24, under an anæsthetic and with the aid of a fluorescent screen, I made an attempt through the tracheotomy wound to remove the pin by means of instruments made out of gum-elastic catheters with the stylet protruding through a cut end and sharply turned over. With one of these I thought I succeeded in grasping the pin, but even on using a fair amount of force it could not be moved, and apparently the instrument frequently passed into different channels to that occupied by the pin. Mr. Lyster at this time localized the pin lying almost vertically, with the head apparently about ¾ in. above the diaphragmatic surface of the right lung.

On July 3 the tracheotomy wound was closing again and no air was entering through it; the child had a slight cough but no expectoration. The breath was offensive, and he was slightly anæmic; otherwise he appeared to be in fairly good health, and on this day the open operation was performed. Mr. Apperly anæsthetized the patient rather deeply. He was turned over on to the left side, and the whole of the back of the right chest painted with tincture of iodine. The marks on the chest wall which had been used for the purpose of localization were still visible, and were used as guides as to the level at which to open the chest. A square flap, consisting of skin and muscles, measuring about 4 in. in width and depth, was then reflected backwards, the edges of the flap being parallel to the direction of the ribs, and the posterior ends of the upper and lower incisions reaching to within about an inch of the middle line of the back. After the bleeding had been arrested four of the exposed ribs were divided subperiosteally at the anterior part of the wound. The deep muscles of the back were then retracted towards the middle line, and the same ribs cut with bone forceps as far back as they could easily be reached, the forceps being passed close above and below the bones; the intercostal muscles and pleura were then divided along the anterior ends of the cut ribs in the whole length of the wound. Air entered freely into the pleural cavity without any serious effect on the patient, but just at this stage he coughed and brought up some very offensive pus. He was soon comfortable again, and the operation was continued by dividing the intercostal muscles and pleura above and below the divided ribs, and then the flap consisting of ribs, intercostal muscles and pleura turned completely backwards on the hinge formed by the posterior sections of the ribs. A window about 3 in. square was thus made into the pleural cavity. The lung was seen to be collapsed and fallen away from the wound, and the diaphragm bulged strongly upwards at each expiration, and had to be kept down by a flat retractor whilst the lung was being examined. A few fine adhesions between the lung and pleura were found posteriorly, but were not sufficient to hold the lung against the chest wall, and were quite easily broken away. A finger was passed under the lung between it and the diaphragm, and at about the centre of the diaphragmatic surface it was thought that a hard spot could be detected in the substance of the lung. Two silk sutures were then passed by means of round-bodied needles through the edge of the lung to hold it in position against the chest wall, but they were not found to be of much assistance. Pushing the lung upwards it was possible to see its under surface, and just where the hard spot had been detected an incision was made into the substance of the lung about ½ in. to ¾ in. deep. This was followed by very slight hæmorrhage. On passing the tip of the finger into the wound the lung receded so much on the least pressure that nothing could be felt. Two fingers of the left hand were then passed into the sulcus between the middle and lower lobes of the lung, and the lower lobe pulled outwards and steadied. Directly this was done the tip of the finger of the right hand in the wound in the lung felt the head of the pin, and it was easily extracted with sinus forceps. It was found to be a steel pin 1½ in. in length with a glass head about ⅛ in. in diameter. Immediately after its extraction a little very offensive pus followed it out of the wound in the lung. One fine silk suture was passed through the edges of the wound in the lung, partially closing it. At this time the child was taking the anæsthetic fairly well, and the breathing was not very much disturbed. After the cavity and surrounding parts had been dried the bony flap was replaced and secured by silk sutures; some of them passing through the periosteum to keep the ribs in position. The skin and muscular flap were then sutured with horsehair. A small drainage-tube was inserted at the anterior inferior angle of the wound passing down to where the incision into the lung had been made, the wound dressed, and ⅛ gr. morphia injected hypodermically.

For the first few days after the operation the child was very irritable and restless. He appeared to be in a good deal of pain, crying out suddenly. The breathing was irregular and jerky, with an occasional slight cough. He was given rather frequent small doses of morphia, but rarely slept more than a very short time. Very little discharge came from the tube, and it was not offensive after the second day. The temperature began to rise immediately after the operation, and was constantly between 101° and 102° F., the pulse and respirations being very rapid (150 to 160 and 44 to 50 respectively). On the seventh day the child appeared to be much better and more comfortable, the breathing easier and less jerky. Air could be heard entering the upper and back part of the lung. A few rhonchi were heard over the back of the left lung, the temperature falling slowly. He was taking food well, and sleeping fairly well without narcotics. From this time onwards recovery was only interrupted by the occurrence of two small spots of necrosis at the anterior ends of the divided ribs. On August 6 an anæsthetic was given, and these removed, after which the wound healed completely. There remained some weakness of the upper part of the right rectus muscle, the result of division of the intercostal nerves. Now the chest moves well, the wound is soundly healed, and air can be heard to enter the greater part of the lung. There is no scoliosis, the child appears to be very well, and not much the worse for his experience.

The literature of foreign bodies in the trachea, bronchi and lungs is extensive, and I have not been able to go through it very thoroughly, but as far as I have gone there seem to be few recorded successful cases of direct incision into the lung tissue through an unaffected pleura for the removal of a foreign body. The presence of an empyema completely alters the physics of the chest, and the operation under those circumstances would be easier and probably more free from immediate risks. The operation I have reported shows how much safer than is usually supposed is the deliberate opening of a healthy pleura and collapsing of a lung, and how such a collapsed lung may be handled and incised with safety. It had been my intention if I succeeded in finding and removing the pin to close the wound completely and aspirate the air from the pleura; the presence, however, of the pus which followed the extraction of the pin compelled the use of a drainage-tube. The case shows, too, I think that if the operation is rendered necessary by failure to extract a foreign body *per vias naturales* the sooner it is performed the better, so as to avoid the risk of suppuration in the lung.

I must not conclude without a word of thanks to the Sister of Broderip Ward, my house surgeon, Mr. Saunders, and dresser, Mr. De Souza, to whose unremitting care and attention a great deal of the successful issue of the case was due.

DISCUSSION.

The PRESIDENT expressed the congratulations of the Section to Mr. Kellock on the attainment of his brilliant result.

Mr. C. R. C. LYSTER demonstrated, by the epidiascope, a skiagram of the lung with the pin impacted, showing the head of the pin pointing downwards. He located the position occupied by the head of the pin, and at the operation it was found within a very short distance of the spot assigned. Three attempts were made to extract the pin, under X-rays. The forceps could be seen holding the pin, but the result of traction was to draw the pin into a horizontal position, apparently dragging lung tissue with it.

Dr. DE HAVILLAND HALL joined in the congratulations to Mr. Kellock on his result. The last case he saw was almost precisely similar. He was asked to see the case in consultation with Sir StClair Thomson, the lady having swallowed a shawl pin. It had been localized by X-rays, and he was called in to settle what should be done if Sir StClair failed to extract it by bronchoscopy. On his past experience of foreign bodies in the lungs, he recommended that should failure ensue the chest should be cut down upon and the pin extracted in the way practised by Mr. Kellock in the present case. The friends agreed to this. But fortunately the second attempt was successful, the pin being seized by the point and removed. Two days later the patient was quite comfortable and the chest had resumed its normal condition.

Clinical Section.

January 10, 1913.

Sir WILLIAM OSLER, Bt., F.R.S., President of the Section, in the Chair.

Diphtheritic Hemiplegia.

By J. D. ROLLESTON, M.D.

BOY, aged 5, was admitted to hospital on September 3, 1912, with very severe faucial and nasal diphtheria on the fourth day of disease. Twenty thousand units of antitoxin given on admission, and another two doses of 24,000 units on each of the following days. Cardiac dilatation and irregularity occurred on the sixth day and was accompanied by vomiting, which persisted till the fifteenth day, and by enlargement of the liver. Knee- and ankle-jerks lost on twenty-first day. Sudden onset of right hemiplegia and aphasia on twenty-first day. Palatal palsy, shown by regurgitation of fluid through the nose, occurred on twenty-ninth day, ciliary palsy on thirty-third day, and pharyngeal palsy, necessitating rectal and nasal feeding, from thirty-sixth to fifty-eighth day. Other palsies noted were paresis of rectal and vesical sphincters, and of the diaphragm during the occurrence of pharyngeal palsy. Albuminuria from ninth to forty-first day; no serum phenomena beyond slight urticarial rash of one day's duration on nineteenth day.

Condition on discharge from hospital on December 23, ninety-four days after onset of hemiplegia: Some spasticity of right arm and leg, and slight right facial palsy, extensor plantar response, ankle clonus, and combined movement of trunk and pelvis on right side. Intelligence intact, but speech limited to a few syllables. Can walk with very little support. When shown at the meeting on January 10 the boy had so far improved as to be able to walk alone with a slight limp. Otherwise his condition was the same as on his discharge from hospital.

Hemiplegia is a rare occurrence in diphtheria. Out of 9,075 completed cases of diphtheria admitted to the Grove Fever Hospital between

August, 1899, and September, 1912, only six, including the present one, developed hemiplegia. Four died and two recovered. A post-mortem examination was held on one case, a boy, aged 3, in whom right hemiplegia and aphasia occurred on the fourteenth day and death on the seventeenth day, and showed, in addition to cerebral embolism, two infarcts in the left kidney. In the second case, a girl, aged 6, right hemiplegia occurred on the twenty-ninth day and was of only three hours' duration, being probably of uræmic origin. In a third case, a girl, aged 3, left hemiplegia occurred on the eighteenth day of disease and was followed by embolism of the lower limbs the next day, when death occurred.

The other two cases I reported in the *Review of Neurology* [11] in 1905, when I was able to collect sixty-five cases from literature. Since then eleven more cases have been recorded: Escherich [3] (two cases), Hecht [4], Moltchanoff [10], Jones and Hamilton [7] (two cases), Humphry [5], Jeanneau [6], Burrows [1] (two cases), and Cubello [2], so that there is now a total of eighty on record, including the present case and the three I have just mentioned.

The majority of the cases occur in the first six weeks of the disease, the time of predilection being the end of the third or beginning of the fourth week. In all the cases in which details are given the diphtheritic angina was severe, and in most cases ordinary diphtheritic paralysis preceded or followed the onset of hemiplegia.

Diphtheritic hemiplegia is usually of embolic origin; in twelve out of eighteen autopsies embolism has been found. Embolism is probably more common in diphtheria than in any other acute contagious disease, being due, as Marfan has pointed out, to an apical endocarditis. The embolism may be found not only in the cerebral circulation, but in the kidneys, spleen, lungs, and even the abdominal aorta, as in Marfan's case. In a case recently shown by me at the Children's Section, of gangrene of the leg following diphtheria, the condition was due to femoral embolism, and was probably associated with embolism in the spleen, kidney, and other limb, though, as the child recovered, the diagnosis of multiple embolism could not be verified.

The prognosis in diphtheritic hemiplegia, as in hemiplegia generally following acute infections, is unfavourable as regards complete recovery. There are only four cases on record in which diphtheritic hemiplegia entirely cleared up (Levi). In addition to the hemiplegia another remarkable feature of the present case is that, in spite of the enormous doses of antitoxin given (68,000 units), the serum manifestations were very slight, as is the rule in malignant diphtheria. My only other case of hemiplegia which recovered had 72,000 units of antitoxin, but had no other serum complications beyond urticaria of two days' duration.

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DISCUSSION.

Dr. MIDELTON asked to what extent Dr. Rolleston considered that the diphtheria bacillus lingered in these patients, and what part they played in keeping up the condition. Could any means be adopted to get the bacilli out of the system?

Dr. GALLOWAY said, in reference to the prognosis, that he had seen that day a boy, aged 6, who had recovered in a remarkable manner from complete right hemiplegia, which developed rapidly, almost suddenly, thirteen months ago. The boy had walked across the ward very alertly, had moved his right arm and hand in his efforts to shake hands, and some recovery of the face also was noticeable. There had been an illness of somewhat indefinite character for a fortnight or three weeks preceding the hemiplegia. The hemiplegia was noticed one morning by his nurse and had occurred, apparently, during the early morning. The previous illness was probably of a septic nature and had been associated with troublesome diarrhœa, but there was no evidence of diphtheria, nor of any infective blood condition so far as could be ascertained. There was no endocarditis. Yet it could not be doubted that the hemiplegia was of vascular origin, and the vessel blocked was probably the main branch of the left middle cerebral artery. The cortical centres, including the speech centre, were no doubt affected. In spite of this grave injury and the very serious paralysis which ensued, the boy had made a gradual recovery up to the present time. His general health had steadily improved, and with the exception of the paralysis and the spastic condition of the affected muscles, especially those of the right upper arm and hand, the boy appeared to be in good health. The prognosis, therefore, in this case, had been a good one. No doubt, in the majority of cases the prognosis could not be looked upon as being favourable, but he hoped that in Dr. Rolleston's case as good a result might be obtained by continuous care and treatment as in the case he had quoted.

Dr. ROLLESTON, in answer to a question by the President, replied that in every case there was a definite cardiac lesion, but in only one case was there an autopsy made to verify the diagnosis. He did not think the bacilli were directly responsible for the hemiplegia, but only secondarily, via the vascular system. There were a large number of methods of trying to get rid of the bacilli from the system, but they were mostly futile. Recently he had tried spraying the throats with staphylococcic cultures, which had been done by men in the States, but that was not entirely free from risk.

Non-syphilitic Arteritis Obliterans ("Thrombo-angeitis" of Leo Buerger) with Intermittent Claudication of the Left Lower Extremity.

By F. PARKES WEBER, M.D.

THE patient, M. M., a Russian Jew, now aged 47, presents the characteristic symptoms of the type of chronic arteritis obliterans which, in Germany, England and America, has been chiefly or almost exclusively observed in male Jews, aged between 30 and 52, who have emigrated from Russia. Syphilis apparently plays no essential part in the ætiology of such cases, and the real cause of the vascular disease remains unknown, though tobacco-smoking may be suspected of being a contributory factor. Most of the patients have been free cigarette-smokers, and many of them have been employed in cigarette factories, where they could obtain cigarettes without paying for them. The characteristic symptoms of the disease, as illustrated by the present case, are: (1) Redness or cyanosis of the foot, when it is allowed to rest in a dependent position; (2) pallor of the foot on movement of the ankle-joint; (3) intermittent claudication with feeling of cramp or pain in the muscles of the calf or instep on walking for a few minutes (Walton and Paul have termed this "angina cruris" when, as in most cases, one of the lower extremities is the part affected); (4) absence of pulsation in the arteries of the foot, notably in the dorsalis pedis artery. In the present case the symptoms are of nine years' duration, and the patient has been shown before the Section on December 13, 1907, and January 14, 1910.[1]

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[1] See also F. P. Weber, *Lancet*, London, 1908, i, p. 152.

The case is again brought forward to show that the condition of the affected limb is no worse than it was in 1907, although the arterial affection was for some time (in 1909 and 1910) complicated by superficial phlebitis, as in several other recorded cases, especially those described by Leo Buerger under the title of "Thrombo-angeitis obliterans." In the amputated leg from a similar case (1904) I found in one of the sections of the affected vessels that both the artery and one of the veins accompanying it were occluded by organizing thrombi a fact which shows that the venæ comites of the arteries may be likewise affected, and may be affected when the superficial veins are apparently unaffected.

In the present case the disease has been rendered more severe by the occasional occurrence of chronic "ischæmic" ulcers. Such "ischæmic" ulcers, when they occur in cases like the present one, give rise to severe local pain and tenderness, which keep the patient awake at night, like the pains of commencing dry gangrene of a limb; and unfortunately, unlike the typical cramp-like muscular pains of the intermittent claudication, they cannot be avoided by keeping the muscles at rest, though they necessitate prolonged confinement to bed and the use of opiates, such as pantopon.

In this class of cases Nature endeavours to establish a *capillary* collateral circulation in default of sufficient *arterial* collateral circulation, and that accounts for the hyperæmia and turgidity of the affected foot, when it is allowed to hang down—a condition which has been confused with "erythromelalgia" of nervous origin. This explains why dilating the blood capillaries in the foot by the temporary application of negative pressure (apparatus after Bier), or by the local application of warmth, or by allowing the foot to hang down, may temporarily ease the pain and ultimately increase the efficiency of the capillary circulation in the part.

In regard to treatment, besides prolonged rest in bed (which is absolutely necessary in the bad cases, especially when there is any ischæmic ulceration) and the application of various methods to induce hyperæmia (by means of apparatus for negative pressure after Bier, and the local application of warmth), and possibly also the use of electrical methods, I believe that prolonged courses of iodipin (such as have been employed in the present case) really do exert some beneficial, though not rapid, effect. As local applications to the ischæmic ulcers, xeroform, calcium iodide ointment (5 per cent.), Scharlachrot ointment (8 per cent.), and an application containing balsam of Peru with a minute quantity of silver nitrate, seem to be useful.

In some cases the disease becomes quiescent, or is arrested, before the period of "ischæmic ulcers" is reached, and then, although the intermittent claudication may persist, there will not be the dreadful nocturnal pain of threatened gangrene. It is this pain, if not the commencement of actual gangrene or septic infection of the part, which is specially likely to call for amputation, but the present case shows that occasionally a patient may safely pass through the painful period and escape amputation for several years at least.

I have had the opportunity of seeing a good many cases of the same affection amongst the poor Jews (from Russia) of the East End of London, but in only one case have I seen it in a Jew of the well-to-do classes. The patients are all males, and apparently free from syphilis. In the present patient (M. M.) the blood serum was twice examined at the Lister Institute (1910 and 1911), and on both occasions was found to give a negative Wassermann's reaction for syphilis. In some cases amputation has been necessary, but in other cases the affection seems to become quiescent. In two cases in which both lower limbs were badly affected (amputation on both sides necessary) the radial artery in one of the upper limbs was found to be obstructed and pulseless, though no definite circulatory or other symptoms had been as yet caused thereby.

In regard to diagnosis, it must be remembered that the single symptom expressed by the term "intermittent claudication," in one or both lower extremities, may, undoubtedly, occasionally occur in the apparent absence of any organic arterial disease sufficient to account for it. Apart from Dejerine's cases of "intermittent spinal claudication,"[1] in a few cases the intermittent claudication of the lower extremities has been supposed to be due only to angiospasm (H. Oppenheim,[2] H. Curschmann,[3] and others), and in a few other cases no satisfactory explanation of the symptom can be obtained (by ordinary clinical methods of examination). Oppenheim[4] has found intermittent claudication combined with neuroses or psychopathic conditions, and occurring in persons in whom the stigmata of degeneration (e.g., medullated nerve-fibres in the retina, malformed fingers, &c.) pointed to the congenital disposition; and he says that his experience has been confirmed by Goldflam, Higier, Idelsohn, and others. In this connexion it may be noted that a son of the present patient (M. M.) is a well-grown lad aged 20, but since the age of 6 or 7 he has suffered from attacks of headache and vomiting, recurring about every four to six weeks on the average. He (the son) also has slight chronic enlargement of the spleen, and his blood serum gave a negative Wassermann's reaction (von Dungern's modification) when tested in July, 1911.

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[1] See Dejerine, *Presse Méd.*, Par., 1911, xix, p. 981, and Dejerine's earlier papers of 1906 and 1909 on the subject; P. Sabrazès, *ibid.*, 1906, xiv, p. 677; F. F. D. Reckford, *Amer. Journ. Med. Sci.*, Philad., 1912, cxliv, p. 721; S. Gavazzeni, *La Clin. Med. Italiana*, Milano, 1906, xlv, p. 165; G. Poggio, *Gazz. degli osped.*, Milano, 1908, xxix, p. 138.

[2] H. Oppenheim, "Text-book of Nervous Diseases," English translation by A. Bruce, 1911, p. 587.

[3] H. Curschmann, *Münch. med. Wochenschr.*, 1907, liv, p. 2519.

[4] Oppenheim, *op. cit.*, p. 587.

One feature in the present case is perhaps worthy of special attention. In my account of 1907[1] I described it in the following words: "If the patient then forcibly flexed and extended the ankle-joint a few times the skin of the foot, in less than a minute, lost its congested look and became blanched and alabaster-like." This phenomenon may still to some extent be observed. A somewhat similar blanching of the hands on movement was noted by Oehler in a case of "intermittent dyskinesia of the arms,"[2] and Erb[3] has proposed to called the phenomenon in question "Oehler's symptom." I have likewise alluded to the symptom elsewhere,[4] and S. Goldflam, of Warsaw,[5] has specially directed attention to it. The symptom is, however, in some cases very little noticeable, or apparently absent. In some cases it may be rendered more definite by first immersing both the patient's feet in a hot footbath so as to produce a preliminary artificial hyperæmia.

DISCUSSION.

Dr. PARKES WEBER said he had furnished rather elaborate notes because the case was otherwise difficult to demonstrate. A gentleman present had kindly brought a skiagram from a case of intermittent claudication showing calcification in the wall of one of the arteries of the leg. He took it that in all chronic diseases of arteries in the extremities there might ultimately be some calcification, but this particular type of case (Dr. Weber's case) was not associated with calcification *at first*. In the present case the most troublesome pain had been associated with the ischaemic ulceration; it had been especially felt at night, and had prevented sleep. The patient no longer had that pain, but if he tried to walk much the intermittent claudication still occurred. Those who read Leo Buerger's contributions on the pathology of the subject would regard the prognosis as very bad. But he (Dr. Weber) found that some of these cases did not get worse, at least for a long time; thus minor cases might remain minor cases, and even severe cases (like the present one) might escape amputation, at least for a long time, if the patient was content to put up with the intermittent claudication.

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[1] *Proc. Roy. Soc. Med.*, 1908, i (Clin. Sect.), p. 44.

[2] Oehler, *Deutsch. Arch. f. klin. Med.*, Leipz., 1907, xcii, p. 154.

[3] Erb, *Münch. med. Wochenschr.*, 1910, lvii, p. 1182.

[4] F. P. Weber, *Proc. Roy. Soc. Med.*, 1910, iii (Clin. Sect.), p. 97; 1908, i (Neur. Sect.), pp. 50, 102.

[5] *See* Goldflam's remarks on this subject in the *Münch. med. Wochenschr.*, 1910, lvii, p. 1747.

Dr. JOHN BRUCE (Grimsby) said he had a similar case under care at present, which brought out clearly the kind of pain of which Dr. Weber spoke. The patient was an Englishman, aged 71, and had had symptoms of intermittent claudication for the past four or five years, coming on in the usual way when walking. One day, during his holiday at Torquay last year, he walked up the steps to the church, and next morning, on awakening, he found his left leg had gone numb. When he went for a walk that day he was brought up at once by most severe pain in the left leg and a bad attack of claudication. He saw the patient on October 6, when he had a cyanosed condition of the foot, and pallor came on after movement. There was most distressing pain at night, so that a couple of grains of morphia had to be given to induce sleep. He could procure some ease by jumping out of bed and allowing the foot to hang down. There was no evidence of syphilis. No pulsation was evident in the popliteals or tibials, but pulsation in the femorals was very marked. He had also had pain in the right leg and cramping pains in the right arm. During the last year he had had sudden, sharp pain in the abdomen, lasting ten minutes or more and then going off. He had been a steadily heavy smoker—about 4 oz. a week since he was aged 15, varied with strong, black cigars. On the left foot he was developing a blue patch, which seemed to be the seat of origin of the pain.

Dr. BEZLY THORNE said that in 1902 a man, aged 65, presented himself for treatment on the ground of angina pectoris coming on after exertion. The heart sounds were so feeble that they were scarcely audible. His blood-pressure was taken with the larger Hill and Barnard instrument, with one band on the armlet, which gave readings about 20 mm. higher than the armlet with three. There was a diastolic pressure of 180 and a systolic of 220, equal to 160 and 200. A few months later his anginous pains were almost gone; the diastolic pressure had dropped to 140 and the systolic to 170, and by the following year the angina had practically ceased. He next came with paroxysmal tachycardia, generally associated with distension of the stomach or of the colon. He again saw the patient in 1904, when the sounds were scarcely audible; there was no anginous pain, but he had sighing respiration. In 1906 he again saw the patient, after he had consulted Sir Victor Horsley on account of pains in his legs, which had been said to be due to gouty changes in the vessels of the legs. He had disabling pain in the right calf after walking 50 yards; the right foot was colder than the left, and he said that the more slowly he walked the longer the pain was delayed. In February, 1906, the right leg had become worse, and the left better, but he was quite comfortable while in a state of repose, and there had been none of the patches of discoloration which existed in the case under consideration. He found no pulsation in the arteries of either foot, but on the left side there was extreme tenderness of the calf on bilateral pressure, and on the right side similar tenderness of the soft tissues adjacent to the outer edge of the tibia. The patient, now aged 75, was last seen in October, and could then walk a mile without suffering pain. When it came on it lasted only about a minute and then he was able to resume his walk. He was in good health, and all he now complained of was an occasional attack of tachycardia in the night, associated with gastric dilatation. With such a history one was justified in forming a favourable prognosis. The feet, during the attacks, had at times become white and icy cold.

Mr. HAMPSON exhibited some skiagrams of a case akin to that under discussion. The areas he wished to demonstrate, though very distinct, did not show densely, as the man was very muscular, and his calves were 5 in. in diameter. The patient was an Englishman, aged 50, had always been very athletic, and had done much hard walking. Two years ago he began to have these troubles. At first, when shooting, he could walk the pain off, whereas now he could not walk more than 100 yards without the pain stopping him, when he also had stiffness and cramp. There was a hardness of the calves of both legs, but chiefly the right, and there one could detect the presence of calcareous plates. The feet and calves became partially numb, and the skin of the calf and foot was pale; but during the last six weeks of treatment those symptoms had disappeared, and under stimulation the part was flushed with blood, though he continued to have claudication. No pulsation was observable in either foot.

Dr. GALLOWAY said that the cases described by the three previous speakers could not be regarded as being of the same nature as the patient shown by Dr. Weber. So far as could be judged by the ages of the patients and the description of the symptoms given, those patients had suffered from disease due to the more ordinary varieties of arterial degeneration, with which we had many opportunities of becoming familiar. Dr. Parkes Weber's case shown that evening was in an entirely different category, and no doubt Dr. Weber in his reply would emphasize the marked lines of differentiation. He would only mention that in some of the cases described by Dr. Weber and Dr. Buerger the symptoms had commenced in young adults, even at the age of 20 and 21, passing on to well-marked failure of nutrition and even gangrene of the extremities. The large majority of the cases in which this peculiar disease of blood-vessels had been described occurred in Hebrews. About two years ago he had taken the opportunity of carefully reading the descriptions given by Dr. Leo Buerger of the cases coming under his observation at the Mount Sinai

Hospital in New York, these cases being almost all in Jews. It was a little difficult to make out from Dr. Buerger's description the exact nature of the histological changes, which occurred, it must be remembered, not only in the arteries but also in the veins and in the smaller blood-vessels. In addition to the actual inflammatory changes in the walls of the vessels occurring in these cases of "thrombo-angeitis obliterans," a process of organization and transformation into fibrous tissue of the thrombi filling the blood-vessels apparently occurred. Dr. Buerger had had the opportunity of making anatomical observations on the limbs of an extraordinarily large number of patients in whom it had been found necessary to resort to amputation for the relief of symptoms. But he believed in this country that Dr. Weber was the only one who had been able to make a complete investigation of the vessels of an amputated limb in this disease. It would be of interest to the Section, and would serve to emphasize the unusual characters of this type of disease of the blood-vessels, if Dr. Weber were able to show histological specimens of the blood-vessels in such a case. He thought that Dr. Weber's patient had chosen the better course. As the result of patience and care it was evident that a large amount of collateral circulation had been established, that he had saved his limbs, and that even the agonizing pain which was such a striking feature in the cases under review had been greatly ameliorated.

The PRESIDENT (Sir Wm. Osler, Bt., F.R.S.) said Dr. Parkes Weber's case was in a class apart from the forms which had been mentioned by the speakers. They occurred in males, usually Russian Jews, tobacco-smokers, and the disease had no relationship with erythromelalgia, the syphilitic and the senile cases referred to by several speakers. It was a very extraordinary malady. A point of special interest was that this man had saved his leg. In many of the patients at Mount Sinai Hospital, New York, amputation had been performed. Dr. Buerger, who had described the affection, had shown it to be a phlebo-arteritis. The malady occurred also in this country in exactly similar conditions, all the cases being also in Russian Jews.

Dr. PARKES WEBER replied that practically all the cases he had seen were in Jews, and Jews from Russia.[1] He would like to hear more discussion on intermittent claudication due to other kinds of arterial disease, such as those cases which were due to syphilitic arterial changes and the so-called senile cases. In some cases showing the symptom—"intermittent claudication of the lower extremities"—he believed the cause had not yet been discovered, and he believed that during life the existence of definite arterial disease, or the nature of the arterial change, might remain doubtful.

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[1] In regard to microscopical examination of the blood-vessels from amputated limbs, Dr. Weber referred to his papers with Dr. Michels, *Brit. Med. Journ.*, 1903, ii, p. 566, and *Trans. Path. Soc. Lond.*, 1905, lvi, p. 223.

Chronic Acholuric Jaundice, with Anæmia, slight Splenomegaly, and Nervous Disorder.

By F. PARKES WEBER, M.D.

THE patient, E. S., is a married woman, aged 43, with a pale, sallow complexion and a slight icteric tinge of the conjunctivæ. There is no emaciation. The spleen can be felt just below the costal margin during inspiration, but is evidently only slightly enlarged. The liver is apparently not enlarged. The fæces are normally coloured. The urine contains urobilin, but no bilirubin. Blood examination (Dr. Bauch): Hæmoglobin, 55 per cent.; red cells, 4,000,000 to the cubic millimetre of blood; white cells, 4,000 (lymphocytes, 16·6 per cent., polymorphonuclear neutrophiles, 83·4 per cent.). Microscopic examination of blood films shows nothing abnormal, except very slight anisocytosis and polychromatophilia. The resistence of her red blood corpuscles towards graduated hypotonic solutions of sodium chloride is normal. The blood serum gives a negative Wassermann's reaction for syphilis (Lister Institute). Von Pirquet's cuti-reaction for tuberculosis is slightly positive. Her teeth are in bad condition. The anæmia was rather more marked two months ago before chalybeate treatment was commenced.

Nervous symptoms: The patient somewhat resembles a case of paralysis agitans in her gait and shakiness and facial immobility. Loss of vision is her chief complaint, but ophthalmoscopic examination shows nothing abnormal, and her sight varies so much from time to time that the ocular symptoms are perhaps entirely of functional origin (Dr. C. Markus). There is no nystagmus, and her pupillary reactions are normal. Her knee-jerks and Achilles-jerks are greatly exaggerated, and there is a tendency to knee and ankle "trepidation," as in many functional nervous cases. Her plantar reflexes are of the flexor type, but exaggerated. Her sensation is normal. She says that she sometimes sees double. Since about September, 1911, according to her own account, she has had an unsteady gait, and first noticed the shakiness in her upper extremities about six months later. She has complained of loss of sight since about July, 1912.

Case of Acholuric Jaundice after Splenectomy.

By W. Essex Wynter, M.D.

M. W., aged 22. This patient was shown to the Section on Friday, November 8,[1] jaundiced, as she had been, in varying degrees, since birth, and exhibiting a large hard spleen which caused dragging pain in the left side. After three weeks' stay at Clacton, splenectomy was performed by Sir J. Bland-Sutton on December 11, 1912. The incision was nearly vertical, about 2 in. to the left of the middle line. The spleen was much enlarged, weighing 3 lb., and showed a deep sulcus due to the gastro-splenic omentum, simulating the hilum. All the mesenteric attachments were clamped and divided, the vessels being at once ligatured. The spleen was then removed. Not more than 2 oz. of blood were lost, chiefly from the spleen itself. The under surface of the diaphragm was found healthy and free from adhesions. The incision was approximated with "through-and-through" stitches; three buried sutures being left. Recovery was rapid, temperature varying from 99° to 100° F. for a week and then remaining normal. Jaundice, which had diminished during the stay at Clacton, had been entirely absent since the operation, and the pains in the left side had disappeared.

Laboratory Report by W. T. Hillier.

Blood Count.

November 8, 1912.		January 8, 1913.	
Red cells	2,710,000	Red cells	3,365,000
White cells	**13,000**	**White cells**	**7,000**
Hæmoglobin	52 per cent.	Hæmoglobin	84 per cent.
Index	**0·86**	**Index**	**1**
Nucleated red cells	**78 per c.mm.**		

Differential White Count.

Lymphocytes	35·4 per cent.	Lymphocytes	40·8 per cent.
Hyaline and transitional	3·6 ,,	Hyaline and transitional	5·6 ,,
Polymorphonuclears	59·2 ,,	Polymorphonuclears	49·8 ,,
Eosinophiles	1·8 ,,	Eosinophiles	3·4 ,,
Mast cells	0 ,,	Mast cells	0·4 ,,

Blood laked in 0·75 per cent. saline solution against 0·45 per cent. control

No urobilinogen or excess of urobilin in the urine

Blood laked in 0·45 per cent. saline solution against 0·375 per cent. control

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[1] See *Proceedings*, p. 33.

DISCUSSION.

Dr. WYNTER said he felt he owed it to the Section to exhibit this case again, after splenectomy, since it was through the encouragement afforded at the previous meeting that he had been emboldened to recommend such a drastic mode of treatment. The result was in every way satisfactory—jaundice had disappeared, as had also the sideache, and there was already definite improvement in the blood and general condition.

The PRESIDENT asked if Dr. Wynter had found a record of any other such case in the literature. He was himself told of one in Rome last year, and in that case recovery ensued. There were many instances of the disease in which splenectomy was not indicated and where the patients lived on to old age, without anæmia.

Dr. HECTOR MACKENZIE said that when Dr. Wynter first showed the case, reference was made to a case under Dr. Box, in which splenectomy was performed and benefit followed, but, unfortunately, the patient died shortly afterwards.

Dr. PARKES WEBER said he referred recently to a paper by Banti on "hæmolytic splenomegaly,"[1] in which good results had been claimed by the help of the operation of splenectomy. Undoubtedly splenectomy had been performed by foreign surgeons in a few cases regarded as hæmolytic splenomegaly (with anæmia and chronic acholuric jaundice). The whole matter required careful analysis and inquiry.

Dr. THURSFIELD said he could speak of a case of splenectomy for this disease, that of a boy aged 9, who was under his care at Great Ormond Street Children's Hospital. He had always been yellow in colour, and when he came under his care his spleen was at the level of the umbilicus and he had all the other signs of acholuric jaundice. There was no bilirubin in his urine. The serum had a yellowish tint, suggestive of the presence of bile. He did not say it was due to bile, because he believed bile had not been demonstrated in the serum of these cases. The patient exhibited abnormal fragility of his red cells. He was encouraged by the observations of some of his colleagues at consultation to have the spleen removed. Mr. Tyrrell Gray performed the operation in October and the boy had done very well. As he lived at Southend he had not yet been shown, but he would exhibit him at a future meeting of the Section for the Study of Disease in Children. Though the boy had improved so much and lost all appearance of jaundice, the fragility of his cells persisted. He had done a large number of fragility experiments in cases of acholuric jaundice and chronic obstructive jaundice, but in cases of the former, with one possible exception, there had been nothing like the normal fragility of red cells. These cases were extraordinarily interesting, because he did not think anyone had any idea of the cause of hæmolysis. He did not think the splenic enlargement had anything to do with the latter, though he could readily regard it as a sequence of it. One of the grounds he had for that belief was that in obstructive jaundice, even in the very early stages, the resistance of the blood corpuscles was so enormously raised that it was difficult to believe that a change could take place within a few days of obstructive jaundice, without some much more complicated mechanism being involved than a mere hæmolysis or auto-hæmolysis of splenic origin.

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[1] *See* Guido Banti, "La Splénomegalie hémolytique," *Sem. Med.*, Par., 1912, xxxii, p. 265.

Dr. GORDON WARD said that, including those mentioned that evening, there were seven cases which had been operated upon, and only one had not done well under the operation—namely, Dr. Box's case, in which an abscess developed in the stump of the spleen and the patient died.

Cases of Pulmonary Tuberculosis before and after Gymnastic Treatment.

By FILIP SYLVAN, M.D.

Case I.—E. G. T., aged 37. Eleven years ago he had very severe subacute pneumonia, and some time after this he had very severe hæmorrhages so that his life was despaired of. In 1902 he was three or four months in Ventnor Sanatorium. He was away from work one year and three months. Since then he has never been well; coughs all the year round, and expectorates chiefly in the morning. Vital capacity, 2,200 c.c.

Case II.—W. P., aged 31. About thirteen months ago he began to feel weak and had a hæmorrhage (about a teacupful of blood). Last October he had another hæmorrhage and went to Brompton Hospital, where the diagnosis was pulmonary tuberculosis of upper lobes (both sides). Tubercle bacilli were found in his sputum. On November 26, 1912, he started gymnastic treatment. Over upper part of right lung dullness on percussion; over upper and middle lobe, crepitation; over lower lobe, prolonged expiration; over upper part of left lung, dullness on percussion; upper lobe, crepitation; lower lobe, prolonged expiration. Vital capacity, 3,100 c.c. January 6, 1913: Vital capacity, 5,500 c.c.

DISCUSSION.

Dr. BEZLY THORNE said he could mention a case bearing on those shown, though, as far as was known, it was not a case of tuberculosis. The patient had an infarct in the right lung and very severe hæmorrhage. The man was so much worse that four days after seeing him Dr. Thorne was summoned to him again, but as he was laid up with influenza Sir Richard Douglas Powell kindly went for him, and reported that he considered the case as absolutely hopeless, that the hæmorrhage had increased, the lung was out of action, and he feared gangrene. Six months later Dr. Thorne saw the patient again and found him quite convalescent, but consolidation of the right lung was complete. As there was a good deal of trouble about the heart he sent the man to Nauheim, and four weeks later he arrived there himself. He found the patient had had baths and was now being given gymnastic exercises. Standing behind the patient, it was almost impossible to tell, on full inspiration, a difference between the two sides, and vesicular breathing could be heard all over the lung. It was one of the most surprising things he had ever seen.

Dr. HECTOR MACKENZIE said he would have liked to know more precisely what benefit the treatment was supposed to have conferred. Practice would increase anyone's vital capacity as much as had occurred in the second case. To show the value of the treatment, cases should be exhibited before the treatment, and then brought up again after the method had been carried out for some time.

Dr. SYLVAN replied that his intention was to show the two cases again later on, so that members could see what improvement had taken place.

Actinomycosis of the Liver in a Syphilitic Subject.

By H. D. ROLLESTON, M.D.

A RAGSORTER, aged 29, who contracted syphilis eight years ago, had a fall on the right side one and a half years ago. About June 1, 1912, burning pain began in the hepatic region, and about June 10 he noticed a swelling in this situation. On admission there was a firm tumour measuring 3 in. by 2½ in. below the right costal arch, fixed so that it did not move with respiration. There were old scars on the face and neck, and an enormous white serpiginous scar over the front of the chest. The spleen was not palpable. The urine contained albumin. The heart and lungs were normal. As the Wassermann reaction was strongly positive and he had irregular fever, the condition being thought to be gummatous, iodides were given. The tumour softened, and on being incised on June 30 gave exit to pus containing streptothrix mycelium. On July 18 the abscess was opened up and thoroughly scraped. Shortly after this operation there was a small hæmatemesis which contained actinomycotic granules. The patient, who is not very tolerant to iodides, has had various modifications such as iodipin and sajodin, and has also had an autogenous streptothrix vaccine prepared by Dr. Slater. The liver is much enlarged, and there are the scars of sinuses which from time to time discharge pus containing a Gram-positive streptothrix with well-developed clubs; some of the mycelium resemble streptococcic chains. The urine constantly contains albumin, the temperature is irregular, and the liver, which has varied in size, has occasionally shown markedly tender spots, as if from collections of pus.

Case of Nephrectomy for Hydronephrosis Thirteen Years after Nephrolithotomy.

By John D. Malcolm, F.R.C.S.Ed.

This patient, a single woman, who was obviously of neurotic temperament, first came under the writer's observation in 1896, when her age was 26. At the age of 9 and again at the age of 18 she was said to have suffered from a contraction of the flexor muscles of the left leg and thigh, from which she recovered. At the age of 16 she had an abscess, which was opened, and discharged for five months, leaving a scar on the back over the right lower ribs. From 1891 onward she had much pain in the left kidney region, and in 1894 this pain became worse and was accompanied by a rise of temperature for three and a half months. This exacerbation subsided, but it was followed by similar, though less acute, attacks from time to time. In 1895 pus was observed in the urine, and the pain then changed its character, becoming much less acute, but more constant. After this there was always pus in the urine, and the patient had least pain when she passed most pus.

The lungs and heart were healthy, the bowels were somewhat constipated. The right kidney was easily felt under the ribs, and it was not tender. In the left kidney region a searching examination was impossible on account of the pain it caused. The urine contained pus and blood cells and many crystals of oxalate of calcium. No tubercle bacilli were found.

The delicate, hysterical appearance of the patient and the history of an old abscess of uncertain origin did not encourage a resort to surgical treatment, and therefore it was not until July, 1898, that the kidney was exposed by an incision in the semilunar line. The right kidney was small and normal to palpation. The left was larger and of irregular consistence. By a second incision in the left loin the stone shown was extracted with much difficulty. A large opening in the pelvis of the kidney was necessary, and even with this the stone broke in two places. The main mass of the calculus exhibited visible crystals, and a spur 1 in. long by $\frac{1}{4}$ in. in diameter projected from it into the ureter. The loin wound was drained.

The temperature rose to 101° F. the first night, but within a week it was practically normal. For a few days much urine escaped from the side, showing that there was considerable healthy kidney substance, although its irregular consistence and enlargement, when considered in the light of subsequent developments, seemed to indicate that there was even then some degree of dilatation. By the fifth day all the urine was passing through the bladder, and the patient made a good recovery.

The health was fairly good until thirteen years later, when she had several attacks of severe pain in the left kidney region, and feared that she had another stone. On investigation it seemed certain that the ureter was blocked, probably by a calculus. The urine was normal.

On October 27, 1911, the kidney was again exposed through the left semilunar line by removing the former scar. The right kidney was examined and found healthy. The left kidney was enlarged, and evidently a mere capsule of renal tissue was left. It was removed very easily. The patient made a good recovery, and her health afterwards was much better than it had been for many years. The kidney contained a clear fluid, and there was no stone in it. The ureter was healthy, except close to the kidney, where the most careful dissection failed to show any communication between it and the hydronephrotic kidney. It seemed clear that the suppurating surface induced by the projection of the rough stone into the upper part of the ureter healed when the stone was removed, but that a process of gradual contraction had been started, which some twelve years later caused an absolute closure of the ureter.

Removal of nearly Half a Kidney for Partial Hydronephrosis Sixteen Years after Nephrolithotomy.

By JOHN D. MALCOLM, F.R.C.S.Ed.

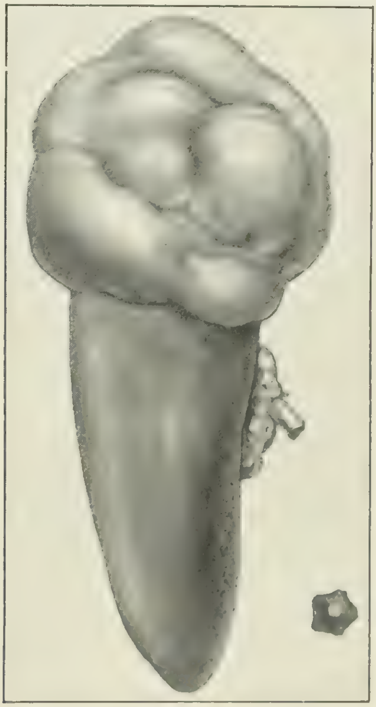
A HEALTHY married woman, aged 25, complained of attacks of severe pain coming and going suddenly, and first felt immediately after a confinement two years before her admission to the Samaritan Free Hospital in April, 1896. The attacks had increased in frequency. Examination showed a woman in all respects healthy, except that the right kidney, which was easily felt, was always tender, and especially so when the patient was in one of her attacks of pain. The specific gravity of the urine was about 1029. It was clear, and deposited albumin to about an eighth of its bulk on boiling, cooling and standing twenty-four hours. It did not contain sugar. There was a considerable light deposit consisting of round and squamous epithelial cells, with numerous oxalates; there were no distinct pus cells, no blood cells, and no casts.

On May 5, 1896, a stone, $\frac{7}{8}$ in. by $\frac{1}{2}$ in. in diameter, was removed through a loin incision, an abdominal incision being made to examine the other kidney, and to assist in the manipulations. The patient made a good recovery, and was free from pain for over fifteen years.

In the summer of 1912 she again sought advice because of a pain in the right kidney region, and in October she brought a stone which she had passed. It measured about $\frac{1}{8}$ in. in its longest diameter. The pain continued. It was quite different from that felt before the first operation, being a dull, constant ache, whereas the other was intermittent, very sharp, and came and went suddenly.

On November 22, 1912, the right kidney was exposed and brought out through a loin incision. It lay across the abdomen, and there was difficulty in deciding which was the upper end. One end, believed to be the lower, and the part opposite the pelvis were of normal shape and appearance, but narrow and elongated. The other end was larger than the healthy part, and evidently consisted of distended hydronephrotic, very thin kidney substance, with the usual lobulated appearance, as shown in the figure.

As the dilated portion of the kidney did not involve the pelvis it was decided to remove it, leaving the healthy part. The removal was effected by a straight cross-section through healthy renal tissue, and this displaced the smaller stone shown in the drawing. An approximation of the kidney substance was impossible until a wedge-shaped piece was excised, after which the cut renal edges were fairly easily brought together by catgut sutures. Hæmorrhage was arrested by ligatures and by the adjustment of the sutures. The loin was drained, very little urine escaped, and the patient made a good recovery.



This drawing is necessarily diagrammatic, as the healthy part is still in the woman's body. Its shape and the almost straight line of juncture between the healthy and abnormal parts are well shown. The small stone shown appeared to be the cause of the dilatation.

It seemed clear that the small stone had blocked or partially blocked the calices of one end of the kidney, and had caused the partial hydronephrosis.

After-histories of cases of nephrolithotomy were not very common, but in the writer's experience they were, so far as he knew, satisfactory. These cases, therefore, seemed of interest, and it was hoped that they might elicit notes of other cases. Sir Henry Morris, Dr. Abbe, of New York, and others had performed partial nephrectomies, but these were believed to have been rare operations.

DISCUSSION.

Mr. RAYMOND JOHNSON noted that the two primary operations in these cases were performed, in one fifteen years and in the other seventeen years ago, and inquired whether Mr. Malcolm still adopted the same method of dealing with an ordinary case of stone in the kidney. Did he open the abdomen through the semilunar line on the affected side, with the object of exploring the opposite kidney, and make use of the hand in the peritoneal cavity to facilitate the removal of the stone, after making the second incision in the loin over the kidney? He asked this because the treatment which had been adopted in these cases, in which such excellent results had been seen from secondary operation, was not the method usually adopted at the present time. If Mr. Malcolm still used the same method, Mr. Johnson asked whether he was satisfied with the investigation of the other kidney with the hand in the abdomen, and whether it compared in value with the investigation of the condition of the urine by catheterization of the ureters, and whether he thought that removal of the stone in such a case was as easy as by dealing with the kidney entirely from the loin. Mr. Johnson was interested to hear that in cases of chronic suppurative kidney, with the kidney adherent to the surrounding structures, Mr. Malcolm selected the abdominal route. All surgeons knew the great difficulty which might be met with in removing such a kidney by the lumbar operation, and he was particularly interested to hear that in such cases Mr. Malcolm had not hesitated to perform the abdominal operation and had not met with ill-results due to the septic condition of the kidney.

Mr. MALCOLM, in reply, said he would now attack an ordinary case of stone from the loin only. He was taught to open the abdomen so that he might examine the other kidney and make a very small loin wound, by the late Mr. Knowsley Thornton. But there were so many better ways of examining the kidney now that he did not open the abdomen to extract a stone. But there were some cases of suppurating kidney which he would rather attack across the abdomen; by that means, especially when the kidney was adherent on its inner side, one could get at the pelvis and vessels much more easily; and he thought that there was, in some cases, even less risk by this method than by operating through the loin. Moreover, more difficult cases could be successfully terminated.

Abscess in the Left Lobe of the Liver Ten Years after an Abscess in the Right Lobe.

By PHILIP TURNER, M.S.

A. D., AGED 38, was admitted to Guy's Hospital on December 19, 1912, for a pulsating swelling in the left epigastric region. In 1897, while serving in the Royal Marines, he was stationed on the West Coast of Africa. He had several attacks of malaria, but never had dysentery. He then returned to England, and has never since been abroad. In 1903 he had three or four shivering attacks, with pain in the right side, loss of appetite, and sickness, for which he was admitted to Guy's Hospital, under Dr. Frederick Taylor. He was then very anæmic and had an enlarged tender liver which extended on the right side 4 in. below the costal margin. Hepatic abscess was diagnosed, and Mr. Lane opened and drained an abscess which contained a pint and a half of chocolate-coloured pus, and which extended through the whole thickness of the liver. The temperature, which had been raised, fell to normal, and convalescence was uneventful. From that time until November last he continued quite well and strong, and had no illness except a slight attack of malaria five years ago. For the past seven years he has been a tram-driver. In the middle of November he had a severe blow in the abdomen from the handle of his brake, which "winded" him, but the effect soon passed off. A fortnight before admission he had a rigor; he, however, continued at work, and had a number of shivering attacks in the next three days. Then pain appeared on the left side and became so severe that he was obliged to rest in bed. The lump was first noticed a week before admission. He has never been jaundiced. On December 19 there was, in the left half of the epigastric region, a tumour about the size of an orange which formed a visible projection and pulsated so freely that he was considered by some to have an aneurysm. If examined while sitting up, however, though the tumour could easily be felt, the pulsation completely disappeared. The right lobe of the liver did not extend below the costal margin, and on percussion the tumour was dull and continuous with the liver dullness. The temperature was 102° F., and the patient looked very ill. Hepatic abscess was diagnosed and an incision made over the most prominent part of the tumour; the liver was adherent to the abdominal wall, and on incising it a large amount of pus, estimated at half a pint, escaped. A drainage-tube was inserted. The temperature next day was normal and convalescence has since been uneventful. A bacteriological investigation of the pus failed to show the presence of any organism.

DISCUSSION.

Mr. PHILIP TURNER desired to emphasize the prolonged interval between the patient's residence abroad and the appearance of the first abscess; also the long interval between the first and second abscess. There was no mention of any amœbæ being found in the pus in the first abscess. Owing to these liver abscesses the substance of the liver must be, by now, considerably destroyed.

Dr. J. G. EMANUEL (Birmingham) asked as to the possibility of these abscesses being suppurating hydatid cysts. Twelve years ago he described[1] a case of a suppurating hydatid cyst in the liver, which had recurred after an interval of ten years.

Dr. ESSEX WYNTER said he had had experience of a trooper who was in the Egyptian campaign and who had been struck by a horse's hoof in the liver region. He was admitted to Middlesex Hospital with an abscess of the liver, which discharged through the lung. The character of the sputum was typical, but he believed there were no organisms found. Possibly a severe blow might lead to necrosis of the liver and abscess.

Mr. TURNER replied that there was no trace of a hydatid cyst, though such was looked for.

Case of Polio-encephalo-myelitis associated with Optic Neuritis, Nephritis, and Myocarditis.

By ARTHUR F. HERTZ, M.D., and W. JOHNSON, M.D.

E. K., AGED 12½, was admitted into Guy's Hospital on March 5, 1912, for paralysis of the left side. This had developed suddenly the same day; the patient fell down, and on being picked up was found to have lost the use of his left arm and leg.

On admission his pulse was 54 and temperature 97° F. He was semiconscious; he could not move his left arm or leg at all, and there was some weakness on the left side of the face. His right pupil was smaller than the left. Both knee-jerks were present, the left being exaggerated; an extensor plantar reflex, diminished abdominal reflex, and ankle clonus were present on the left side only. There was some twitching of the right side of the face. Incontinence of urine and fæces was present. The heart was greatly dilated and a loud systolic murmur was heard at the apex, but there was no history of rheumatism, scarlet fever or previous cardiac disease. The case was diagnosed as cerebral embolism.

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[1] *Lancet*, 1900, ii. p. 1131.

The patient became fully conscious the next day. A tremor now occasionally affected the right arm and leg. There had never been any rigidity of the arm and only slight rigidity of the leg; but in the course of three days both became completely flaccid and the tendon reflexes disappeared, but the extensor plantar reflex remained. The knee-jerk and ankle-jerk disappeared from the right side also, but there was only a very slight degree of weakness in the right leg. The temperature had hitherto only been taken in the mouth, where it was normal; the rectal temperature, however, was now found to be 101·2° F. The pulse was still very slow, but the cardiac condition was otherwise unaltered. On March 8 complete paralysis of the left external rectus muscle developed. Lumbar puncture showed that the cerebrospinal fluid was under increased pressure; it contained numerous lymphocytes and polymorphonuclear cells in approximately equal numbers, but no organisms could be detected. Definite optic neuritis was present, which was more marked on the left side than on the right.

On March 16 some atrophy of the left side of the tongue was noticed. On March 20 marked hæmaturia developed, and the urine contained many pus cells and granular casts in addition to blood. The urine became normal again in about ten days.

The patient gradually regained some power in the left leg and to a less extent in the left arm. Slight wasting of the affected muscles took place, but there was no loss of electrical excitability. In the course of the next four months the knee-jerks reappeared on both sides; the knee-jerk and arm-jerks became exaggerated on the left side, on which well-marked patellar and ankle clonus also developed. The plantar reflex remained extensor on the left side. The temperature gradually fell to normal in the course of three weeks. The systolic murmur became less distinct and the heart less dilated. The paralysis of the left external rectus did not disappear until October.

At the present time the patient has recovered to a very considerable extent. There is now scarcely any evidence of the optic neuritis. The left arm is very weak, but the left leg is comparatively slightly affected. The jerks on the left side are still exaggerated, and the plantar reflex extensor. The urine is now normal, and the heart is normal except for an occasional faint systolic apical murmur.

The subsequent history showed that the original diagnosis of embolism was incorrect; acute polio-encephalo-myelitis seems a more probable diagnosis. The same toxins which affected the nervous system probably caused the myocarditis and nephritis.

DISCUSSION.

Dr. HERTZ said his colleague and he were anxious to hear suggestions as to what could be the cause of the widespread localization of the disease. He thought it was possibly a case of infection with the unknown organism which produced poliomyelitis and polio-encephalomyelitis. Dr. F. E. Batten had suggested that it might be a case of infective endocarditis, that the hemiplegia and sixth nerve paralysis were embolic, and that the nephritis was the result of the infective endocarditis. Against that diagnosis was the sudden onset, the loss of consciousness, and the rapid recovery from the acute symptoms. Lastly, it was possible that the condition was due to some other infection of the brain, spinal cord, myocardium and kidneys.

Dr. PARKES WEBER did not see why it might not almost equally well have been a case of cerebral embolism from non-malignant (rheumatic?) endocarditis, in spite of the absence of any history of preceding rheumatism. In a patient with rheumatic mitral valve disease, under treatment in the hospital, he (Dr. Weber) had seen the occurrence of temporary hemiplegia from cerebral embolism cause the immediate appearance of Babinski's sign on the affected side. But he could not understand why there should have been any temporary hemiatrophy of the tongue (nor would one expect optic neuritis in non-malignant endocarditis). That was an objection against the cerebral embolism suggestion.

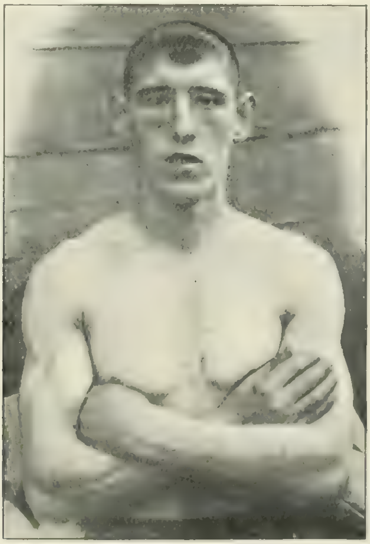
The PRESIDENT remarked that in embolism in children with simple hemiplegia one did not often find there was loss of consciousness.

Case of Bilateral Atrophy of the Face.

By ARTHUR F. HERTZ, M.D., and W. JOHNSON, M.D.

T. T., AGED 26, is the only member of his family affected in this way. He had double otorrhœa from infancy up to the age of 14; there has been no recurrence of this, but he is rather deaf. Six years ago he had a cut on the right side of his face which did not suppurate, and healed rapidly. Two years ago he noticed some puffiness under the eyes, which gradually passed away, but his face, especially the right side, from this time became progressively thinner. This became so marked that his friends thought that he must be consumptive, and he therefore came to hospital. He was first seen by us six months ago; the condition has steadily increased, especially during the last three months. The right side has throughout been the most affected.

He feels in perfect health and, except for his face, he is physically very well developed. His chest is normal and there is no evidence of nervous disease. There is no weakness of the facial muscles, all of which react normally to electricity. His tongue is unaffected; cutaneous sensibility and taste are normal.



Bilateral facial atrophy.

We suggest that this case is of the same nature as the more familiar hemiatrophy of the face, and corresponds with the cases recorded by Wolff and by Oppenheim as examples of "bilateral hemiatrophy of the face."

Dr. PARKES WEBER said he had found the record of one English case which nearly corresponded with the present one—namely, that shown by Dr. Batty Shaw at the Clinical Society of London in 1905.[1] The patient was a boy, aged 10, who commenced to show bilateral wasting of the subcutaneous tissues of the face when he was aged $2\frac{1}{2}$. The boy was brought to the hospital because the mother feared he might have consumption. Bilateral wasting of the subcutaneous tissues about the nose had been noticed after ozæna in a case reported in 1907 by Okouneff in a Russian monthly journal of throat and ear diseases. In regard to cases of hemiatrophy of the face or of the face and body, there were two classes which were associated with morphœa or other kind of sclerodermia. In one class[1] the facial hemiatrophy was actually produced by sclerodermia and was part of the sclerodermatous process. In the other class the hemiatrophy of the face, or of the face and body, was merely accompanied by patches of sclerodermia.[2] There was also (thirdly) the classical kind of hemiatrophy of the face, with ultimate wasting of the bones as well, and sometimes of one half of the tongue. Then there were (fourthly) cases of hemiatrophy of the face and body without sclerodermia. There were also bilateral cases of wasting of the subcutaneous tissues of the face, which were very rare. Such were the case of Hertz and Johnson and the case of Batty Shaw already alluded to. He asked whether the bilateral cases referred to by Oppenheim involved only the soft parts or the bones also. There was still another (sixth) class in which almost all the subcutaneous tissue of the face and body down to the pelvis was bilaterally affected, with almost complete loss of fat. The face and body were emaciated, but the lower extremities remained well covered with fat. A typical case of the kind had been shown in England by Dr. Harry Campbell,[3] and a typical case had been described and illustrated in Berlin by Dr. Eugen Holländer.[4]

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[1] See *Trans. Clin. Soc. Lond.*, 1905, xxxviii, p. 222.

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[1] See J. H. Sequeira, "Two Cases of Fronto-nasal Morphœa," *Brit. Journ. Derm.*, 1911, xxiii, p. 40; also J. Galloway, "Morphœa affecting Right Frontal Region and Orbit," *Proc. Roy. Soc. Med.*, 1911, iv (Clin. Sect.), p. 27.

[2] See Wilfred Harris, *Trans. Med. Soc. Lond.*, 1911, xxxiv, p. 440; also, perhaps, P. C. Knapp, *Proc. Roy. Soc. Med.* 1911, iv (Neurol. Sect.), p. 28. For cases of "Progressive Facial Hemiatrophy with Crossed Pigmentation and Diffuse Sclerodermia," see F. Volkard, *Münch. med. Wochenschr.*, 1903, l, p. 1108. On the association of sclerodermia with facial hemiatrophy, see A. Afzalius, *Arch. f. Derm. u. Syph.*, Wien u. Leipz., 1911, cvi, p. 3. The literature bearing on typical and atypical cases of this class is very large.

[3] Harry Campbell, *Trans. Clin. Soc. Lond.*, 1907, xl, p. 272.

[4] E. Hollander, *Münch. med. Wochenschr.*, 1910, lviii, p. 1794. In the discussion on a case shown by Laignel Lavastine and Viara at the Société de Neurologie of Paris, on July 11, 1912, Pierre Marie thought that it was not rare to meet with females the upper half of whose bodies is emaciated in proportion to the lower limbs. A striking example of the kind referred to by Pierre Marie has been seen by F. P. Weber in the case of a young woman, aged 27, who had lost almost all the subcutaneous fat from her body, but apparently none from the orbits or the mammæ, and less from the lower extremities than from the parts above the pelvis.

Clinical Section.

February 14, 1913.

Sir Wm Osler, Bt., F.R.S., President of the Section, in the Chair.

DISCUSSION ON CERVICAL RIBS.

The Anatomy of Cervical Ribs.

By Frederic Wood Jones, M.B.

Introduction.

That the rib series of man is subject to a certain amount of numerical variation is, of course, an old observation in anatomy; that abnormal ribs may develop, or normal ribs fail to develop, at either end of the thoracic region was well known to the old masters of anatomy.

Many authors mention specific instances, and it is interesting to note that increase rather than reduction in the number of ribs was considered to be the most frequent anomaly. Helkiah Crooke, in his "Mikrocosmographia" of 1651, says: "They are commonly both in men and in women on each side twelve, oftener more than fewer. For Nature would rather there should be an abundance than want. And in a publick anatomy when a malefactor was cut up, Bauhine found thirteen on each side; the first on the left side was perfect, but the first on the right side was imperfect. Fallopius also twice found one too many, Columbus once eleven at Padua" (p. 741). It is not difficult to see why Crooke laid stress upon the fact that the number of ribs is the same in both men and women, for such a finding was not readily acceptable to all men of his time.

Such an anomaly of development was naturally likely to attract attention as a mere anatomical curiosity, and as such it has accumulated its literature—which may be termed the literature of the first period.

But very early some more advanced workers saw in this variation a certain similarity to the condition found in some lower animals. Edward Tyson, in his "Anatomy of a Pygmie" of 1699, says: "It (the anthropoid) had thirteen ribs of a side, six and twenty in all. In man there is but twenty-four, though sometimes there has been observed thirteen to a side."

The time was not near advanced enough for such ideas to be carried further, but such an observation may almost be said to usher in the second period of the literature of rib anomalies. This period properly begins when Darwin's views came to be appreciated by human anatomists, for then these numerical variations were eagerly seized upon as a subject to which study was directed in order to discover some order in the irregularity of development which might throw light upon the processes of evolution. From this inquiry a great mass of literature has arisen. Rosenberg (1876) believed that in these variations a progressive development was manifested, and that the general shortening of the thoracic region, which is at work throughout the mammalian series, was evidenced as a distinct and ordered evolutionary process from ape to man. Confirmation of his views was freely provided by other biologists who extended these studies, but Holl, of Innsbruck (1882), disputed the theory, and Paterson (1893), and Bardeen (1905) brought forward many facts which definitely contradicted it. Dwight, of Harvard, after a prolonged study of abnormal human vertebral columns, saw no progressive change in any direction, and recognized no evolutionary tendency in the process, but decided that all these anomalies "are merely variations round a mean, which for want of a better word we must call accidental" (1911).

The idea underlying most present-day theories appears to be that these variations are compensatory efforts to restore to normal proportions a vertebral column in which some initial error of segmentation has occurred. Around such discussions the bulk of the anatomical literature of cervical ribs has been built up. Cervical ribs, in fact, like so many other parts of the human body, passed through a period during which their sole interest to the anatomist was purely morphological. This period was fertile in the production of literature, yet furnished but little material towards the application of the study of human anatomy to the practical needs of medicine and surgery.

These abnormal structures were, however, rescued from this condition of mere morphological signposts by the clinician: for cases were met with in practice which showed a definite train of symptoms associated with the presence of an abnormal rib in the cervical region. Anatomists again turned to the study of rib anomalies, and such studies may be considered as constituting a third period in the literature of cervical ribs.

The anatomical literature of this period remains scattered as isolated papers in various publications; so far it has not influenced text-book teaching, and it is not available to the clinician in any consecutive form. It is the purpose of this paper to review the present state of anatomical knowledge regarding the causation of abnormal rib development, and to collect such material as has accumulated which throws light on the anatomical condition and the clinical manifestations of the anomaly.

COMPARATIVE ANATOMY AND DEVELOPMENT OF RIB ANOMALIES.

Of the many segments which enter into the composition of the human vertebral column only a certain number are destined to bear ribs. Normally twelve such segments in the thoracic region carry ribs, but abnormally in man as many as fourteen or as few as ten vertebræ may be rib-bearing or thoracic vertebræ. These rib-bearing vertebræ are preceded by and followed by ribless regions of the vertebral column; but it must always be remembered that, even in the ribless regions, the vertebræ possesses very definite rudiments of the ribs which develop fully only in the thoracic region.

Now such a condition is not common to all vertebrate types, for amongst the snakes every member of the vertebral series may carry a well-developed rib. Such animals have no cervical or lumbar region or—popularly—possess neither neck nor waist. All stages in the production of ribless cervical and lumbar regions are seen in the gradual change of structure which takes place in the transition between snakes and lizards, and the clue to these changes is to be found in the study of the developing perfection of the limbs.

It may be stated as a general rule that the functional development of the limbs is accompanied by a reduction of the rib series, and that a ribless neck and a ribless waist are the outcomes of the development of a functional arm and leg. To discover the reason for this association it is only necessary to turn to the developing embryo.

In the early human embryo the epiblastic bases of the spinal nerves run as girdles around the body. They are, in fact, the expression of the primitive segmentation of the body, for segmentation "finds its expression in the arrangements of the primitive segments and the nerves supplying these, and not in the skeleton, which is a later development" (J. Arthur Thomson). But between each spinal nerve band there run alternate mesoblastic bands which constitute the basis of the future ribs; a rib may be regarded as an intersegmental mass of mesoblast, and a spinal nerve as the representative of the primitive segmentation. Such a simple condition of segment and intersegment is seen in the human embryo, and as such it persists in the adult snake. The ribs, however, when they are laid down are placed upon a more superficial plane than the nerves, so that the nerves become partially sheltered by the encircling ribs, and they emerge from this shelter in order to reach the muscles and the skin which they supply. This simple girdle arrangement may persist so long as each individual nerve possesses only the function of supplying a segment which is itself a girdle of the animal body. But in the embryo of a limbed animal it is readily seen that when the limb buds appear they are formed as the derivatives of several body segments, and at first occupy an area coterminous with these body segments. Into these limb buds the nerves from several body segments are consequently continued. At an early stage, when the limb bud is coterminous with

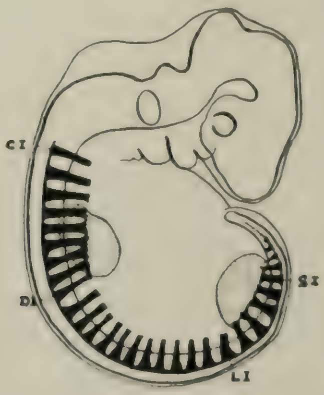

FIG. 1.

Diagram, after a reconstruction of an embryo of 6·9 mm., by Streeter, to show the nerve-roots running almost parallel to the limb buds.

the body segments from which it arises, the parallel arrangement of the nerves, even to their distal terminations in the limb buds, will be maintained. But it is very soon manifest that this orderly arrangement becomes upset. The limb bud ceases to keep pace with the body segments as the latter become of increasing breadth. The bud grows out at right angles to the body axis and so becomes an attenuated derivative of several body segments, which body segments have long outstripped the area of the original base from which the limb budded. This change necessitates an alteration in the arrangements of the segmental nerves which run to the limb bud; for now, instead of running parallel to each other from the body to the limb, they become gathered together in a leash in the root of the limb and are widely separated at their origins from the spinal cord. In other words, the segments and intersegments have grown apart in the body but are still closely approximated in the limb, and nerves, representative of several widely separated segments in the body, are therefore focused upon one point in the limb. This constitutes the formation of a limb plexus.

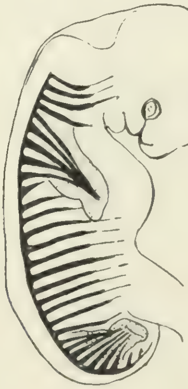

FIG. 2.

Diagram of a somewhat older embryo to show the nerve-roots entering limb buds in an oblique manner: formation of a plexus.

It is the formation of the brachial plexus which produces the ribless neck and the formation of the lumbo-sacral plexus which produces the ribless waist. The manner in which the ribless areas are brought about may be followed readily in the developing embryo. When first the body segments which are represented in the limbs begin to grow apart, and so outstrip their prolongations into the limbs, they are composed of epiblastic nerve and mesoblastic rib basis. The epiblastic nerve passes from within outwards through each mesoblastic segment and intersegment, for lying at first beneath the shelter of the rib basis it has to emerge into the outgrowing limb. If the primitive condition were retained each limb base would be as extensive as that area of the body from which its segments are derived, and each limb nerve would emerge between two ribs to run into this enormously broadened limb base. Such a conception makes the actual process more easy to realize, for it is not difficult to picture a relative shrinkage of the limb bud, a gathering together of the limb nerves in the narrowed limb, and a consequent interruption by epiblastic nerves of all the intervening mesoblastic intersegmental bands. This is what actually happens in the developing embryo, for the rib bases begin to develop in the ribless areas and continue to grow until the concentration of the emerging nerves on the limb root cuts short their distal extension. The ribs may therefore be said to be shorn from the neck region (and from the loins) by the straining of the nerves across the mesoblastic basis in which they would be laid down. So far as the bony element is at liberty to develop between the emerging nerves it does so, and rib rudiments are of course present as the anterior tubercles of the transverse processes in the normal cervical vertebræ.

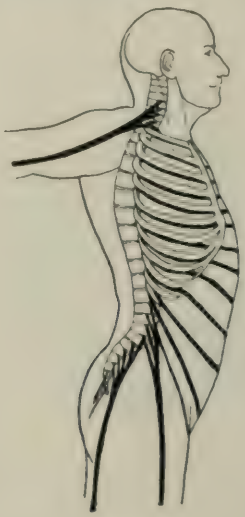

FIG. 3.

Diagram to show the directions of the spinal nerves and their gathering into plexuses in an adult.

Note.—There is one class of animals in which limbs and limb plexuses are developed without the sacrifice of any ribs; for the bony fish, though possessing functional pelvic and pectoral fins, have no waist and no neck. I take it to be a support to the present argument that in bony fish the ribs are not upon the same morphological plane as are those of the other vertebrate classes. In bony fish the ribs, instead of springing from the neural arches—and so being superficial to the nerves—are developed from the vertebral centre—and are deep to the nerves. With such an arrangement a plexus of limb nerves may be produced without transgression of the underlying ribs.

The whole of this question has been more fully discussed in a previous paper, "On the Relation of the Limb Plexuses to the Ribs and Vertebral Column" (*Journal of Anatomy and Physiology*, July, 1910, xliv, p. 377).

THE ANATOMICAL RELATION OF THE COSTAL ELEMENT AND THE PLEXUS.

It would therefore seem to be apparent that there is an antagonism between the formation of a nerve plexus for the supply of the limb and the development of ribs in that region from which the nerves are derived. This antagonism is manifested in the bony elements by the many pressure marks stamped by the nerves upon the developing bone. The reality of the influence of nerve-pressure has not been by any means sufficiently appreciated, because the nerves of the adult appear to be such trivial things to have any determining influence upon the disposition of bone; but it is in the early stages of the embryo, when the nerves are of preponderant size, and the mesoblastic osseous basis is yielding and trivial, that the process is in its active stages.

One remarkable instance of nerve-pressure that has for long been overlooked is to be found in the so-called *sulcus subclaviæ* which marks the upper surface of the normal first rib. This groove is formed by, and lodges the lowest cord of the normal brachial plexus, and the reason for its being mistaken for an arterial impression is, I imagine, that when the arm is extended at right angles to the body (as it is during the progress of an ordinary dissection) the nerve-cord is somewhat raised from the surface of the bone, but the subclavian artery is not. It was during the dissection of a body in which the arms were pressed close to the sides that I first noted the relation of the nerve-cord to the groove; and abundant confirmation of the fact has since been forthcoming from several sources (for first note on nerve-cord pressure on the normal first rib, and formation of the groove, see *Anat. Anzeig.*, 1910, xxxvi, i, p. 25).

The recognition of this groove as a sign of nerve-pressure is of fundamental importance in any inquiry as to the conditions of cervical ribs or any discussion of the symptoms which they produce. Although this groove was so long mistaken as an arterial impression, a similar but deeper groove which marks the upper surface of a developed seventh cervical rib has been recognized as a nerve groove by many observers (among them Turner, Lane, Dwight, and Phillips). This groove upon a cervical rib is of great interest, for it tells much of the story of the antagonism between the developing limb nerve and the developing rib. The groove starts upon the inner margin of the rudimentary rib, and runs across its upper surface very obliquely from behind forwards and outwards: the anterior and outer limit of the groove coincides, as a rule, with the distal termination of the rudimentary rib. Beyond the tip of the bony cervical rib upon which

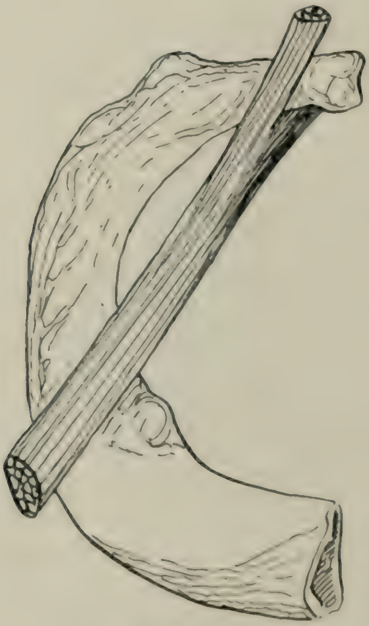

Fig. 4.

The normal first rib and its relation to the lowest cord of the brachial plexus.

the nerve groove ends a ligamentous structure generally connects the rudimentary rib either to the first rib or to the sternum.

If, now, the normal costal element of the seventh cervical vertebra be examined, it will be seen that it is abbreviated in exactly the same manner by the passage of the nerve-cord. Moreover, the elements above this show increasing evidence of the downward straining of the nerves that join the brachial plexus, and a proportionately diminishing costal element. These nerve markings on the cervical vertebræ have been discussed in a separate paper (see *Journal of Anatomy and Physiology*, 1911, xlvi, p. 41).

The markings on the lumbar vertebræ show equally well the influence of the nerve elements (see *Journal of Anatomy and Physiology*, 1912, xlvii, p. 118), but do not concern the present inquiry; nevertheless, they add weight to the supposition that the distinctive features of the elements of the vertebral column are impressed upon them early in development by the influence of the nerves and their manner of distribution as plexuses for the supply of the limbs. Such a supposition leads further than the current explanations of vertebral anomalies already mentioned, and it is capable of many helpful extensions, for it presumes that anomalies in the arrangement of the limb plexuses are primary, and not secondary to anomalies in the disposition of the ribs and vertebral elements.

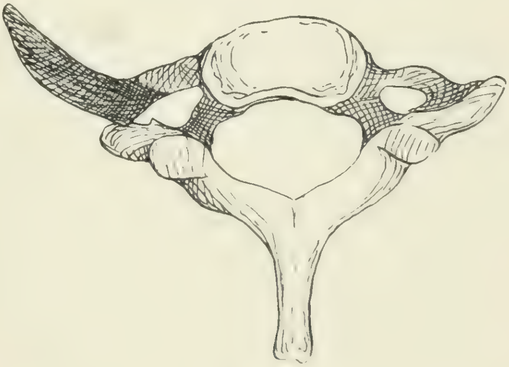

FIG. 5.

A left cervical rib showing nerve groove impressed on its upper surface by the brachial plexus.

For direct support for such a supposition it is only necessary to turn to the body and examine the condition of the first rib in relation to the constitution of the brachial plexus. It is well known that the brachial plexus is subject to many variations about a mean which is taken as normal. The plexus may be shifted somewhat nearer to the head, or somewhat nearer to the hind end of the body. Such a shifting may not manifest itself by the absorption of nerves that are normally outside the range of the plexus, but may limit itself to a greater or less contribution from the nerve-trunks which mark its cephalic and caudal extremities. In a typical plexus a portion of the first thoracic nerve ascends within the thorax to pass out over the first rib with the brachial plexus. The amount of contribution made by the first thoracic nerve to the brachial plexus is subject to wide variations; it may furnish a slender twig, a large cord, or practically the whole of its bulk to the plexus in cases that we are accustomed to regard as within the limits of the normal.

It is also well known that the *sulcus nervi brachialis* (so-called *sulcus subclaviæ*) upon the upper surface of the first rib is also subject to much variation, and I have determined, by an examination of a series of bodies in the post-mortem room, that the depth of this groove increases as the contribution from the first thoracic nerve increases. In cases which hardly come within the limits of the abnormal this interaction of the strained caudal end of the plexus and the first rib may proceed to such a degree that the rib becomes bent downwards at the site of crossing of the lowest cord. Such a bending is often evidenced in cases of cervical rib, and has been noted by Dwight. With a large caudal contribution to the brachial plexus, therefore, the tension between the lowest cord and the first rib increases (for further details *see* "Variations of the First Rib associated with Changes in the Constitution of the Brachial Plexus" (*Journal of Anatomy and Physiology*, 1911, xlv, p. 249). Now, for the purpose of this paper it becomes all important to determine if such a pressure between a normal first rib and the lower cord of the brachial plexus can produce such symptoms as are usually ascribed to the presence of a cervical rib. It is certainly a fact that this may happen.

In the *Australian Medical Journal* of October, 1910,[1] Dr. Thomas Murphy described a case, of which an abstract was published in the *Lancet* of December 17, 1910. Briefly, in this case, a woman, aged 28, had for eight years shown varying, but usually severe, symptoms of brachial neuritis; and the condition present pointed to pressure on the nerve-trunks. The diagnosis of cervical rib seemed obvious, but a

[1] *Austral. Med. Journ.*, Melbourne, 1910, xv, p. 582.

skiagram showed that no cervical rib was present. Since pressure upon the plexus at the root of the neck caused an increase in the pain, an operation in this site was wisely determined upon. The portion of the normal first rib upon which the lowest cord lay was excised, and the plexus was allowed to sink to a lower level. Five hours after the operation the relief of the symptoms was noticed, and by the end of a week all pain was gone, nor was there any return of symptoms even after severe exercise.

The case is not an isolated one, for other instances in which the symptoms of cervical rib have been manifested in the proved absence of a cervical rib have been met with. It is, of course, a mere matter of conjecture as to why in these cases a normal first rib should produce symptoms; but there is much support for the supposition that even though the rib was normal the brachial plexus was probably of the post-fixed type.

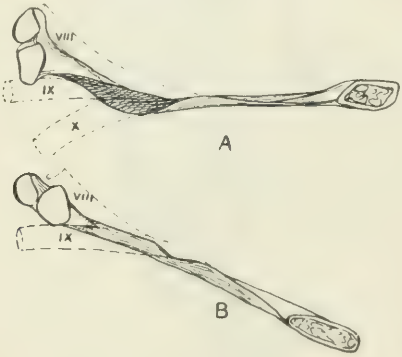

Fig. 6.

A, first thoracic rib, seen from within, to show the bending of the axis of the rib at the site of the *sulcus nervi brachialis*, contrasted with the corresponding margin of the typical form, **B**.

When a brachial plexus becomes more profoundly altered from the normal so as to include caudal nerve-roots, which are normally outside the limits of the plexus, the interference with the development of the rib may naturally be expected to reach its maximum.

At times the second thoracic nerve becomes added in whole or in part to the plexus; and then profound anatomical changes may be produced, for the antagonism between nerve and rib element may lead to a curtailment of the first rib, which then presents characters surprisingly like those seen in a case of developed cervical rib. Such a case I have met with in a bilateral condition in a female child, aged 7. The condition present was as follows: The first ribs were only partially developed, their posterior bony part was short, and terminated at the point where the lower cord of the brachial plexus passed outwards to the arm. Beyond this point they were continued as ligamentous bands, into which the fibres of the anterior scalene muscle were inserted, and which were attached to rudimentary costal cartilages which were imperfectly separated from those of the second rib. The eighth ribs reached the sides of the sternum in the manner of the normal seventh ribs. The twelfth ribs were long and unusually well developed, but no rib was attached to the first lumbar vertebra. Upon both sides the arteria subclavia passed over the rudimentary first rib with the brachial plexus in the normal manner. Dukes and Owen, Keith and Hertslet, and Lane have recorded similar cases of rudimentary first ribs in which the second thoracic nerve made considerable contributions to the brachial plexus. In the case described the whole of the second thoracic nerve did not join the plexus, but it gave a very considerable contribution to it. These cases are to be regarded as instances of a caudal migration of the limb plexus, accompanied by a partial reduction of the normal first rib,

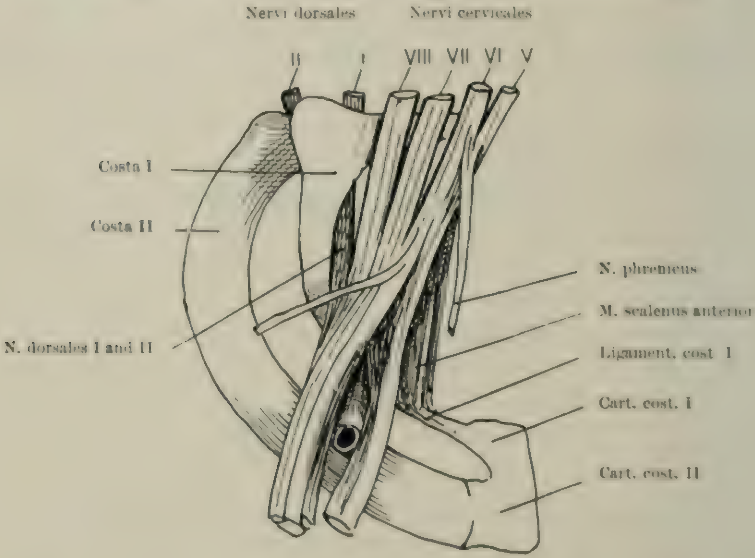

FIG. 7.

Case of rudimentary first thoracic rib. Right side seen from above. The muscles have been dissected away.

and evidently take their origin early in embryonic life by the developing limb bud arising from one segment nearer the hind end than is normal.

These rudimentary first ribs have been repeatedly met with and described as dissecting room curiosities (in addition to those mentioned; *see* cases by Bradley, Gruber, Halbertsma, Helm, Honauld, Hunter, Knox, Leboucq, Le Double, Low, Macphail, Muller, Struthers, Turner, Zaaijer, and others), but it seems almost certain that some forms of them would be capable of producing symptoms such as are produced by cervical ribs. Indeed, I believe that these cases would by any ordinary methods of examination be taken for, and recorded as, instances of cervical rib. Clinically these aborted first ribs have received no recognition; yet there are many of them recorded in anatomical literature, and it is extremely probable that under the general heading of "cervical ribs" many such cases are included in clinical records. Nothing short of a count of the vertebræ lying cephalad of the disputed rudimentary rib can be considered as an adequate criterion for properly diagnosing these cases. Such cases show the interaction that may be produced between the costal element of the first thoracic segment and the lowest cord of the brachial plexus.

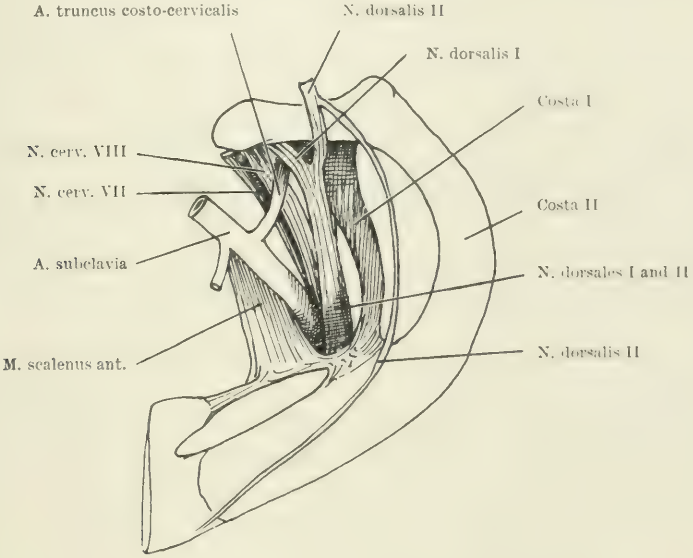

FIG. 8.

Case of rudimentary first thoracic rib. Right side seen from below.

When the costal element of the seventh cervical segment is developed the lowest cord of the plexus passes over it, and the resulting pressure is naturally increased. We may infer that there is in these cases a tendency for the plexus to be prefixed, and we have Eisler's assertion that when a cervical rib is well developed the plexus either receives no contribution, or only a very small one, from the first thoracic nerve; also some cases that have been met with in the dissecting room demonstrate this condition (cases recorded by Black, Hertslet and Keith, &c.). All people who have cervical ribs do not exhibit symptoms of pressure upon the nerve-cords, and it is well known that some of the cases which present no symptoms possess particularly well developed cervical ribs; indeed, it has been laid down as a rule by Lewis Jones that, "where a bony prominence can be felt with ease, the brachial plexus is usually free from pressure" (*see* Theodore Thompson, *Brain*, 1908, xxvi, part cxxii, p. 286). In these cases it is to be presumed that the prefixation is considerable (involving probably a whole nerve-root), and the plexus and the bony elements have readjusted themselves at a more cephalic level than is normal. On the other hand, other cases make it quite clear that although there is a tendency to prefixation of the plexus, such prefixation is not sufficient in degree to counteract the strain produced by the presence of a costal element developed at an abnormally high level. Just as varying grades of imperfection of development of the first thoracic rib are the outcomes of varying degrees of post-fixation of the plexus, so the varying grades of perfection in the development of a cervical rib are the outcomes of varying degrees of prefixation of the plexus. And just as the post-fixation may readjust itself with the rib elements at a lower level, so may the prefixation readjust itself at a higher level. It is in the intermediate grades, in which the development of the costal process is in excess of the plexus alteration, that the strain is produced and symptoms are developed.

THE ANATOMICAL RELATION OF THE COSTAL ELEMENT AND THE ARTERY.

Some years ago the surgical teaching concerning cervical ribs was practically limited to the doctrine that the rudimentary rib, by raising the subclavian artery to a higher level in the neck, and so making its pulsations more apparent, might lead to the incorrect diagnosis of subclavian aneurysm. This straining upwards of the artery has also been held responsible for the production of the vascular disturbances frequently displayed in the forearm and hands in these cases.

Mr. T. Wingate Todd (*Journal of Anatomy and Physiology*, 1911, xlv, p. 304) declared that the "arterial symptoms are caused by the action, directly by the scalenes and indirectly by the diaphragm, on an artery with a further and more tortuous course to pursue than is normal." Others have spoken of the artery being compressed beneath the rudimentary rib. In all cases of rudimentary first thoracic rib it is certain that the artery passes over the rib or its fibrous continuation. It seems also definite that in some cases of undoubted cervical rib the artery is also elevated above the rib; nevertheless, the surgeon meets with cases in which the artery lies below a rudimentary rib that is certainly not derived from the sixth cervical vertebra. More information is needed on this point, and when a clear distinction is made in diagnosis between rudimentary first thoracic and rudimentary seventh cervical ribs, this information will rapidly accumulate; but in the present state of knowledge some uncertainty must exist as to the normal relation of the artery to seventh cervical ribs.

Whatever the relation of the subclavian artery to the abnormal rib, it seems certain that the vascular symptoms in the arm are not produced in all cases by compression of the artery, either by it being stretched over the rib or pressed beneath it. Indeed, in a subsequent paper published by Mr. Todd it is asserted that "any explanation of the vascular phenomena may be found inadequate if it depends only on direct mechanical pressure of the subclavian artery" (*Lancet*, August 10, 1912). In a still more recent paper (*Journal of Anatomy and Physiology*, January, 1913, xlvii, part ii, p. 250), Mr. Todd appears to have reversed his former theory entirely, for he says: "First the artery is cirsoid and cannot be stretched; secondly, the contracting scalenes cannot compress it."

Just as the pressure on the lowest cord of the brachial plexus produces the muscular and sensory changes, so it may in some cases also produce the vascular symptoms. It is the compression of the vasomotor fibres in the lower cord of the plexus which causes these symptoms, and this fact Mr. Todd has clearly demonstrated. The sympathetic contribution to the plexus is mostly supplied to the first thoracic nerve by a communication with the second nerve within the thorax. Such an interchange of fibres has been noticed by many anatomists, and Mr. Todd has recently demonstrated its sympathetic nature and its clinical importance in producing vascular symptoms. Spinal fibres and sympathetic fibres are, in fact, both subject to the pressure effects caused by the antagonism between rib development and plexus formation.

The last phase in the study of this subject has been the examination of the histological condition of the vessels involved, and here Mr. Todd has shown that, at any rate in one case, they were trophic in nature and consisted of a definite change in the nature of the vessel walls. Bechterew, Cehanović, and Lapinski, have studied the histological changes of the vessel walls produced by interference with the sympathetic nerve supply, and the appearances that they describe are similar to those displayed in this case of vascular disturbance caused by the presence of a cervical rib. Quoting from Todd's most recent paper, the changes are displayed in both artery and vein as follows: "*Artery*—(*a*) Adventitia, no marked change. (*b*) Media, increased in amount; cells apparently healthy. (*c*) Intima, reduplication in places of elastic lamina: normal appearance lost in places owing to proliferation of the lining endothelium. (*d*) Lumen partially obliterated owing to development of connective tissue, which blocks the channel, and which, judging from the pigmentation present, has resulted from the organization of blood-clot. *Vein*—Hypertrophy of muscular coat as in artery."

We may therefore sum up the anatomical condition by saying that pressure on the lowest cord of the plexus may produce all the symptoms, motor, sensory, trophic, and vascular, displayed in cases of cervical rib; or in cases in which there is any disharmony in rib and plexus development.

ANATOMICAL RELATION OF THE SHOULDER-GIRDLE TO THE RIBS.

Symptoms of brachial neuritis from pressure of a costal element, and its accompanying manifestations, appear in adolescents, in people who show loss of muscular tone, in patients who display in one form or another the symptoms of visceroptosis, and in women recently delivered. In all its manifestations brachial neuritis from rib pressure is more common in women than in men. Children with cervical ribs show no symptoms, and the reason for the late onset of any neuritis has given rise to considerable discussion. It has become stereotyped in clinical literature to regard the late development of symptoms as due to the late ossification of the rib rudiment. There was never any anatomical justification for this view; there is no evidence that a cervical rib does ossify late; and when a normal first rib is the offender this theory must naturally fail. Again, such a theory affords no sort of explanation of the appearance of symptoms in cases in which the outstanding feature is a general loss of muscular tone.

It is, however, the latter class of cases which gives the real clue to the causation of symptoms, for these people display in an exaggerated form that dropping back of the shoulder-girdle and fore-limb which is a normal change in adolescents. It is a matter of lay observation that the shoulders drop back as growth proceeds; it is also a matter of common knowledge that in women they usually drop back farther than in men. In a child the clavicles rise markedly from their sternal ends towards their outer extremities; in adult men they more nearly approach the horizontal; and in adult women they not uncommonly slope downwards from their sternal ends. Such an observation is as old as anatomical literature. In cases in which muscular laxity is well marked this downward sloping of the outer end becomes more pronounced. It is true that in all these cases there is an accompanying fall of the whole thoracic cage, and a consequent lowering of the sternal end of the first rib and also of the sternal end of the clavicle; but the fall of the shoulder outweighs this, so that despite the fall of the sternal end the acromial end drops relatively to it.

The normal age changes in the position of the shoulder-girdle have been measured and tabulated by Todd (*Anat. Anzeig.*, 1912, xli, p. 385), and his figures confirm these general statements. It is, of course, obvious that the greater the drop of the arm relative to the highest rib, the greater will be the tension between the lowest cord of the plexus and that rib. It is also obvious that when a drop of the shoulder-girdle has produced sufficient tension to cause the development of symptoms, such symptoms would probably be relieved by a deliberate elevation of the shoulder-girdle. As a matter of fact, most sufferers from brachial neuritis which is due to rib pressure have evolved this much for themselves.

It would seem to be not unlikely that in normal people the anatomical relation of the normal lowest cord and the normal first rib may produce some slight pressure symptoms; but this only to the extent that an "arm-" chair is welcome after a day's work. Indeed, the whole secret of the "arm-" chair might well be that by the support it affords to the elbows and the consequent raising of the shoulder-girdle that it produces, it relieves the plexus from pressure which has been acting during the hours in which the arm was dependent. A patient with cervical ribs gains relief by sitting in an armchair, and it has been noticed repeatedly that they are most comfortable when they sit so that the shoulders are hunched up almost to the ears. When such patients sleep they select a position which will raise the shoulder of the affected arm as far as possible from the top of the chest wall, and I have been very much struck, in the limited experience which as an anatomist I have of the clinical aspect of these cases, with the diagnostic value of this position of rest. When lying on the side it is easier to insure that the underlying shoulder shall remain elevated, and patients often lie in bed with this shoulder so far raised that the head lies rather on the shoulder than on the pillow. One woman rested in so strange a pose that it was her habit to say she slept "like a bird—with her head tucked under her wing." So far is this carried in some cases that Mr. Percy Sargent has told me of a woman who was in the habit of slinging one axilla to the head of the bed to prevent the falling of the shoulder-girdle during sleep. Such an extreme method would, of course, at once attract attention; but I think that suspicion should be aroused by the fact that in brachial neuritis caused by rib pressure, the patient lies on the painful side, whereas in most other painful conditions of the arm they lie upon the sound side.

THE ANATOMICAL INTERPRETATION OF SKIAGRAM OF COSTAL ANOMALIES.

The introduction of radiography has assisted in a marked manner in solving the problems connected with abnormal rib development; but its utility has not yet been generally employed to its utmost. To the expert radiographer there is no difficulty in the interpretation of the shadows cast by rudimentary ribs, and the diagnosis of a rudimentary rib on the negative is sufficient for clinical purposes. But we know, anatomically, that by no means all of these rudimentary ribs are seventh cervical ribs, though clinically they are all commonly classed as such. What remains for the radiographer to do in this inquiry is to determine, beyond any dispute, which member of the vertebral series gives origin to the rudimentary rib. Probably many interesting facts will come to light when this is done as an ordinary detail of routine examination.

Unfortunately, radiographers have relied on a very false criterion for determining vertebral levels in this region. It has been laid down as an axiom that the slope of the transverse process is diagnostic, for it is said that "those of the seventh cervical are short and usually set at right angles with the vertebral column, or have a downward tendency, while the first dorsal processes are larger and have an upward tendency."

(S. Gilbert Scott, "The Diagnosis of Cervical Ribs from a Radiograph," *London Hospital Gazette*, 1911, xviii., p. 92.) No reliance is to be placed upon this slope of the transverse processes even in the normal skeleton, and in cases in which abnormal rib development is present it is a most misleading feature. Many of the published skiagraphs which illustrate classical cases of cervical ribs are, judged by this criterion, cases of rudimentary first dorsal ribs. Such, in fact, they may be, and I believe it is extremely likely that with proper radiographic examination many cases of first rib pressure will be detected; but the slope of the transverse processes cannot determine which rib is rudimentary. (Further details are given in a paper on the "Radiographic Diagnosis of Rudimentary Ribs," *London Hospital Gazette*, 1912, xviii, p. 166.)

It is only by counting the number of vertebræ which lie cephalic to the disputed member that the real nature of a costal element may be determined with certainty. Such a method is now employed in some hospitals, and its universal adoption would add to the present sum of knowledge of rudimentary ribs.

Surgical Treatment.

By William Thorburn, F.R.C.S.

Mr. Thorburn said he did not propose to detain the meeting by going into the symptoms of cervical rib, because sometime ago he did that elsewhere,[1] and had nothing to add. He would say a few words about the surgical treatment of the condition, considering the indications for operation, the method of operation, and the results.

With regard to the indications for operation, he suggested that there were three great possible groups of cases in which the surgeon might be called upon to operate: (1) Those in which the rib might be removed on account of deformity; (2) those in which it might be done for the relief of vascular symptoms; (3) those in which the operation might be done for nerve symptoms. The first group could be set on one side, as it was waste of time to discuss it. With regard to those causing vascular symptoms, he had not been called upon to operate for these alone. He had only met with cases in which there were combined vascular and nervous symptoms. Still, he clearly recognized that one might be called upon to operate where there was marked congestion or anæmia, or threatened gangrene of the part. Before speaking of the third group he wished, however, to say a word on a point of interest mentioned by Mr. Wood Jones—namely, that he could not quite agree with the anatomists that the vascular symptoms in this condition were solely due to pressure on sympathetic fibres, because, beyond all question in cases in which the radial pulse is markedly diminished by cervical rib, one can by raising the arm readily restore the pulse by unlocking a kink of some kind: whether this kink is produced by the artery resting on the rib or by its resting on the tissue between the cervical rib and the first dorsal he did not know. His second reason was, that in nearly every operation which he had performed, if there had been a difference between the two radial arteries, that difference had ceased to be apparent from the moment the patient had recovered from the anæsthetic, which could not be the case if it were due to injury to sympathetic fibres.

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[1] *Med.-Chir. Trans.*, 1905, lxxxviii, pp. 109-125, and "Dreschfeld Memorial Volume" (University of Manchester Publications, No. xxxv), 1908, pp. 85-111.

With regard to the nervous symptoms, he divided them into two great groups, which merged into one another. There was the *neuralgic* group, in which the essential symptom was merely pain. Next there was the *paralytic* group, in which there were symptoms of more or less paralysis, with cramps, atrophies and anæsthesia, which he did not propose to describe in detail. The main question was, as to which of those groups one should operate on, if operation was to be done at all. Pain alone could be relieved by other measures, and he would not think operation necessary for the relief of pain only, at all events as a routine procedure, unless it were severe. But one met with cases in which other symptoms came on with extreme suddenness: the neuralgic group of cases might pass rapidly into the paralytic, and in these operation would become necessary, on account of the severity of the symptoms. He had not yet seen a "paralytic" case which showed any tendency to recover under any treatment short of operation; but he had seen cases of long standing entirely crippled by being allowed to go untreated. The indications for operation were then either (1) pain which was not relieved by other measures, such as postural devices, or (2) the severe symptoms which were associated with the paralytic group.

With regard to the nature of the operation, he would not take up much time. He had tried various incisions which had been recommended, and in the earlier days he had to feel his way. But he was now strongly of opinion that the best and easiest incision—and some of the cases were not easy—was a long incision straight down and well back on the neck, over the anterior border of the trapezius muscle. The "collar" incision he found did not give enough room in a difficult case, and short, stumpy ribs were difficult to find, while the rib might be higher up in the neck than the operator expected to find it. If the rib were low down, no doubt the collar incision gave a better cosmetic result. By using the incision he recommended there was very little bleeding, for one got down by blunt dissection, and he struck the rib as near to the vertebral column as he could get. The only structure encountered which might give trouble if damaged was the suprascapular nerve. By attacking the rib from the back he had found it most convenient to cut it through at the base and work forward, dissecting the rib forwards, and dealing with the anterior end according to what was found. Sometimes there was a bony ankylosis with the first dorsal, sometimes a fibrous ankylosis; one dealt with whatever condition arose. All that had to be remembered by those not in the habit of doing it was, that the brachial plexus was a vulnerable structure, and therefore must not be subjected to anything like roughness, such as one could use with ordinary rib resection and in other operations upon bone. He had adopted drainage in all his later cases, for the reasons he would mention subsequently.

As he had done operations on these cases longer than had most members of the Section, he would detail the results he had had. He had operated for the condition fourteen times, and had collected records of the cases which had been operated upon by his colleagues in the Manchester Infirmary, numbering six. Therefore, he could give the results in twenty cases. They extended back to fourteen years ago, and included no case of less than a year's duration. He would describe the ultimate results in relieving the principal symptoms met with in these cases. *Pain* was present in all the twenty. It was completely cured in twelve, relieved in three, and five were lost sight of; they were done at so early a date that he could not now trace them. *Paralysis* (of sufficient intensity to be subjectively notable, not mere slight weakness) was noted in twelve; it was completely cured in five, greatly relieved in five; two were not traced. There was no case in which no improvement was obtained. *Atrophy* of the hand muscles, which was a prominent symptom in most paralytic cases, he had never been able to satisfy himself was ever entirely cured. There was improvement, but in every case he had been able to test subsequently, even after a long period, there was visible a certain amount of atrophy. Tactile *anæsthesia*, one might take it, could be cured by operation in practically every case, and generally at a comparatively early date. On the other hand, the sense of *coldness* and interference with the temperature sense was apt to remain in the majority. Patients generally complained that the hand was still somewhat cold, and that the paralytic symptoms, if any, were somewhat more marked in cold weather.

Summarized, he would say that all the twenty cases were greatly improved. The case with which he personally was least satisfied was that of a patient who came back to him to have the other side operated upon because of the relief she had obtained. Pain was cured in four-fifths of the cases which could be traced afterwards. Paralysis, which had been sufficient to be inconvenient, was cured in certainly half the cases, though some weakness remained in the others. Anæsthesia was cured in all, though recovery from this was slow, and the sense of coldness never entirely disappeared.

He wished to mention the other side of the picture also, because it was important in regard to a surgical operation, especially one in which he had had to feel his way, that he should tell others the difficulties he had experienced. In two cases he produced a transient paralysis of the brachial plexus. In neither was that at all serious, and it lasted only a few days, and did not affect the after-result. In one case he produced definite paralysis of the suprascapular nerve, which, by the method he had described, was open to much tension; and there was in that case a definite wasting of the supraspinatus and the infraspinatus. It was three months before that cleared up. In two cases he had opened the pleura in the course of the operation, and in one of them the patient suffered for two or three days from pleural pain, but she left the nursing home well in a fortnight. In the other pleuritic case also the condition completely recovered in some ten days.

There remained one other important question, which of course was cleared up considerably by more recent views on the anatomy—namely, the question whether the surgeon always did remove a cervical rib. He did not doubt that in one of his early cases, which he had recorded, and figured, in the "Dreschfeld Memorial Volume," he removed a perfectly normal first dorsal rib. He knew that two of his colleagues had done the same thing. But it did not seem to matter very much as regards the symptoms, as the proceeding seemed to cure the case equally well. One of the most satisfactory cases he had had was the one in which he removed a first dorsal rib. The patient was a young lady engaged in teaching music, and she wrote to him recently that although she had had a particularly busy year at teaching, she now had no symptoms.

The rib removed might be a rudimentary first dorsal; and it was important to remember that the cervical rib might be very high up in the neck, and the incision must not be made too low.

There was another inconvenience which was met with in two of his cases, and one in the practice of a colleague—namely, that a considerable time after the operation (in one case three weeks and in another two months), and after the scar had been well healed, there had developed a small sinus, burrowing deeply into the neck. Such cases gave a good deal of trouble. In one case it did not heal for a year, and in another for eighteen months. He could not give the reason for this, but suggested that it might be due to some peculiarity in connexion with the fixity of the parts and the mobility of other parts in the neck. He asked the meeting to accept his statement that it was not due to sepsis, and he suggested that it was advisable to drain the operation wound for some twenty-four hours, which he had not done in his earlier cases.

He was glad to say that none of the difficulties he had detailed as occurring in his experience had been permanent, neither had they militated against the ultimate success of the operation.

Some Points in the Surgery of Cervical Ribs.

By Percy Sargent, F.R.C.S.

The points concerning cervical ribs to which I wish briefly to draw attention are: (1) anatomical, (2) the mode of causation of the symptoms, (3) the value of X-rays in diagnosis, and (4) a brief summary of my own series of thirty-four operations.

(1) Anatomy.

When dissecting some time ago a brachial plexus obtained from the post-mortem room, I found that I had chanced upon a specimen of the prefixed type of plexus to which the first dorsal root made no contribution, whilst the fourth cervical was abnormally large, and was made up of the three separate bundles, each with a definite distribution, into which the normal fifth root can be resolved, as was the first demonstrated by Mr. Sydney Scott. A large cervical rib was also present (fig. 1), and I naturally wondered whether there could be any connexion between the two anomalies. Dr. Wood Jones, to whom I showed the specimen, kindly referred me to a paper of his own, in which the correlation of variations in the brachial plexus with well-recognized anomalies of the ribs is fully discussed. Dr. Wood Jones contends that the groove upon the upper surface of the normal first rib is caused by the pull of the lower cord of the brachial plexus, and not by the subclavian artery as is commonly supposed: and he prefers to call the groove, usually named the "subclavian groove," the "sulcus nervi brachialis." Whether or not the groove is produced entirely by the nerve, to the exclusion of the artery, is a point on which I cannot offer any opinion; but there is no doubt that the nerve-trunks do produce well-marked grooves upon the normal first rib, as can be seen in any well-marked bone (fig. 2). Grooves occupied by nerve-trunks also occur on cervical ribs, over which, in my experience, it is unusual, contrary to the general belief, for the subclavian artery to pass. Such grooves are clearly produced by the pull of the nerve-trunks which lie upon them, and Dr. Wood Jones has shown that the shape and number of the costal elements are directly related to and dependent upon the arrangement of the nerves entering the plexus. It would seem, therefore, that a supernumerary or cervical rib tends to be

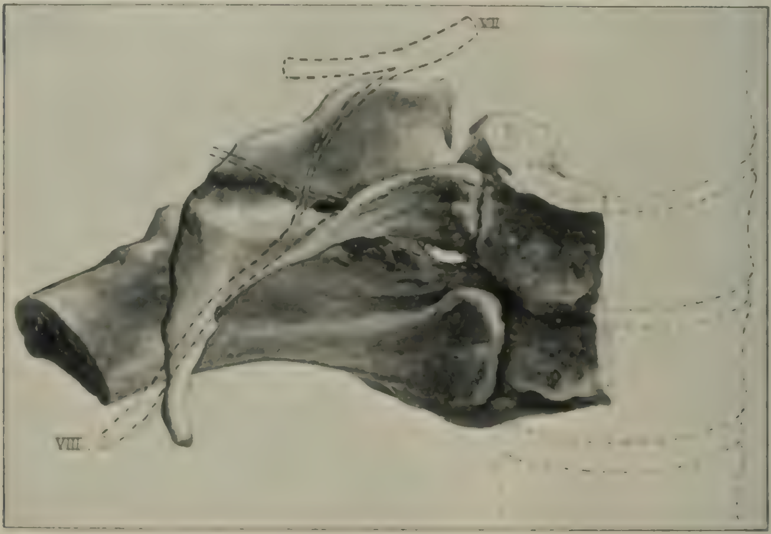

FIG. 1.

Cervical rib from a post-mortem specimen, showing costo-central and costo-transverse articulations; also its relation to the first thoracic rib and, diagrammatically, to the seventh and eighth cervical nerves.

associated with a cephalic movement (prefixed type) and a rudimentary first dorsal rib with a caudal movement of the plexus (post-fixed type). Although this is the general tendency, yet it is only true within certain limits, as may be seen in the drawings of my own cases. More recently Mr. Wingate Todd has published a detailed description of his dissections of four specimens, and on this point concludes that "cervical ribs may be present in cases where the composition of the brachial plexus is normal," and that "the disposition of the nerve-trunks alone is insufficient in many cases to account for the presence and length of rudimentary ribs."

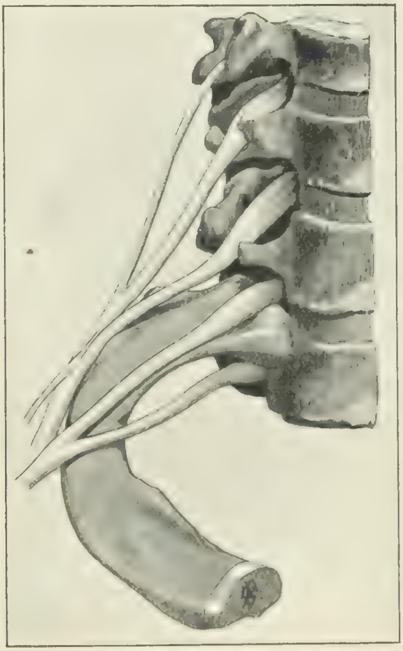

FIG. 2.

Dissection showing the relations of the seventh and eighth cervical and first thoracic nerves to the bones, and the grooves produced by them upon the first thoracic rib.

Rudimentary first dorsal ribs are not so common as accessory cervical ribs, and this fits in with the fact that the post-fixed is not so common as the prefixed type of plexus in man.

A large contribution from the second dorsal nerve, passing out over the first rib would, according to Wood Jones's theory, exert so much pull as to prevent its proper development, so that the rib would be membranous instead of osseous at the site of pressure (fig. 3); or the anterior end might be wanting, and the abbreviated rib pressed down upon and fixed to the rib below. Both these types of rudimentary rib are found represented in accessory cervical ribs (figs. 4 and 5).

Relation of the Brachial Plexus to the Cervical Rib.

The relation of the roots and trunks of a "type" plexus to the normal first rib and to the costal processes of the cervical vertebræ is well shown in fig. 2. The seventh cervical root lies above the costal process of the seventh cervical vertebræ, and crosses the first rib just in front of its angle in a definite groove. The eighth cervical root lies in a groove on the head, neck and shaft, and the first dorsal root occupies the posterior part of the sulcus subclaviæ. The costal process of the seventh cervical vertebra, therefore, projects between the seventh and eighth roots. This may be taken as the normal condition of affairs. When this costal process attains sufficient size to constitute a cervical rib it almost invariably presents a knob which

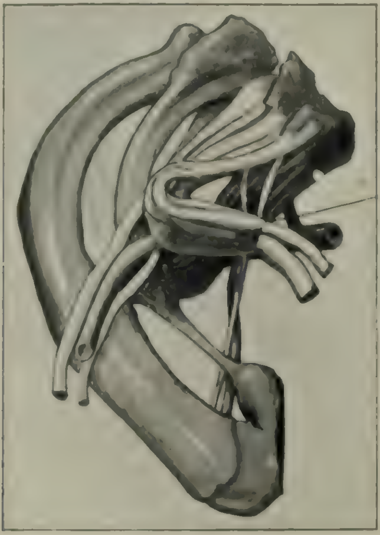

FIG. 3.

Dissection of a rudimentary first thoracic rib which is partly bony and partly membranous.

separates two nerve-roots, commonly the seventh and eighth. The eighth usually passes below and in front of the rib. This knob is an almost constant feature; it can be traced upon nearly all my specimens, and can usually be seen clearly in skiagrams.[1] In one instance, in which the rib was fused with the first dorsal, the eighth root lay below the rib at its point of emergence, and thereafter passed superficially to its anterior end (fig. 5). This is exactly comparable with the relation of the first dorsal to the normal first rib, the point of junction of the two ribs representing the part of the sulcus subclaviæ normally occupied by the first dorsal, and the artery lying in front of that point on the first rib. I could discover no first dorsal root in this case, and, therefore, probably this is an instance of a prefixed

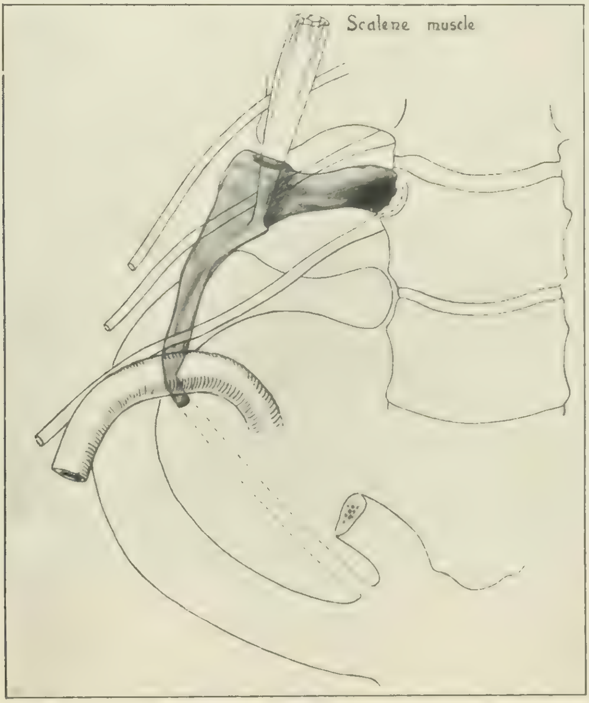

Fig. 4.

Cervical rib resembling the rudimentary first thoracic rib shown in fig. 3.

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[1] *See*, for example, the picture of one of Dr Parkes Weber's cases in *Proceedings*, p. 55.

plexus. This last case supports the theory of Wood Jones, that the pull of the nerve passing over the rib is the cause of its abbreviation.

In the majority of my cases (twenty-two out of thirty-four) I have found that the accessory rib is continued onwards as a tough fibrous band, which represents its non-ossified part. It is exactly comparable with the fibrous continuation of the rudimentary dorsal rib shown in fig. 3. To this band I shall refer presently in discussing the causation of symptoms.

The relation of the artery to the rib is of some interest, because it is usually supposed that the artery is carried over the rib with such constancy as to provide a diagnostic sign. This I believe to be erroneous, and I attach little importance to the supposed elevation of the artery in these cases. The distance to which the subclavian artery rises above the clavicle is very variable, and even apart from its actual position with relation to the vertebræ, its situation varies greatly with the position of the clavicle. Not only that, but it is quite easy to mistake the pulsations of the carotid artery for those of the subclavian. Further, a cervical rib may so thrust the artery forwards as to make its pulsations more obvious.

The various positions of the subclavian artery, therefore, may be summed up as follows: It may lie in a groove upon a rudimentary first dorsal rib; when the anomalous bone is a cervical rib of moderate length it may groove either that rib itself or the first rib immediately in front of their junction; when the cervical rib is short, the artery most commonly grooves the first dorsal rib, passing either anterior to or beneath the fibrous continuation of the accessory rib.

(2) CAUSE OF THE SYMPTOMS.

In some instances, no doubt, the injury of the nerves is brought about by the bony part of a long rib, but in the majority of cases the nerves are not in such relation with the bone that they can be damaged against it. The two lowest roots of the plexus are commonly affected, and these are the nerves which are most often found to be in close relationship with the non-bony part of the anomalous rib. In twenty-two of my cases I have carefully observed this non-ossified portion of the rib. It is a dense fibrous cord embedded in muscle, and attached above to the rudimentary bone, whilst below it is most commonly attached to the first dorsal rib behind the sulcus subclaviæ. The band has many times been observed to be tightened by inspiration, and also by traction upon the arm. I have demonstrated this fact several times by detaching the cervical rib completely from its vertebræ, and observing that it is pulled down away from its former level when an inspiration occurs.

The explanation lies in the fact that the band is attached behind the transverse axis of rotation of the first dorsal rib. As the sternum rises in inspiration the posterior part of that rib tends to be depressed, so that the band, the upper attachment of which is immovable, is tightened. One can also observe that as the band tightens the nerves crossing it are partly lifted forwards and partly rolled over. I believe this to be the manner in which the nerves are damaged, and the possibilities of such damage are increased by any excessive use of the arm. In some cases the onset of the symptoms is sudden and abrupt; in one of my cases it was during childbirth, and in another whilst lifting a patient. It is easy to see how a nerve which is so related to a tight band that it can be affected by repeated slight injuries can easily be severely damaged by a single sudden traumatism.

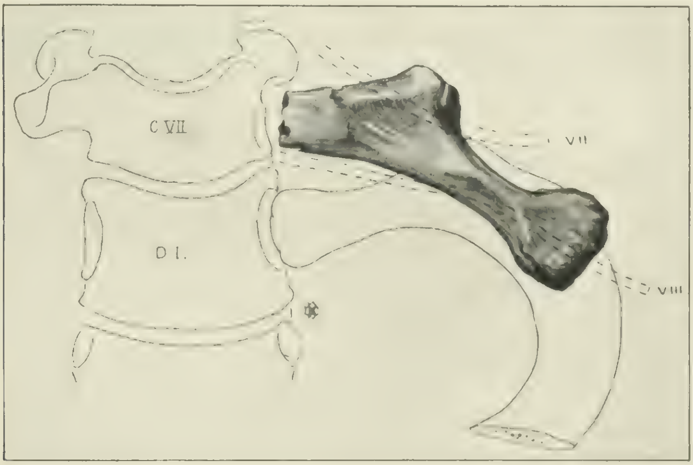

FIG. 5.

Cervical rib pressed down upon and fused with the first thoracic rib.

It has been suggested that a progressive ossification occurs in the band as age advances, and that this may assist in the production of the symptoms. In most of my cases the bony rib ended abruptly, the transition from bone to ligament being clear and precise. In two, however, gritty particles were present in the band beyond a vague and indefinite bony tip. These cases perhaps lend some support to the theory of continued ossification, but I do not think there is much to encourage that view. The largest cervical ribs which I have encountered were in the younger subjects, and with the exceptions mentioned the rib ended in a tip consisting of a bony shell denser than that in the interior.

Anything which tends to depress the shoulders, such as muscular laxity, would naturally encourage the onset of symptoms in a patient with cervical ribs; whilst absence of fat from the supraclavicular region would also tend to bring nerve and band into closer contact. No doubt these are accessory causes, but I believe the salient cause to be the constant rubbing of the nerves over a tight band, due in part to the respiratory movements and in part to the movements of the arm.

(3) THE VALUE OF X-RAYS IN DIAGNOSIS.

It is difficult to lay down any rule as to what, in a skiagram, constitutes a "cervical rib." The degree of the symptoms depends in no way upon the size of the bony portion. A small bony knob which might reasonably be considered little more than a large transverse process may be anchored down to the first rib by an extremely firm band by which the nerve or nerves may be subjected to continual and severe damage. On the other hand, as is well known, a large or even complete bony accessory rib may be present without inconvenience.

Although all the cases in which I have operated have exhibited a definite abnormality of the costal process, I am not prepared to suggest that a costal process which in a skiagram shows no trace of abnormality cannot be causally associated with symptoms of injury to the nerves. Increasing familiarity with the condition causes one to attach less and less importance to the size of the bone as shown on X-ray examination. I believe that the direction of the bony point of the rib is more important than its size, but I have not yet seen enough stereoscopic views of these cases to enable me to speak confidently on this point. I should, however, not be deterred from operating in a case where the symptoms were those of cervical rib, after eliminating all other possible causes, merely because the radiographer's report was negative.

(4) SUMMARY OF CASES.

I have operated upon twenty-nine patients with cervical ribs. In five cases both sides were dealt with, making a total of thirty-four operations.

Sex.—Twenty-six of these patients were females and three males.

Age.—The average age was 38·7, the youngest patient being 13 and the oldest 62. Four were under 20 years of age; five between 20 and 30; two between 30 and 40; seven between 40 and 50; and eight between 50 and 60.

Occupation.—Of the males, two were house-painters and one an artist. In all three pain was the most prominent symptom, and all obtained immediate relief from the operation. None of them exhibited any symptoms or gave any history of lead poisoning. Of the females, fifteen were engaged in domestic work, three were seamstresses, two telegraphists, two clerks, one a pianist, and one a letter-sorter. As regards the side affected in these cases, it is interesting to note that with the seamstresses the right hand was affected, with the two clerks and two telegraphists the left; and in the case of the pianist and letter-sorter both hands were affected.

Symptoms.—The first symptom noticed by the patient was as follows: Pain, seventeen; numbness, five; clumsiness in the fingers, three; and swelling or coldness of the hand, four. The onset of symptoms was ascribed to a definite exciting cause in nine instances as follows: Excessive sewing, two; use of a sewing machine, one; piano-playing, one; house-painting, two; wearing a heavy overcoat, one; lifting a patient, one; and childbirth, one; in the last two the onset of symptoms was sudden. The average length of time between the onset of symptoms and the operation was over three years, the extremes being one month and fourteen years. The symptoms for which the patient sought relief were: Pain alone, four; pain and wasting, nine; pain and numbness, six; wasting and numbness, five; and wasting alone, five. In every case the abnormal ribs were bilateral, though rarely symmetrical; the symptoms, on the contrary, were bilateral in only five of the twenty-nine patients. As a rule, when the symptoms were unilateral the arm on the side of the smaller rib was affected; thus in nineteen cases in which the relation was specially noted the symptoms were on the side of the larger rib in only eight. The actual size of the abnormal bony rib bears no relation to the severity of the symptoms.

Operation.—I do not propose to say much about the operative technique, although there are many points of detail in this connexion which I believe to be of importance. Briefly to mention one or two, I may point out that an essential to success is aseptic healing. There was no instance of wound infection in my series. A second point of importance is the necessity for gentle handling of all the tissues, more particularly of the nerves; the bony points should never be felt for with the finger, lest the nerves be bruised. If the wound is kept dry, as it easily can be, the parts can be dissected and inspected with the utmost ease. I do not now consider it necessary in all cases to remove the bony rib. When this is very short, and the nerves are obviously being damaged only by the non-ossified part, it is sufficient to divide the band, together with the muscle in which it is embedded. I have done this in two instances with a completely satisfactory result. The operation is thereby considerably simplified. I may mention one other point which I consider to be of great importance—namely, that the superficial cutaneous branches of the third and fourth cervical nerves should be most carefully preserved. They are likely to be wounded if the incision behind the sternomastoid is carried too high, and this accident may be followed by persistent pain referred to their area of distribution.

Results.—The immediate result has been bad in two cases. My first operation was followed by a fairly complete paralysis of the upper limb, and this unfortunate sequel was doubtless due to bruising of the nerves against the bones in the attempt to feel the object of attack, with the position of which I was not at that time familiar. Recovery took place slowly, but was not complete when the patient disappeared from observation six months later. The second unfortunate instance was one of complete hysterical monoplegia, for which the patient refused treatment. In this case a functional hemianæsthesia, together with loss of taste and smell, had been present before the operation. A few patients showed a transient numbness or weakness of the arm, from which they quickly recovered. For the rest, the most gratifying immediate results were obtained in those cases where pain had been the prominent feature, in proof of which I may remark that all the five patients with bilateral pain asked to have the other side operated upon. I have never done both sides at one sitting. In only one case was the pain unrelieved and in this patient there was reason to believe that the osteo-arthritic changes present in the cervical spine were responsible for the failure.

The Results of Operative Treatment.

By C. M. Hinds Howell, M.D.

As I have dealt elsewhere[1] with the symptomatology of this condition, I propose to give you briefly to-night the results of operative treatment which I have been able to follow up in twenty-five cases, in which thirty operations were performed. I have personally seen or written to all these patients for the purposes of this inquiry. I wish, at the outset, to express my thanks to my colleagues at the National Hospital, who have very kindly allowed me to trace the after-history of their cases, and also to Mr. Waring and Mr. Rawling, who have given me similar permission with regard to cases operated on by them in St. Bartholomew's Hospital. Five cases of the series have been under my own observation. Operations were performed by Mr. Sargent (twenty), Mr. Armour (three), Mr. Rawling (four), Mr. Waring (one), Mr. Mower White (one), and Dr. Hutchinson (one).

I propose first to consider briefly the immediate effect of the operation itself, for it will be seen that it is often followed by symptoms of more or less severe, though usually transient, character. Thirty operations were performed, as in five cases bilateral ribs were removed. Of the twenty-five patients operated on, of whom two only were males, there were symptoms due to operation in eighteen instances. Such symptoms were pain, more or less severe, in neck, shoulder, or arm, and muscular weakness, which varied considerably both in extent and duration in different individuals. *In eighteen cases pain was complained of*, in some instances being very severe. The duration of this symptom was, as a rule, from one to three months, but there are four cases showing an exceptionally long period of pain, as the direct result of the operation. The periods in these cases are as follows: (*a*) Two in which pain lasted one year; (*b*) one case of pain in the scar and shoulder lasting three years, to the present time, and still present to some extent; (*c*) one case, in which pain is still complained of seven years after the operation; in this case there is, however, a decidedly neurotic element. The pain is peculiarly liable to affect the skin area over the shoulder, and a dragging sensation was complained of in that region in all the cases that persisted over a year. It is probably to be explained by the implication of the superficial cervical nerves in the scar tissue.

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[1] *Lancet*, 1907, i, pp. 1702-7.

Fifteen patients showed motor symptoms.

The usual complaint is of weakness of the whole arm, lasting from two weeks to three months, but in the following cases the symptoms were more pronounced: (*a*) Two patients had a flaccid paralysis of the whole arm for three and four months respectively; this paralysis was accompanied by changes in the electrical reaction, and ultimately there was partial recovery, but only partial in both cases; (*b*) one patient had paralysis of the serratus magnus with considerable, but incomplete, recovery after a year; (*c*) one patient had paralysis of the deltoid and paralysis in the muscles of the hand supplied by the median nerve; the deltoid recovered, but the hand muscles have remained atrophic. In this case there was no muscular weakness before operation, which was undertaken for relief of sensory symptoms. In only *twelve instances was there absolutely no motor or sensory disturbance* following operation.

With regard to the ultimate effects of operation, I propose to divide the cases into four classes, according to the symptoms they exhibited:—

Class I: Vasomotor symptoms only, two cases.

Class II: Subjective sensory symptoms only, five cases.

Class III: Motor symptoms and subjective, but no objective, sensory symptoms, eleven cases.

Class IV: Complete cases which include all the above symptoms, and exhibit in addition objective sensory disturbances.

Class I.—One case, shown by Mr. Waring to-night, in which gangrene was present in the fingers, and which, I think, may be regarded as cured. The second case was one which Dr. Hutchinson, of Lowestoft, sent me, in which symptoms of Raynaud's disease had been present in very marked form for more than two years. There were bilateral cervical ribs, and Dr. Hutchinson subsequently removed the left one, as the symptoms were more pronounced on that side. The result has been very considerable improvement, but not cure. The left hand is now, however, much the better of the two.

Class II: Symptoms purely Subjective—i.e., Pain in the Upper Limb.—Difficulties arise as to the exact diagnosis in such cases as these, brachial neuritis from other causes being common in middle-aged women. As a result, statistics as to the effect of the operation are likely to be misleading. *Five cases of the series are in this class;* seven operations were performed on these. In five instances, pain, which had existed for periods of from eight months to fourteen years, was much relieved in two, and cured in three cases. In two instances, both of these being cases in which two operations were performed at intervals for bilateral ribs, the second operations have been, so far, unsuccessful. In one instance, pain of one year's duration in the right arm has only

TABLE I.—IMMEDIATE EFFECTS OF OPERATION IN EIGHTEEN CASES OF THE SERIES. THE REMAINDER HAD NO POST-OPERATIVE SYMPTOMS.

No.	Name	Sex	Pain	Pain persisting	Motor disturbance
1	A. C.	F.	No pain	—	Arm useless for 2 weeks
2	G. T.	M.	No pain	—	Paralysis of serratus magnus
3	A. W.	F.	Arm and shoulder	1 month	Arm weak 2 weeks
4	A. H.	F.	Pain in scar	Several months	*Nil*
5	M. C.	F.	Shoulder	2 months	*Nil*
6	M. M.	F.	Shoulder	2 months	Arm weak for 2 months
7	D. J.	M.	Arm and shoulder	2 months	Whole arm weak for 3 months
8	F. M.	F.	Slight in arm	1 month	Weakness of arm for 3 months
9	M. B.	F.	Severe pain	Some months	Weakness of arm for some months
10	E. C.	F.	Shoulder and arm	3 months	Arm weaker; wasting of deltoid and median muscles, the latter permanent
11	F. S.	F.	Shoulder	2 months	Whole arm weak for 6 months
12	N. W.	F.	Arm and shoulder	6 months	Paralysis of arm for 6 months, with alteration in the electrical reactions; still weaker than before operation 14 months ago
13	A. T.	F.	Elbow to fingers	12 months	Very weak for 12 months
14	R. H.	F.	Arm and shoulder; much pain	14 months	Arm weak for 2 months
15	M. D.	F.	Arm and shoulder	7 years	Flaccid palsy, complete for 3 months; little or no strength for 12 months; hysterical element in this case
16	E. W.	F.	Severe, neck and shoulder; persists, but improving	3 years	Marked weakness; arm almost paralysed after operation for 2 months
17	E. D.	F.	*Right arm operation:* very severe pain	6 months	*Nil*
			Left arm operation: very painful	10 months since operation; still persists	*Nil*
18	E. T.	F.	In shoulder	8 months; still persists	Arm weaker since operation

been partially relieved, eighteen months after the operation. In the second case, pain of four years' duration in the left arm, with operation in April, 1912, is still unrelieved.

Class III.—Eleven cases. Of these, four are greatly improved. In all these the pains have ceased, the hand muscles have increased in size, and strength has returned. The circulation has also greatly improved. I do not think that any of these cases with muscular wasting and vasomotor disturbance, due to cervical rib, are ever *completely* cured as a result of operation. In my experience, neither the muscles nor the circulation ever *quite* regain the *status quo ante*, though for all practical purposes the patients may be regarded as cured. *Four cases have improved.* In one of these the muscles of the hand are again almost normal, but pain persists from the scar. Still, so far as the symptoms referable to the rib go, the patient is practically cured. Another patient, a year after operation, finds the muscle still wasted, but the power of the hand "a lot stronger," and in the third case "there is considerable improvement" in the muscles, and the hand can now be used, whereas before the operation it was useless. There is, however, still pain in this case. The fourth case shows improvement in the wasted muscles, and as only six months have elapsed since the operation, it is probable that the case will make still further improvement. *One case shows no change.* In this case, a band running from the tip of the cervical rib to the first dorsal rib was removed, the cervical rib being left in position. The operation was bilateral, and a similar condition was found on either side. *Two cases are worse than before operation.* In one, eight months after the operation, the arm is

TABLE II.—CASES WITH SUBJECTIVE SENSORY SYMPTOMS AND NO MUSCULAR WASTING.

No.	Name	Sex	Age	Duration of symptoms	Date of operation	Result as regards symptoms complained of
1	E. C.	F.	38	3 years	1908	Cured
2	M. C.	F.	52	3 years	November 29, 1911	Pain relieved
3	A. W.,	F.	53	8 months	August 20, 1912	Pain relieved
4	E. S.	F.	55	1 year, right and left arms	(*a*) April, 1911, left (*b*) June, 1911, right	Cured Unrelieved
5	E. D.	F.	39	Pain, 14 years right Pain, 4 years left	(*a*) July, 1911, right (*b*) April, 1912, left	Cured Unrelieved

still weaker than it was before, and there is no improvement in the muscles. In the other, two years after the operation, the arm has only partially recovered from the flaccid palsy which followed operation.

Table III.—Motor Symptoms and Subjective Sensory Disturbance, but no Objective Sensory Loss.

11 Cases.

No.	Name	Sex	Age	Duration of symptoms	Symptoms	Time since operation	Result
1	R. H.	F.	31	18 months	Severe pain, left; slight wasting of thenar muscles and interossei, left	14 months	Muscles increasing; hand stronger; no pain
2	F. M.	F.	53	4½ years	Severe pain, right; wasting of thenar muscles and first and second interossei	2 years	Muscles increasing; hands decidedly stronger; no pain
3	D. J.	M.	31	2 years	Slight pains for 1 year; wasting of hand and forearm, right	1 year	Hand much stronger; muscles still wasted; no pain
4	A. C.	F.	26	18 months	Paræsthesia of thumb and first and second fingers; wasting in thenar muscles	18 months	Hand stronger; muscles increasing; no paræsthesia
5	N. W.	F.	40	2 years	Pain, right; wasting of interossei and thenar muscles, left	2 years	Worse; flaccid palsy of arm, from which only partial recovery
				9 to 10 years	Ditto, left	2 years	Improvement
6	F. S.	F.	41	18 months	Wasting in left hand and forearm	3 years	"Arm and forearm splendid"
7	N. W.	F.	45	2 years	Pain in arm and shoulder, left; wasting of thenar muscles, left	3 years	Muscles almost natural again; pain severe, still in neck and shoulder
8	E. T.	F.	28	12 months	Paræsthesia; wasting of intrinsic hand muscles	8 months	Arm weaker than before; no improvement in muscles; pain in shoulder
9	A. T.	F.	43	18 months	Pain in shoulder and arm, right; wasting of intrinsic hand muscles, right	2½ years	Considerable improvement in muscles; can now use hand; still some pain
10	E. I.	F.	43	12 months	Wasting of thenar muscles, right and left; pain right and left for many years	6 months, right and left	No improvement; *band* only divided and rib left behind
11	M. M.	F.	50	Years	Pain in right and left arms; wasting of intrinsic muscles, right and left	6 months	Some improvement in muscles; less pain

Class IV: Complete Cases.—There are seven patients in this group. Two of them may be regarded as greatly improved, four others are certainly benefited, and one is certainly worse as the result of the operation. In the case of the two patients who are greatly improved, symptoms had only existed for eighteen months and seven months respectively. This is what one would naturally anticipate, for it would be unreasonable to expect to cure the muscular atrophy in such a patient as Mr. Rawlings's second case shown to-night, where the wasting is of many years' duration. Yet in this case the pains, which were very severe before operation, have been cured, and the patient has returned for a second operation on the opposite rib, which has now begun to give rise to symptoms.

I think it would be fair to summarize the results of operative treatment of these cases as follows: In a large proportion of cases some symptoms, such as pain and weakness in the arm, may be expected to follow the operation, but not to last more than three months or so. The vasomotor symptoms, which are present in almost all the cases, will be certainly improved, and in the majority of cases pain will be relieved or cured. With regard to muscular weakness and atrophy, the expectation is that the operation, if it is not too long delayed, will greatly improve this condition. There is not, as a rule, complete restoration of the wasted muscles, nor complete recovery from the vasomotor disturbance.

TABLE IV.—COMPLETE CASES.

No.	Name	Age	Duration of symptoms	Time since operation	Result
1	A. H.	22	2 years	22 months	Improvement in muscles, in circulation and in sensation; absence of pain
2	M. B.	36	15 years	7 years	No improvement of muscles, but improvement of pain
3	F. T.	44	18 months	3 years	Great improvement in every respect
4	G. T.	37	5 years	1 year	Very slight muscular improvement; circulation and sensation much improved
5	F. V.	27	8 years	1 year	Some improvement of muscles; circulation better; sensation as before
6	M. D.	49	10 years, pain; 1 year, wasting	7 years	Worse; still pain; flaccid palsy of arm followed operation, from which patient has not yet recovered completely; large neurotic element in this case
7	K. R.	41	7 months	1 year	Improvement marked in muscles, sensation and circulation

Some Points in the Symptomatology of Cervical Rib, with Especial Reference to Muscular Wasting.

By S. A. Kinnier Wilson, M.D.

The symptomatology of cervical rib is wide and varied, and it is not my intention to discuss it this evening with anything approaching to completeness. Such matters, therefore, as the shape of the neck in cases of cervical rib, the position of the subclavian artery—which in all probability is of much less significance than was formerly supposed—the question of difference in the radial pulses, and so on, will not be referred to here. Ordinarily, the symptoms of cervical rib may be divided into those of the motor, sensory, and vasomotor systems respectively. Again, for the sake of brevity, no reference will be made in this place to the last of these, although vasomotor symptoms, when they occur, are of considerable interest.

As far as sensory symptoms are concerned, they may be classed as subjective and objective. In the former group are placed the symptoms which are likely to be those to which the patient's attention is primarily directed. In the great majority of cases he complains of tingling, numbness, pins and needles; in a word, of paræsthesiæ in the hand and fingers, often in the finger-tips. In a majority of cases, moreover, these symptoms are unilateral, often strictly so; hence, unilaterality of paræsthesiæ is of importance in coming to a diagnosis. Again, it is in my experience very frequently the case that they are referred either to the ulnar or to the radial side of the hand, much more frequently than to all the fingers. This is a matter which should be carefully inquired into, and may constitute a point of diagnostic value. Pain, similarly, usually described as shooting or darting, is an early symptom, and its distribution is identical. It is rare to find that the pain is described as actually starting from the region of the neck or shoulder; commonly it is experienced only in the forearm, hand, or fingers, and most patients say, further, that it always radiates in a downward direction. The possible significance of downward as opposed to upward radiation is a difficult matter. In most cases of trigeminal neuralgia the pain is described as radiating from the periphery centralwards; in tabes, the lightning pains apparently radiate always upward in some cases, and always downward in others. It is conceivable that where the lesion is in the sensory ganglion itself pain is described as radiating towards the centre, whereas if the lesion is distal to that point the pain radiates towards the periphery. Examination of a number of cases of different varieties of neuralgia, however, has not enabled me to establish this distinction with any certainty.

Objective sensory disturbances are notoriously variable. In some cases no definite alteration in cutaneous sensibility can be discovered. In others there is a diminution or loss to all forms of cutaneous sensibility, the limits of each not always strictly corresponding, in the distribution, roughly speaking, of the radial or ulnar nerve. It will be found, as a rule, that the distribution is never exactly that of either of these nerves, and, on the other hand, it does not exactly harmonize with a root supply, at least not in its entirety. Clinical experience shows us that sometimes only that part of a root distribution which concerns the fingers is impaired, while the remainder of the root area escapes. In fact, the cutaneous loss, in relation to the motor loss, may present anomalies difficult of explanation.

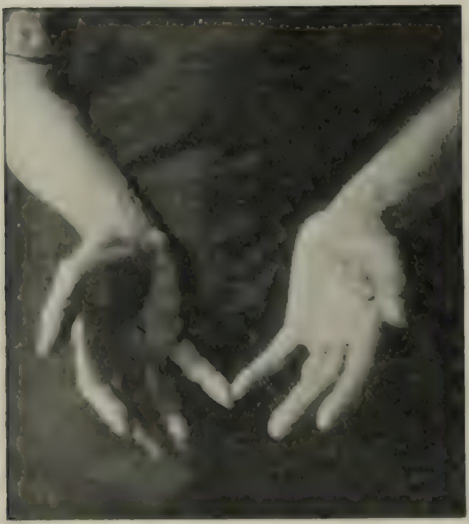

Fig. 1.

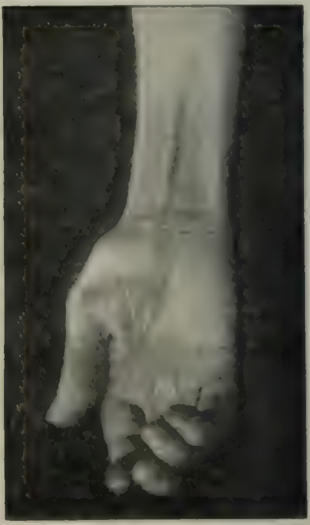

Fig. 2.

It is my purpose this evening to direct attention more particularly to the motor phenomena accompanying the lesion, again premising that such motor phenomena may not be present in a given case; when they do occur their nature is such that it is, in my experience, of considerable diagnostic, if not actually of pathognomonic, significance.

There are two main types of muscular involvement. The first of these, which may be designated the median type, is very frequent, yet apparently it has not been noted or sufficiently emphasized by previous writers on the subject. The first case of cervical rib that came under my observation was under the care of Dr. Buzzard in the National Hospital in 1906, and was eventually operated on by Mr. Sargent. A photograph of this patient's hands is reproduced as fig. 1. It will there be seen that there is a curiously local early wasting of the muscles of the thenar eminence on the right side, in which the muscles involved are the abductor pollicis and opponens pollicis alone; all the other thenar muscles, including the flexor brevis pollicis, being intact. This definitely partial atrophy of the thenar muscles has come under my notice a large number of times, as a glance at the series of figures (figs. 2 to 6) will show. Sometimes the wasting is comparatively slight, sometimes it is profound, as in fig. 6, and yet the other thenar muscles escape. These cases have been under the care of one or other of my colleagues at the National Hospital, to whom I wish here to express my gratitude for kindly granting permission to refer to them. Fig. 6, for instance, representing the hands of a patient under the care of Dr. Aldren Turner, is peculiarly instructive. The outline of the metacarpal bone of the thumb is very readily traceable through the skin, the bone being

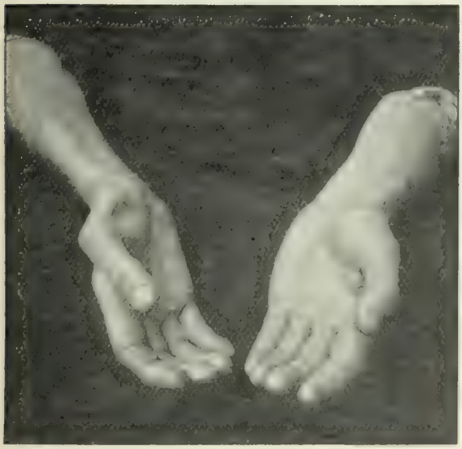

FIG. 3.

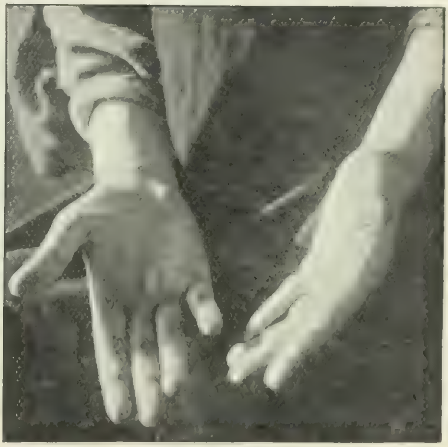

FIG. 4.

practically subcutaneous as a result of the atrophy, and the flexor brevis pollicis remains, perhaps not of normal size, but only little wasted and not paralysed, while the adductor groups are quite normal. Fig. 5 is from a case under the care of Dr. Farquhar Buzzard. It shows the special form of thenar atrophy very clearly. As a result of operation in this case the muscular wasting has largely disappeared. Again, through the kindness of Dr. Parkes Weber I was afforded an opportunity of examining for myself the patient with cervical rib whose hands are shown in fig. 4. This patient was seen only a week ago, and she presents the same muscular lesion as the first case of seven years previously.

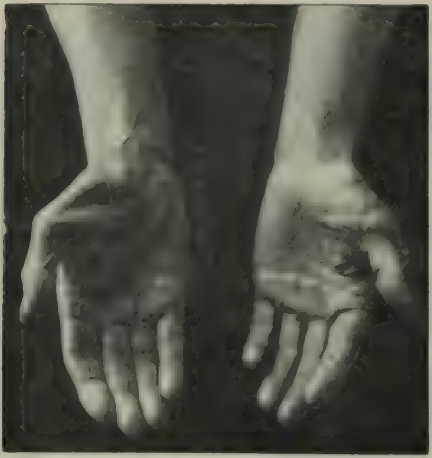

Fig. 5.

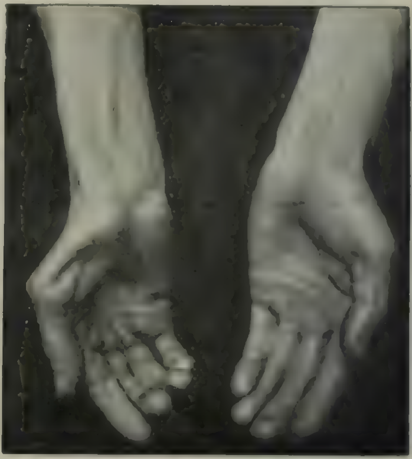

Fig. 6.

The importance of this local atrophy will be recognized when it is remembered that the median nerve supplies the abductor pollicis, opponens pollicis and flexor brevis pollicis; yet of these muscles two are taken and the other left. This can only mean, I submit, that although their peripheral supply is from the same source their root supply is from two sources, probably the seventh and eighth cervical; for these are the roots that may be involved in this condition, and the lesions of cervical rib, therefore, afford interesting evidence of the exact segmental supply of these muscles, and serve to establish their position in the cord. It may be fairly concluded that the root supply of the abductor pollicis and opponens pollicis is the seventh cervical, and of the flexor brevis pollicis the eighth cervical.

Further, a curiously local atrophy of this kind, of which the series of photographs furnish striking evidence, contrasts so strongly with what obtains in progressive muscular atrophy that it is difficult for confusion to arise, although it has done so in the past. In the latter condition the atrophy is essentially a *global* atrophy, that is to say, it affects the muscles of the thenar group as a whole, and though it be slight to begin with, the observer will never find that selective atrophy which it has just been shown is so frequent in cervical rib. The figures here reproduced also indicate that the hypothenar eminence may remain unaffected, in fact does remain unaffected, although the atrophy of the two muscles concerned is complete. In progressive muscular atrophy, to have so great a change unaccompanied by atrophy in the hypothenar group would be a rarity, if indeed it ever occurs at all. In this way, therefore, the muscular lesion here described has often proved of considerable diagnostic value.

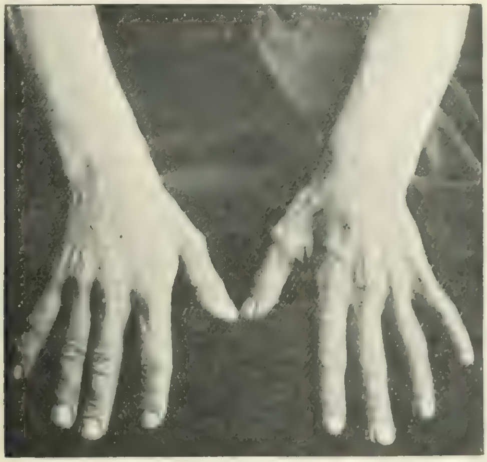

FIG. 7.

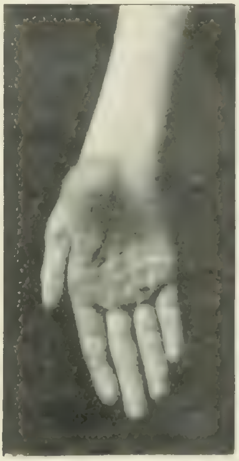

FIG. 8.

This local atrophy presents another point of interest. In Mr. Sargent's interesting contribution to the evening's discussion, attention was drawn to the importance of the fibrous band, which is often all that represents the continuation of the aborted cervical rib to its insertion in or junction with the first dorsal rib. The roots which pass over the rib and its fibrous prolongation are the seventh and eighth cervical, and the symptoms of cervical rib are due to the involvement of one or other or both of these roots. Now in more than one of the patients who have had the local thenar wasting already referred to, the sensory change has been along the radial border of the wrist, hand and fingers, that is to say, a distribution which from a root point of view is accepted by neurologists as representing the sixth cervical. That root, however, is not involved in cervical rib. We therefore find ourselves face to face with an apparent anomaly. The only way, so it appears to me, in which this can be satisfactorily explained is by utilizing our knowledge of the facts of pre- and post-fixation of the plexus, and by supposing that in cases of post-fixation the seventh root and not the sixth is the one that is implicated.

The other type of muscular atrophy in cases of cervical rib corresponds, roughly speaking, to an ulnar distribution, in other words, we find general wasting of the interossei and an approximation to the *main en griffe*, as in fig. 8. In one of the cases shown here this evening, by Dr. Hinds Howell, it is specifically noted that all the hand muscles are wasted, with the exception of the two above referred to, indicating that the eighth cervical was the root involved. In most of the cases of this kind it will be found that the patient complains of paræsthesiæ along the ulnar border of the hand and in the ulnar fingers, whereas in the cases offering the local thenar atrophy of the first type the paræsthesiæ are on the radial side. Fig. 7 also represents the eighth cervical type, with early wasting of the abductor indicis and interossei generally. In this case the thenar muscles escaped, so that it is a purer instance of the second type than the case represented in fig. 8, which is really a combination of the two. This patient was operated on by Mr. Donald Armour, and here also marked amelioration of the muscular defect has ensued.

It should be mentioned that there is another possible explanation of the local thenar atrophy—viz., that when a nerve trunk is involved in a lesion, certain muscles as a result may become impaired before others do; that is to say, there may be a selective action. This was pointed out by Sir David Ferrier in a paper on "Atrophic Paralyses" which appeared in *Brain*[1] thirty years ago. He believes there is a special proclivity for abductor muscles to be paralysed before adductors. Whether this explanation will suffice in cases where the abductor and opponens wasting is profound, while other muscles supplied by the same nerve are still normal, seems doubtful.

Occasionally muscular cramps in the hand and fingers are noted by the patient, without any muscular atrophy being discoverable, but it is not necessary to dwell further on this symptom at present.

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[1] *Brain*, 1881-82, iv, pp. 217, 303.

Dr. FARQUHAR BUZZARD said he had been particularly interested in Dr. Wilson's observations about the types of muscular atrophy occurring in cases of cervical rib, and especially in relation to that type in which there was marked wasting of the abductor and opponens pollicis with comparative escape of the flexor brevis. He had noticed and figured a similar appearance in some cases which he had described under the title of uniradicular palsies of the brachial plexus about ten years ago. He was now convinced that some of those cases were cases of cervical rib, but the special connexion between the type of atrophy referred to and cervical rib had never occurred to him until Dr. Wilson had pointed it out.

Dr. Buzzard drew attention to a case he was showing in which there had been a complete recovery from muscular atrophy in the hand, after removal of the cervical rib by Mr. Sargent, and he referred to it as an exception to Mr. Thorburn's disappointing experience in this type of case. The recovery was to be attributed to the fact that operation was performed within six months from the onset of symptoms.

For some years he had been submitting almost every case in which pain in the arm was the predominant symptom to an X-ray examination of the neck. The results of these observations had been rather interesting, because they had convinced him that pain of almost any description and any distribution within the upper extremity could be associated with the presence of cervical ribs. He used to hold the opinion that in the cervical rib cases the pain was nearly always distributed longitudinally on either the radial or the ulnar side of the arm and hand. Now, however, he had come to recognize that cases of typical acroparaesthesia with pain confined to the hand and fingers could also be associated with cervical ribs. He referred to cases, generally in middle-aged women, who complained that soon after going to bed they woke up with intense burning pain in the hands and fingers. This was relieved to some extent by keeping the hands outside the bedclothes, but disturbed nights often resulted. When they got up in the morning their hands felt useless and clumsy, but after a time this would improve and the patient be able to carry on her work throughout the day. The pain and discomfort would return again at night. In several cases of that kind there were distinct cervical ribs, and in one such case Mr. Sargent had operated and given the patient relief. On the other hand, he had found that the symptoms could be relieved by other methods in several cases. Cervical ribs were also present in some cases of occupation neuroses associated with the upper extremity. He was convinced that the question of occupation was a most important one, almost as important as the presence of the rib. There were a number of patients who first showed symptoms after changing their work or after beginning to work for the first time. Other patients were relieved by change of occupation. One lady he knew always had pain in the right arm when she wore a heavy fur coat, and subsequently lost the pain when she gave up that particular garment. The pain returned with driving and piano-playing, two forms of occupation in which the arm was used extended in front of the patient. In the course of operations which Mr. Sargent had done on his cases it had been demonstrated that the tension on the cords of the brachial plexus was more pronounced when the arm was extended forward than when it was abducted. Further, it was always intensified by inspiration. The pain of brachial neuralgia, however, was generally exaggerated by a forward position of the arm and by inspiration, whether a rib was present or not. He doubted whether there was anything about the sensory symptoms which were quite characteristic of cervical rib. Dr. Buzzard also referred to two cases of cervical rib in which vasomotor disturbances were the prominent features. One of them had been operated upon with excellent results. In conclusion, he felt that the cervical rib was only part of an anatomical peculiarity and that the questions of occupation and posture were extremely important with regard to the development of symptoms. Operation was the only treatment in some cases, but there were others of a milder type in which other measures, for instance, change of occupation when it was possible, might be attended with success.

Sir RICKMAN J. GODLEE sent the following communication: I have taken much interest in the question of cervical ribs ever since I saw Lister remove one—in the days when they were called exostoses—from a middle-aged major, who found trouble in raising his rifle. He was cured by the operation. I have never seen serious consequences follow the operation. My impression is that pain is always relieved, but that the recovery from paralysis is generally very slow. My principal object in writing is to say a word about the method of performing the operation. One reads shocking accounts of its difficulties and dangers. I have removed a considerable number of cervical ribs, large and small, and have not met with the difficulties described. I believe they may be avoided by making a sufficiently long *transverse* incision *above* the swelling—not over it—and first exposing the upper part of the brachial plexus without thinking about the rib. A broad retractor placed on the plexus then pulls it downwards, and with it goes the suprascapular nerve, which should thus stand no chance of injury. If the retractor is broad, no damage will be done to the plexus even by firm traction. By placing another broad retractor on the inner part of the wound, the sternomastoid, the large vessels and nerves, and the anterior scalenus are drawn aside, and the rib is satisfactorily exposed. By adopting this procedure the subclavian artery is never seen. The only vessels that come into view are the transverse cervical, and they are easily dealt with. I think the troubles that have been experienced must have come from (1) making a vertical incision, and (2) attempting to work straight down on the rib by separating the cords of the plexus. I have generally, but not always, left the periosteum. This avoids any risk of damaging the pleura; and I have not seen any trouble from re-formation of the rib. It is possible by such an incision as has been described, in most cases, to remove the head of the rib if one be present; but this appears to be an unnecessary refinement.

DEMONSTRATION OF CASES OF CERVICAL RIB.

Case of Cervical Rib (Operation).

By C. M. Hinds Howell, M.D.

K. R., a woman, aged 41, has the following history: Eighteen months ago she noticed that her left hand was becoming weak and the muscles wasting; there was no pain in the hand or arm. She was admitted into the National Hospital under the care of Dr. Risien Russell. Examination showed wasting of all the intrinsic left-hand muscles except the abductor and flexor brevis pollicis. There was also weakness and some wasting in the flexor profundus digitorum; reaction of degeneration in wasted muscles. There was slight, ill-defined sensory loss to cotton-wool and pin-prick in the hand and ulnar side of forearm. There was definite vasomotor disturbance in left hand. X-rays showed bilateral cervical ribs.

Mr. Sargent operated, and removed the left rib, which was noticed to be in direct contact with the lower trunk of the brachial plexus. Result of operation: (1) Much increase in power of hand, with marked diminution in wasting of the affected muscles; (2) the sensory disturbances have quite gone; (3) still some slight vasomotor disturbance, but this is not nearly so severe as it was.

Two Cases of Cervical Ribs (Operation).

By L. Bathe Rawling, F.R.C.S.

Case I.

A. H., a woman, aged 23. The patient gave the following history: She had complained of pain in the left hand and arm at times since the age of 14. There was marked tendency for the left hand to feel cold and to go "blue"; particularly was this so in the case of the third and fourth fingers. For eighteen months or so before being seen the muscles of the left hand, and to a less extent the flexors of the wrist and fingers, had been wasting and were weak. When examined the intrinsic muscles of the hand were much wasted, and all, with the exception of the adductor transversus pollicis, showed reaction of degeneration (Dr. Lewis Jones). There was, in addition to the muscular symptoms, definite anæsthesia along the ulnar side of the hand and the ulnar side of the forearm (C_8 and D_1 segments), and also definite vasomotor disturbance in the left hand. X-rays showed bilateral cervical ribs. The left rib was removed.

Result: (1) Pain in the hand and arm ceased at once and there has been no return of this; (2) the circulation has been greatly improved, the hand never goes numb and blue like it used to; (3) the power of the hand and muscular wasting have both improved very much, but are not yet normal—she can only "type" with the second finger, the first and third seem to have no pushing power; (4) the forearm has improved in power considerably.

[N.B.—Vasomotor disturbances are now beginning in the right hand.]

Case II.

M. B., a woman, aged 36, has the following history: Aged 14: Noticed great pain in arm and right shoulder; right hand weak, so that she dropped things. Aged 18: Wasting first noticed in right hand, beginning in thenar muscles; this was progressive, pain continued, and hand and arm became numb. 1906, aged 29: Came under observation; marked muscular atrophy in all intrinsic hand muscles, right, with weakness and wasting in flexors of wrist and fingers; cutaneous sensory loss in C_8 and D_1 segments.

X-rays showed bilateral cervical ribs. Operation was performed and right rib removed.

Result: (1) Very great pain for some months after operation in shoulder and arm: this has now gone, as well as the pain experienced before operation; (2) great weakness of the whole arm for some months after operation: gradually recovered. No improvement in wasted muscles, and very little in sensory loss.

Aged 35: Complained of pain in left arm and shoulder, then weakness and wasting began to appear in intrinsic muscle of left hand, and slight sensory loss in C_8 and D_1 segments, left. Patient returned to hospital to have the left rib removed. Operation (January, 1912): Left rib removed.

Case of Bilateral Cervical Ribs; Operation upon the Left Side—Unsatisfactory Result.

By F. J. Poynton, M.D.

M. L., aged 43. This patient was shown at the Medical Society of London in November, 1911, by Dr. Poynton, with the special intention of obtaining the opinion of Fellows upon the advisability of an operation. The case was called then one of "loss of power in the hands of four years' duration." The duration of the symptoms had been four years, and had been attributed to excessive work at scrubbing pans. The weakness had increased and the hands had become useless. The chief complaints had been of severe pain in the thumb and hollow of the left hand, passing up the radial side of the arm to the supraclavicular fossa. To a lesser degree this was present on the right side. There were also occipital headaches, and cramp-like pains in the sole of the right foot and the right side of the abdomen. The muscles of the thenar and hypothenar eminences were wasted more on the left than the right. The peculiar distribution of this wasting, described by Dr. Kinnier Wilson, is well shown. The interossei were slightly wasted on the left side. Both pupils were small. Some vague blunting of sensation (tactile) was elicited over the forearms and hands. The left hand twitched more than the right. The mental state, very responsive to suggestion, was unsatisfactory. The skiagram showed two cervical ribs.

The case seemed an unfavourable one, and yet the patient's hands appeared to be not only painful but useless. At the meeting diverse opinions were given—the majority in favour of operation.

Some weeks later (December, 1911) the left cervical rib was operated upon. There was improvement for a while, and the muscles of the left hand became stronger, but for the last six months the condition has been very unsatisfactory. There is continual complaint of severe pain in the left shoulder and up the left side of the neck, and the patient is unable to work or do anything with his hands.

Case of Cervical Rib (Operation).

By R. H. ANGLIN WHITELOCKE, F.R.C.S.

E. G., AGED 25, states that ten years ago, one evening after dancing, she suffered a "burning, pricking pain in the right inner forearm, extending from the finger-tips to the elbow and settling there." The pain lasted some seconds and returned at irregular intervals, lasting longer and becoming more violent as time went on. A few weeks later she noticed an occasional helplessness in the thumb and first finger, so that she was unable to pick up small objects, the difficulty increasing steadily, though slowly. She discovered that during these phases of loss of power the lower forearm and hand were cold and that by plunging these parts into warm water not only was the sense of cold dissipated but the muscles regained their use.

About three years ago it was observed that the muscles in the wrist, thumb, and palm were visibly wasting. The symptoms steadily became worse; in addition to increased loss of power, the pain became more severe and constant. During this time the patient was having no special treatment, but was advised to go to the National Hospital in Queen Square. There she saw Dr. Batten, who had a skiagram taken and advised an operation for the relief of the symptoms.

In July, 1912, the patient was admitted, under my care, at the Radcliffe Infirmary with the above history, adding that the right hand became spasmodically semi-clenched two or three times a day. Examination revealed wasting in the thenar and hypothenar eminences, more especially in the former, also shrinkage of the dorsal and palmar interossei and the muscles of the forearm. The hand-grip was feeble. There was slight diminution of sensation on the ulnar side of the hand and more markedly so in the palm. There was also tenderness on pressure over the course of the brachial plexus and near the angle of the scapula. A skiagram revealed the presence of a cervical rib on either side, the right one being slightly the larger.

On August 2 an operation was performed, the details of which are as follows: A 4-in. incision was made immediately above the clavicle, along the lower border of the supraclavicular triangle. The skin, platysma muscle, and superficial layer of the cervical fascia were divided together and drawn upwards. The transverse cervical, and suprascapular veins were cut across and tied, while the corresponding arteries, together with the posterior belly of the omohyoid, were retracted upwards. The scalenus anticus muscle was defined and followed downwards to its tubercle of insertion on the first rib. The subclavian vessels were then displaced downwards and held aside, when the accessory rib could be distinctly felt. It presented a tuberous extremity, devoid of cartilage, from which passed a strong fibrous ligament to be attached to the first rib close to its angle. Passing over the extremity of the rib, proximal to its ligamentous attachment, could be felt the lowest trunk of the plexus formed by the eighth cervical and first dorsal nerves. This trunk was tense and could be felt to be stretched in its course as it passed over the rib near its tip. With a pair of cutting bone-forceps I removed the end of the rib for about $\frac{1}{3}$ in. as well as the dense ligament attached to it. The nerve-trunk having been drawn forwards, passed under the divided rib, whereby its tension was relieved. The parts were then restored, the fascia and platysma accurately united with fine catgut, and the skin by a subcuticular suture of silkworm gut. The healing was by primary union. The incision was planned with the object of leaving as little disfigurement as possible and as of being, perhaps, the most direct route of approach to the rib. The dissection necessitated caution, and although deep, when the subclavian vessels had been retracted, afforded sufficient space for the resection of the bone.

Skiagrams show that there is a corresponding rib on the other side of the neck, but its presence has, so far, not accounted for any symptoms. On the side of the operation, the absence of the tip of the rib is all that is noticeable.

For a few days after the operation there was increased pain in the forearm and hand and in the region of the wound, but this gradually subsided. After the wound healed, and for some weeks later, the loss of power was increased so that the patient could not even close her hand. Lately, the pain has diminished and the power is returning. The hand cannot be closed spontaneously, but the atrophy of the thenar and hypothenar muscles is less noticeable. The increased pain after operation was controlled by aspirin and the salicylates; massage and electrical stimulation have been used and are being continued.

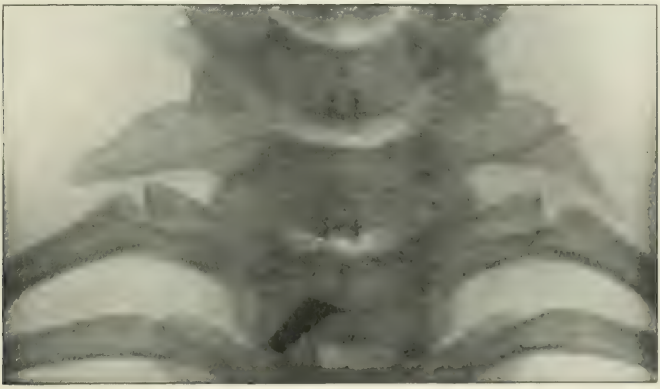
I believe the case to be steadily improving and that the prognosis as to restored functions is, on the whole, good.

Mr. WHITELOCKE added that the girl had no occupation, yet her symptoms were severe and aggravated. She showed not only the neuralgic group of symptoms, but those of the paralytic or atrophic group. There were serious symptoms at first, but she was now rapidly improving.

Bilateral Cervical Ribs; Symptoms on Right Side only; Operated on Twice—last Operation January, 1912.

By A. S. BLUNDELL BANKART, M.C.

E. M., FEMALE, aged 33 (under the care of Dr. Wilfred Harris at Maida Vale Hospital). Symptoms began with pain in right shoulder, which she had for two years before seeking medical advice. Pain worse on exertion. Also had "pins and needles" in ulnar side of right forearm and hand. Treated by hot-air baths and electricity for "neuritis."



Bilateral cervical ribs.

Then she developed pain in the neck, which was very much worse than the original pain in the shoulder. Operated on in June, 1910 (eighteen months before I saw her), through a lateral transverse incision. Rib not found; symptoms aggravated; extremity of rib palpable and tender. Radiating pain over side of neck and shoulder. No wasting of hand muscles. Skiagram showed well-marked cervical rib on each side. No symptoms on left side. Operation (January 8, 1912): Rib removed through posterior incision. Patient relieved of all symptoms.

A Case of Removal of a Left Cervical Rib.

By WILLIAM SHEEN, M.S.

E. E., FEMALE, aged 15. May, 1912: Commenced to learn violin; daily playing, no "pad" used. June: Left hand occasionally numb and cold, with blue nails: father, who is a doctor, found radial pulse "small." August: Condition more persistent; arm easily tired; painful, particularly when dressing: maid had to do her hair; tingling; pain in thenar eminence, particularly when stretching octaves. October: Violin lessons given up because of hand aching and getting white when playing. November: Father found no radial pulse. December: Dr. Mitchell Stevens saw patient, had skiagram taken, and diagnosed cervical rib.

Seen by Mr. Sheen on December 21. Fully developed, healthy girl. No pulse left limb. Limbs slightly smaller throughout than on opposite side. Hand pale and cold; nails bluish. Hard, painless swelling above middle of clavicle, with pulsation over it. Condition of limb stated to be intermittent; hand sometimes warm and nails pink; worse when cold, on exertion, when hand held above head, when nervous. Pain and wasting in thenar and hypothenar eminences. Pain in inner side of elbow when elbow fully extended; elbow then held flexed to side with other hand. Right side: Nothing definitely abnormal.

Skiagram: Left cervical rib; cervico-dorsal scoliosis, convexity to left.

Operation (January 10, 1913): Transverse incision above clavicle. Pulsating subclavian artery coming down almost vertically, with cords of brachial plexus outside it. Vein not with artery, but parallel to clavicle. Artery and nerves retracted out: rib exposed. Pleura separated from inner margin of rib; not opened. One and a half inches of rib removed, including articulation with, and fragment of, rib below (removed rib shown).

Coloured drawing (kindly made by Mr. Owen Ll. Rhys from pencil sketch of Mr. Sheen's) exhibited to show relation of parts at operation.

February 8 (one month after operation): No pulsation, a little swelling above clavicle. Limb pulses not reappeared. Nails pink and hand warm in all positions, quite different from before operation. Limb still smaller than other, and thenar and hypothenar eminences flattened.

Dynamometer: Left, 80; right, 100. Patient expresses herself relieved of all symptoms; able to do her hair, play piano without pain, &c. Skiagram shows site of removal.

Patient shown at meeting (February 14) five weeks after operation.

The skiagrams (figs. 1 and 2), kindly taken by Mr. Owen Ll. Rhys, are

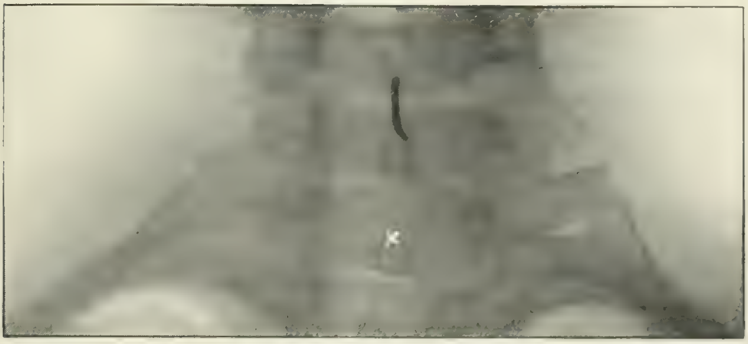


FIG. 1.

Before operation. × is on body of first dorsal vertebra.

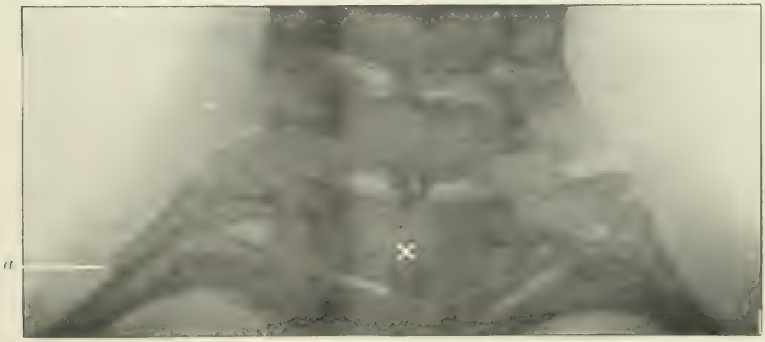


FIG. 2.

After operation. *a*, site of section of rib. × is on body of first dorsal vertebra.

puzzling and difficult of interpretation. Left side: Cervical rib, jointed to transverse process of seventh cervical vertebra; impossible to say how it ends anteriorly. Apparently about 1 in. of vertebral end left behind at operation. First dorsal rib not visible. Right side: Shadow of first dorsal rib well marked.

The anatomical conditions present: Left cervical rib about 2½ in. long, reached from costal element of transverse process of seventh cervical vertebra to rib below at its point of greatest convexity, articulating there by roundish diarthrosis of about 8 mm. diameter; joint formed not capable of much movement. Joint surfaces on lower, posterior aspect of upper and upper anterior aspect of lower rib. Rib ran down, forwards and out, slightly curved, with convexity outwards; cylindrical, with sharp inner and outer margins, except at lower end, where it is slightly flattened antero-posteriorly. Relations of rib: Inside, pleura and lung; outside, cords of brachial plexus; in front, scalenus anticus, subclavian artery, cords of brachial plexus; behind, neck muscles.

COMMENTARY ON CASE.

(1) Onset at puberty. ? due to alteration in relative rates of growth of parts concerned, causing artery and nerves to be more stretched.

(2) Onset coincident with beginning of violin lessons. Violin pressed exactly on site of swelling above clavicle.

(3) The scoliosis, convexity to left. Does its development determine the onset of symptoms?

(4) Interpretation of the skiagrams. Doubtful whether transverse process from which rib, portion of which was removed, originates belongs to seventh cervical or first dorsal vertebra. No shadow of first dorsal rib on left as on right. Therefore, is removed rib an abnormal, rudimentary left first dorsal rib with pleura and artery rising higher in neck than usual?

(5) The vascular symptoms. Subclavian artery pulsated before and at operation. It did not appear to be compressed between scalenus anticus and rib. Was absent pulse due to artery being so raised up as to be kinked and thrombosed behind clavicle? Position of vein agreed with absence of œdema.

(6) The nervous symptoms. All nerve-cords stretched over rib. Symptoms showed lowest cord particularly affected.

(7) The separate course taken by subclavian artery and vein is to be noted.

(8) Should rib have been removed right up to its vertebral end? Considerable retraction was used to get to point of section, and further removal would have necessitated a vertical skin incision. Artery appeared free; nerves may still be stretched.

Cervical Rib with Neuritic Symptoms; Operation with Successful Result.

By A. E. Naish, M.B.

E. C., aged 12. Shortly before Christmas, 1908, she competed for a swimming prize and had covered several lengths of the bath. Almost immediately afterwards she began to suffer pain in the right shoulder, passing down the outer and posterior aspects of the arm, and a few weeks later she noticed some loss of power in the right hand which prevented her writing. When first seen on March 9, 1909, the right hand was held in the *main en griffe* position, there was wasting and loss of power of the interossei, thenar and hypothenar muscles. There was also slight paresis, without obvious wasting, of the flexors of the wrist and fingers. There was no loss of sensation in the hand or fore-arm. A skiagram showed a seventh cervical rib present on both sides; but whereas on the left side it was only a short stump, on the right it was long and curved abruptly forwards and downwards. For the next seven weeks the arm was kept in a sling and the affected muscles were treated with massage and galvanism. The condition, however, gradually became worse.

On April 24 an operation was performed by Mr. Garrick Wilson, the rib with its periosteum being removed.

On May 11 there was some slight improvement in power of the intrinsic hand muscles, but there was widespread paresis of the muscles of the shoulder-girdle and arm. The rhomboid, serratus magnus, lower part of the trapezius, deltoid, biceps, triceps and extensors of the wrist were affected. By June 18 this paresis had almost entirely cleared up, there had been steady improvement in the power of the hand muscles, and she was able to write for a short time. By September she was able to write without tiring, and the appearance of her hand was quite normal. During the cold weather of the ensuing winter she again had some difficulty in holding her pen, but since then the power has completely returned, and she is now able to play the violin and do secretarial work for her father. The pain in her right arm gradually abated, but for a period of a few months she suffered from a similar pain in the left arm.

Case of Cervical Ribs; Improvement without Operation.

By James Galloway, M.D.

Mrs. B., aged 57, has for the last ten years felt discomfort, with pains in the arms and hands on exertion. These indefinite symptoms have increased during the last four years, especially in the left arm and hand. She sought medical advice on account of the increasing feelings of pain and weakness. Distinct wasting of the muscles of the left thenar eminence was noticeable, and to a less extent the other muscles of the left hand were affected. The presence of cervical ribs was diagnosed, but after consideration it was decided not to attempt their removal. The patient was in a position to diminish the amount of manual work she was in the habit of doing, and has had regular massage for both right and left upper extremities. The symptoms of pain have diminished—almost gone; she has more power in the hands and the wasting of muscles is less pronounced. It will be noted that the greater amount of the wasting is on the left side, but the skiagram indicates that the right cervical rib is more prominent than the left.

Case of Cervical Rib with Muscular Atrophy and Sensory Disturbance successfully Operated on.

By E. Farquhar Buzzard, M.D.

A. C., a dressmaker, aged 26. Came to the National Hospital in June, 1911, complaining of numbness and pain and wasting in the right hand during four months. The onset had been somewhat rapid. The numbness, usually confined to the first and second fingers, spread on exertion over the whole hand. The pain was worse when the hand was hanging down and after any exertion. On examination there was found wasting of the thenar eminence, slight loss of sensibility to cotton-wool over the first and second fingers, and no other signs of disease of the nervous system. A double cervical rib was revealed by skiagram. The right rib was removed by Mr. Sargent in July, 1911, and from that time there has been steady improvement in the condition of the hand. It is described now as being quite well.

Case of Bilateral Cervical Rib with Vasomotor Symptoms successfully Treated by Operation.

By E. Farquhar Buzzard, M.D.

F. S., aged 40, employed as a telegraphist. In November, 1907, began to complain of pain, redness, and swelling of the left forefinger near the inner side of the nail. This was incised by a doctor but no pus was found. In January, 1908, a similar swelling occurred on the palmar aspect of the terminal phalanx of the middle finger. From that time until seen at the National Hospital in March, 1908, there was constant pain in both fingers, with low temperature, a blue colour, and a variable amount of swelling. On examination the fingers of the left hand were cold and blue, and extremely hyperæsthetic. There was also slight wasting of the small muscles of the hand and of the flexors of the wrist. A long period of treatment by means of galvanic baths, &c., gave a certain amount of relief without definite cure, and in September, 1909, Mr. Sargent removed the left cervical rib.

In January, 1910, the left arm was still aching a good deal, but she was able to use the fingers better. She was also complaining of aching in the right arm. In January, 1911, the left arm was practically well, but the patient complained of pain in the whole of the right arm, and there was much cyanosis of the fingers of both hands. In April, 1911, Mr. Sargent removed the right cervical rib and since that time there has been steady improvement in the right arm and hand. In this case the influence of occupation and change of occupation have to be considered as well as that of operation.

Case of Cervical Rib giving rise to Pronounced Vascular Symptoms; Operation; Cure.

By H. J. Waring, F.R.C.S.

L. G., a woman, aged 35, has the following history: She had good health till July, 1910, when she noticed that the left hand had a decided tendency to feel cold and painful. Shortly after this the nails on the first and second fingers of the left hand turned black, and the fingers then began to go black at the tips, this being most marked in the case of the first and second fingers. The hand remained affected in this way till September of same year, when the patient was admitted to St. Bartholomew's Hospital.

When she came under observation the entire left hand and part of the forearm were distinctly colder than on the other side. The tips of the index- and middle-fingers and the nails were black and shrivelled. No pulsation could be detected in the radial or ulnar arteries, but there was slight pulsation in the brachial. There was no muscular atrophy or objective sensory disturbance. A hard, prominent swelling was palpable in the posterior triangle of the neck. Lying on the swelling was a hard cord which pulsated feebly, whilst internal to it there was marked pulsation. On the right side the radial pulse was perceptible, but it *increased in force* when the arm was raised, suggesting that in the dependent position the circulation was interfered with by a cervical rib on that side.

X-ray examination showed bilateral cervical ribs, the larger being on the left side.

Operation was performed on September 23, 1910, and the left cervical rib removed. The condition of the hand steadily improved from that date, though the gangrene resulted in a small amount of permanent injury to the tip of the index-finger. Pulsation did not return in the left radial artery till some weeks after operation.

Case of Bilateral Cervical Rib with Vascular Symptoms in the Right Upper Limb.

By Sidney Boyd, M.S.

L. C., aged 24, housemaid, came to hospital complaining of swelling in the right armpit and pain in the right arm of twelve months' duration. Both the swelling and the pain are worse after using the arm for any length of time, and the hand becomes blue. The pain is chiefly felt on the outer side of the shoulder and at the front of the elbow. On examination the whole limb, with the exception of the hand, was found to be swollen, the circumference of the upper arm and also of the forearm being increased ¾ in. as compared with similar measurements of the left. The hollow of the axilla is filled up with a soft, puffy swelling. The right radial pulse is distinctly smaller than the left. The cutaneous veins are not more obvious in this limb than in the other. There is no loss of power or muscular wasting, and sensation is perfect everywhere to all forms of stimulation. The subclavian artery feels as if it were pushed out slightly by some deeper resistant structure. There is nothing in the past history to suggest previous thrombosis of the axillary vein. The small scar on the front of the shoulder was caused by the excision of a subcutaneous lipoma in 1905.

A skiagram shows short bilateral cervical ribs.

Intrinsic Cancer of the Larynx after Laryngo-fissure; Case in which the Growth appears to have been completely removed by Endo-laryngeal Operation.

By Sir StClair Thomson, M.D.

The patient, A. P., aged 53, had been hoarse for eight months, when she was sent to me on October 19, 1912, by Dr. Shaw, of Great Yarmouth. The right vocal cord was replaced by reddish, cauliflower masses. This somewhat impeded the movement of the cord, but it was not in itself fixed. Both arytænoids were mobile; there were no glands, and the Wassermann reaction was negative. As the growth did not infiltrate the cord, and as it projected above it, I decided to remove a portion for microscopic examination. Using a large-sized duck-billed forceps of Morell Mackenzie, I seized as large a piece of the cord as possible, and, finding it very tough, I removed it by avulsion. The toughness and naked-eyed appearance were confirmatory of my suspicion of malignant disease, and the examination by Dr. d'Este Emery showed a typical epithelioma.

On November 28, 1912, anæsthesia was induced by Dr. Silk by means of intravenous infusion of ether and hedonal, administered through the internal saphena vein. The usual laryngo-fissure operation was carried out—no Hahn's tube was used, and the remains of the right cord, right ventricular band, and vocal process of the arytænoid were dissected up on their perichondrium, and removed *en masse*. The tracheotomy tube was removed before the patient left the table.

The patient would have been allowed up the next day, but she was still exceedingly drowsy from the hedonal. She was sitting up in forty-eight hours, and taking solid food. She returned to the country a fortnight after operation.

Towards the end of January, 1913, the patient began to show symptoms of what many would have taken to be a recurrence of the growth. But I had no anxiety myself, partly on account of the above negative report, but chiefly from experience with previous cases in which a re-growth during the first two months nearly always proves to be a simple granuloma. In this case it was removed through the mouth a fortnight ago, and was found to consist of simple granulation tissue, showing no epithelioma or sign of malignant disease in any part of the section.

The case is brought forward (*a*) to show how very favourable for operation is the pathological anatomy of epithelioma of the vocal cord; (*b*) to give support to those who claim to have effected a lasting cure of intrinsic cancer of the larynx by endo-laryngeal operation; and (*c*) to show that re-growth in early days after thyrotomy is not necessarily malignant.

It is not suggested that endo-laryngeal operation for cancer should ever be relied upon. This would be a dangerous doctrine, although it might have proved successful in the present instance.

Two Cases to illustrate the Advantages of Lateral Rhinotomy (Moure's Operation) in dealing with Malignant Growths of the Nose and Accessory Sinuses.

By Sir StClair Thomson, M.D.

The elder lady, aged 70, was operated on on July 4, 1910, when the right ethmoid and right maxillary sinus were found to be crowded with fleshy growths and of typical lymph-endothelioma. There is no trace of recurrence after an interval of two years and seven months.

The younger patient, aged 50, noticed a swelling of the left cheek since the beginning of August, 1912, and grumbling pain of a toothache character. A deep-seated swelling bulged into the left canine fossa. It was taken to be of a cystic character, and some semisolid gelatinous material was removed by another surgeon and discovered to be a glandular epithelioma (section shown). On October 3, 1912, lateral rhinotomy was carried out.

A similar operation was carried out in both cases. The inside of the nose was prepared with cocaine and adrenalin, chloroform was administered, and each posterior choana was plugged with a tethered sponge. Two incisions were started from the inner extremity of the eyebrow, one descending to the nasal orifice and the other curving outwards below the orbit. Both these incisions are carefully designed so as to correspond with the natural wrinkles in this neighbourhood. The triangular flap thus outlined is turned down; the periosteum is saved as much as possible, and the bone is exposed so that it can be divided with chisel and hammer in the three following lines: (*a*) Vertically between the two nasal bones, or to one side of the middle line; (*b*) from the upper part of this horizontally inwards to the orbit; and (*c*) from the lower border of the sinus pyriformis obliquely upwards and outwards into the orbit in front of the infra-orbital foramen. If this piece of bone is seized with forceps it will twist off with part of the lachrymal bone, generally exposing the lachrymal sac and lachrymal canal. This resection is varied according to the case. When, as in the present cases, the growth was almost entirely limited to ethmoid and antrum, the bridge of the nose is less interfered with, and more of the front wall of the antrum is removed. In any case a large opening is obtained, bringing into very immediate observance any neoplasm in this neighbourhood, particularly those of the ethmoid area and the antro-nasal wall, where, in fact, the majority of malignant growths originate. Extensions upwards into the infundibulum, or disease in the sphenoidal sinus, can be readily and directly inspected. Hæmorrhage is less when compared with such mutilating operations as the removal of the upper jaw. Bleeding is also more readily controlled, and enables the surgeon carefully to examine and remove growth in, and around, the antrum. In the second case a bottle is shown containing several ounces of removed growth.

There is no anxiety with regard either to the hæmorrhage or the administering of the anæsthetic. The incision is closed with a few horsehair stitches. No dressing is required, and the wound heals like a shaving cut.

It is very seldom that malignant disease of the antrum starts from the floor of that cavity. Excision of the alveolus is, therefore, an unscientific and clumsy way of approaching it. The external operations for malignant disease of the nose, called after various surgeons—such as Dupuytren, Langenbeck, Ollier, &c.—are all bloody, disfiguring, and ineffective when compared with lateral rhinotomy. This operation, or Rouge's operation, or some combination or modification of them, affords much better methods of satisfactory treatment.

In the second patient shown there is no trace of recurrence four months after operation. The lymphatics beneath the lower eyelid have remained congested longer than usual. There has been no neuralgia, although I had to expose and destroy the infra-orbital nerve. There is a slight amount of epiphora, although I took great care with the lachrymal canal, and it did not appear to be injured at the time.

In the elder lady there is no recurrence of disease two years and seven months after operation. There is a clear view into the ethmoid and sphenoidal cavities, and a large opening from the nose into the right antrum. There is not the slightest disfigurement; it is difficult to detect any scar, and the patient has the full benefit of her alveolus. Her only complaint is of numbness of the side of the face.

Nephritis with Ascites, Bilateral Hydrothorax and General Œdema in Secondary Syphilis (Result).

By F. PARKES WEBER, M.D.

THE patient, K. H., aged 22, a well-built and well-nourished young man, a German waiter in London, was admitted to the German Hospital on December 29, 1911. Four months before admission he had had a primary syphilitic chancre on the penis, and he had been treated by intra-gluteal mercurial injections. Ten days before admission the signs of nephritis had been noticed by the doctor who was treating him. No secondary syphilitic eruption had been observed. In the hospital, after admission, the patient felt fairly well, but he had slight ascites and œdema of the sacral region. The urine varied from 600 c.c. to 1,200 c.c. *per diem*, and was of high specific gravity (1024 to 1033) and very rich in albumin. By Esbach's tube the albumin was estimated at about 22 *per mille* (25 *per mille* on the day of admission); there was no sugar; microscopical examination of the centrifuge sediment showed the presence of hyaline tube-casts (containing also granules), but no real granular or epithelial casts, and no blood; there were a few epithelial and white cells. Reflexes, normal. Ophthalmoscopic examination showed nothing special. Condition of the teeth and gums, good. Pulse (on admission) about 84 per minute. The temperature chart showed slight rises, but not up to 100° F. The blood serum (January 1, 1912) gave a positive Wassermann reaction for syphilis (Lister Institute). Nothing abnormal in the thorax, or, by palpation, in the abdomen. The arterial blood-pressure was low; on admission the brachial systolic pressure was about 100 mm. Hg.; on January 18, only 88 mm. Hg. A striking feature of the case, as in typical severe cases of early syphilitic nephritis, was the very large amount of albumin in the urine. On January 7 the patient passed 1,100 c.c. of urine, containing 40 *per mille* albumin. On January 25 he passed 400 c.c. of urine with 48 *per mille* albumin. Later on, between February 8 and February 22, the urine averaged about 2,000 c.c. *per diem*, and contained from 4 to 12 *per mille* albumin. Blood examination (February 27, 1912): Hæmoglobin, 78 per cent.; red cells, 4,900,000, and white cells, 11,500, to the cubic millimetre of blood; nothing special by microscopical examination of stained films. In the second half of January, and for a long time afterwards, there was general dropsy with ascites and bilateral hydrothorax; the latter condition needed occasional paracentesis, especially on the left side. Blood examination (April 25, 1912): Hæmoglobin, 70 per cent.; red cells, 3,880,000, and white cells 5,600, in the cubic millimetre of blood. There was decided improvement in the patient's condition about April, though for a time he had a little pyrexia, of uncertain origin. In the second half of April it was noted that the urine (about 1,500 c.c. in twenty-four hours) contained about 10 *per mille* albumin. In July there was a little pyrexia again. The left pleura was tapped for the two last times in September, on which occasions respectively 1,550 c.c. and 550 c.c. of nearly clear fluid were withdrawn. By October 7 the patient was up most of the day. He looked well and had no ascites or general œdema, but there was still considerable dullness at the base of the left lung. The urine averaged about 2,300 c.c. *per diem*, containing about 4 to 6 *per mille* albumin. On November 16 it was noted that the urine for the past week averaged about 2,370 c.c. *per diem* (specific gravity about 1010), with only about 2 *per mille* of albumin. The systolic brachial blood-pressure was then 90 mm. Hg. No ocular complication was ever noted.

The patient was discharged on December 20, 1912. There was still a great deal of dullness at the base of the left lung, apparently due to thickened pleura. The urine then averaged about 2,500 c.c. *per diem* (specific gravity about 1011), with 2 *per mille* albumin. Heart not obviously hypertrophied. Pulse 80 to 96 per minute. Brachial systolic blood-pressure, 95 to 100 mm. Hg. No tube-casts were found when the urine was microscopically examined before he left the hospital. He is now employed as a waiter at a restaurant, and feels well in spite of long daily hours at work. There is considerable dullness and contraction of the lower left part of the thorax, and the urine (January 27, 1913), which is clear and of specific gravity 1011, contains 4½ *per mille* albumin; microscopical examination of the scanty centrifuge sediment shows a few red blood corpuscles and white cells, but no tube-casts. The blood serum, when tested again (Lister Institute) in December, 1912, gave a negative Wassermann's reaction for syphilis.

The treatment adopted at first was chiefly the ordinary treatment for acute nephritis. For a time either strophanthus or digitalis was given, with or without diuretin, or else diuretin by itself. For nearly all the time from the end of April to the end of December, 1912, he was given a tablespoonful of liquor ferri et ammonii acetatis, three times daily, after meals. It is not certain that a "salt-poor" diet did much good in this case. For some days when an ordinary amount of common salt was allowed (March, 1912) the patient passed less urine, with a higher percentage of albumin, than when he was having "salt-poor" diet.

Very little special anti-syphilitic treatment was attempted. A mixture containing perchloride of mercury and iodide of potassium was soon discontinued on account of diarrhœa. Two small intravenous injections of the original salvarsan (0·1 and 0·2 grm. respectively) were given in January, 1912.

The existence of a really syphilitic parenchymatous nephritis, occurring in the primary and secondary stages of the affection, was formerly much disputed; but recent observations, especially on the Continent, have made it certain that genuine cases of the kind do occur. I would compare such genuine cases to cases of secondary syphilitic affection of the liver, including several instances of fatal icterus gravis with acute hepatic atrophy, occurring during the secondary stage of syphilis, like the one which I brought before the Pathological Section of the Royal Society of Medicine in January, 1909.[1]

The present case resembled typical examples of severe secondary or early syphilitic nephritis in regard to the very large amount of albumin at first present in the urine. In January, 1912, the albumin reached 48 *per mille*, as measured by Esbach's tube; in a case of early syphilitic nephritis related by E. Hoffmann[2] it reached 70 to 85 *per mille*, and I believe still higher figures are on record. The urine in the present case was not examined, like the patient's blood serum was, for Wassermann's reaction. According to R. Bauer and others, very albuminous urine from syphilitic patients will give a positive Wassermann's reaction.[3] But it is doubtful whether a positive Wassermann's reaction from a patient's urine can be relied on as a diagnostic criterion of the presence of syphilis, still less so as a criterion that a nephritis from which a patient is suffering, even supposing him to be a syphilitic patient (with old or recent syphilis), is of genuinely syphilitic nature. The *Spirochæta pallida* has apparently been shown to be present in the urine of some cases of secondary syphilitic nephritis, and Vorpahl[4] claims to have lately succeeded in detecting this pathogenic organism in the urine from a case of tertiary syphilitic nephritis. Certain it is that typical cases of early syphilitic nephritis are not the result of mercurial treatment, for in some such cases no mercury had been employed before the onset of the albuminuria.

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[1] "Acute Hepatic Atrophy in Early or Secondary Syphilis," *Proc. Roy. Soc. Med.*, 1909, ii (Path. Sect.), p. 113. In that paper I gave references to all the literature (that I at the time knew of) bearing on the subject.

[2] E. Hoffmann, "Ueber Nephritis syphilitica acuta praecox mit enormer Albuminurie," *Berl. klin. Wochenschr.*, 1902, xxxix, pp. 113, 166.

[3] *See* R. Bauer, "Die klinisch-serologische Diagnose der luetischen Nierenerkrankungen," *Wien. klin. Wochenschr.*, 1911, xxiv, p. 1458.

[4] *Münch. med. Wochenschr.*, 1912, lix, p. 2811.

On the whole, I think that the present case was really one of secondary or early syphilitic nephritis and not merely one of nephritis in the course of syphilis. As the nephritis developed so soon after the primary affection, and as no ordinary secondary manifestations of syphilis were observed, the renal affection in this case ought perhaps to be called "early" syphilitic nephritis ("Nephritis syphilitica præcox") rather than "secondary" syphilitic nephritis. A question which suggests itself, but which I shall not attempt to answer, is whether the patient's continued low blood-pressure was in any way responsible for the gravity and chronicity of the nephritis.

Intermittent Claudication of Lower Extremities from Quiescent Non-syphilitic Arteritis Obliterans.

By F. Parkes Weber, M.D.

The patient, B. R., is a Russian Jew, aged 44, who has been seventeen years in England. From the age of about 12 he has been engaged in the cigarette trade, at first in a cigarette factory in Russia; for the last thirteen years he has worked as a tobacconist on his own account. His general health has been, on the whole, good and he denies intemperance in alcohol or having had any kind of venereal disease. His blood serum (January 16, 1913) gives a negative Wassermann's reaction for syphilis. For the last four and a half years he has been troubled more or less from "intermittent claudication" in the left lower extremity. After walking for about five minutes, he has to rest on account of pain or a cramp-like feeling, chiefly in the calf muscles. When this pain comes on, the lower part of the affected leg is cold and pale. Then, after a short pause of about two minutes, he can walk on again for another five minutes, and so on. To a lesser extent he has the same trouble in the right lower extremity, but, of course, the symptom coming on first in the left leg obliges him to pause in his walk and prevents the full onset of the pain in the right leg. The left foot seems to be usually somewhat colder and paler than the right foot, but although the intermittent claudication is of four and a half years' duration, the objective signs can easily be altogether overlooked. The foot does not become red or cyanosed, like it does in most chronic cases, when he allows it to hang down. Pulsation cannot be made out in the dorsalis pedis artery of either foot, but is present in the femoral artery at both groins, as it also is in the posterior tibial artery at the right ankle.

No pulsation can be detected in any of the arteries of the left foot. Dr. N. S. Finzi found no skiagraphic sign (January, 1913) of any arterial calcification in the left leg below the knee. No disease in the thoracic and abdominal viscera or in the blood-vessels of the upper extremities has been discovered. Brachial systolic blood-pressure, 115 mm. Hg. A blood examination (January, 1913) gives: Red cells, 4,800,000 to the cubic millimetre of blood; white cells, 14,100; hæmoglobin, 80 per cent. The urine is free from albumin and sugar. Ophthalmoscopic examination shows nothing abnormal. There is no obvious muscular wasting in either lower extremity. The knee-jerks are excessive (as in some neurasthenic conditions). The plantar reflexes are normal, of the flexor type, in both feet. The patient is now, apparently, very moderate in his use of tobacco—he still smokes a few cigarettes—but on account of his business he will not give up smoking altogether. After treatment at the German Hospital in 1908, chiefly by rest in bed and iodipin, he was able to walk with less frequent pauses, but the necessary pauses have become more frequent again.

The case was shown before the Clinical Section on January 14, 1910,[1] and it is because of the relatively favourable course of the affection, the long quiescence in a relatively early stage of the disease, and because of possible difficulty in the diagnosis of such cases, that the patient is now brought forward again.

Thoracic Aneurysm in a Woman.

By F. Parkes Weber, M.D.

The patient, W. R., a healthy-looking, well-built woman, aged 34, was for some time under treatment at the German Hospital a year ago. At that time there was slight bulging, together with expansile pulsation and dullness to percussion, over a somewhat circular area on the upper portion of the right side of the front of the chest, extending from the first to the third rib (inclusive) and from the middle of the sternum outwards for about 7 cm. Röntgen-ray examinations showed a pulsating shadow, corresponding to that area, projecting from the aorta to the right of the sternum. Over that area loud systolic and diastolic murmurs could be made out. The cardiac apex beat was felt slightly to the left of the nipple line, but there was no sign of enlargement of the heart upwards or to the right. Systolic and diastolic aortic murmurs were heard over the base of the heart, and there was a presystolic murmur, with a "thumping" first sound, at the apex. The radial pulse was of the sudden and collapsing kind characteristic of incompetence of the aortic valve; it was slightly stronger at the left than at the right wrist. The pupils were equal and reacted normally to light and accommodation. There were no signs of any other disease; no paralysis of vocal cords; no dysphagia; no "tracheal tugging." Brachial systolic blood-pressure, 125 mm. Hg. The urine was free from albumin and sugar.

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[1] *Proceedings*, 1910, iii, p. 98.

The signs clearly pointed to the presence of an aneurysm of the ascending thoracic aorta, together with disease of the aortic and mitral valves. The history was that in November, 1910, she had commenced to suffer from pains on the right side of the chest, but these disappeared whilst she was in the German Hospital (December, 1911, to March, 1912), where she was treated by rest in bed and restriction of fluids. She was likewise given potassium iodide and, for a time, calcium chloride with sodium sulphate, and she also underwent a course of mercurial inunction. Since leaving the hospital (March, 1912) the patient's state has remained much the same, but the bulging over the upper right chest front has become rather more obvious, and the apex beat is now perhaps a little farther to the left. The absence of thrill over the aneurysm and the fact that by Röntgen-ray examination no pulsation can now be detected in the shadow to the right of the aortic arch, both suggest the presence of a good deal of clot in the aneurysmal sac.

Dr. Weber thinks that the rarity of thoracic aneurysm in women is over-estimated, probably as a result of bygone statistics derived chiefly from military and similar official sources. He thinks that at present amongst the poorer civilian classes (in which the women do hard household or other work) thoracic aneurysm is not very much rarer amongst females than males. In the present case there is no history of traumatism or of syphilis, but the blood serum has twice given a positive Wassermann reaction for syphilis (February 9, 1912, and February 7, 1913). The patient, who has been married thirteen years, has had no children and no miscarriages. Four years ago she is said to have had muscular rheumatism.

Paralysis of the Serratus Magnus.

By E. C. HUGHES, M.C.

H. L., aged 33, a police-constable, was assaulted by five men on December 23. He remembers being thrown against a gate, and striking his right shoulder. The following day his shoulder was stiff, and the next day began to ache. He is now unable to perform any overhead actions. When standing with his arms by his side, the inferior angle of the right scapula is somewhat prominent. This prominence increases and the inferior angle approaches the mid-line as the deltoid is thrown into action. The arm can be raised almost to the level of the shoulder. When the arm is passively extended above the shoulder, the vertebral border of the scapula overhangs the spines of the vertebræ.

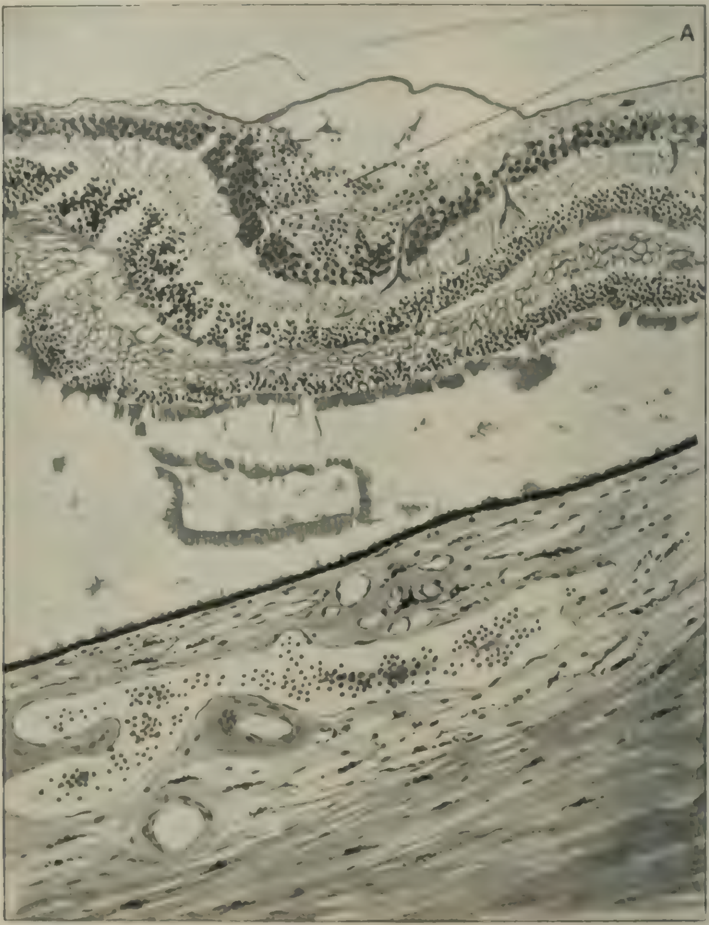
Case of Secondary Hæmorrhages in the Retina in Secondary Anæmia.

By W. HALE WHITE, M.D.

RETINAL hæmorrhages have been observed in patients suffering from profound secondary anæmia, yet they are probably very rare, although much oftener seen in those suffering from pernicious anæmia. As I was fortunate enough to obtain a histological section of the retina of a patient suffering from profound secondary anæmia, I thought the following case perhaps worthy of record:—

E. B., aged 43, was admitted into Guy's Hospital on May 11, 1912, for extreme weakness. She was very wasted, and of a grey, anæmic tint, so that she looked exactly like a painting of a corpse. A tumour about the size of a small hen's egg could be felt in the region of the cæcum. Red cells, 2,200,000; white cells, 17,800; hæmoglobin, 18 per cent. A second count, two days later, showed red cells, 2,498,000; white cells, 14,000; hæmoglobin, 17 per cent. On both occasions the increase of the white cells was due to an excess of lymphocytes.

A diagnosis of malignant disease of the cæcum was made. There were no other points of special interest except the condition of the retina, in which several minute hæmorrhages could be seen; they were quite small, but of various shapes and sizes, but mostly little round dots; one or two were flame-shaped. They could be seen in both eyes, but they were more numerous in the right eye than the left. After death the tumour felt during life was found to be a cancer of the cæcum. There were no secondary deposits anywhere. Several sections of the retina were cut, and the one which is under the microscope shows several minute subhyaloid hæmorrhages and some œdema of the retina. The accompanying figure shows a well-marked subhyaloid hæmorrhage. None of the sections happened to show any hæmorrhages



The figure shows a well-marked subhyaloid hæmorrhage at **A**. Many of the blood corpuscles have dropped out, but the pressing apart of the hyaloid membrane and the retina is well seen.

other than subhyaloid, but during life a few flame-shaped hæmorrhages were seen. The patient died. At the post-mortem examination a cancer of the cæcum was found; there were no secondary deposits. All the other viscera were normal. The kidneys were healthy.

Clinical Section.

March 14, 1913.

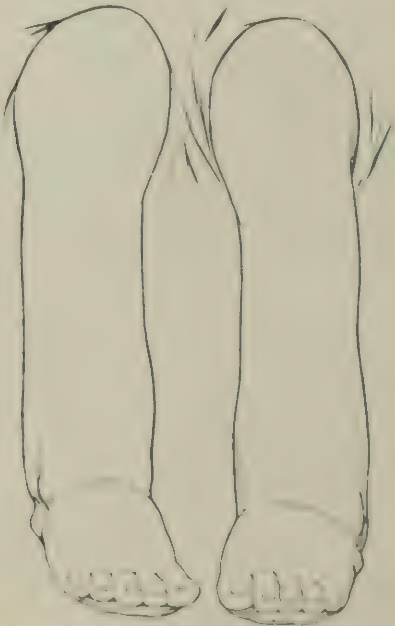
Dr. A. E. Garrod, F.R.S., Vice-President of the Section, in the Chair.

"Baggy" Subcutaneous Fat, simulating Symmetrical Œdema of the Legs. Disorder of Internal Secretions.

By F. Parkes Weber, M.D.

The patient, M. S., is a woman, aged 40, who looks much older than she really is. She has been married twelve years, but has had no children and was never pregnant. Menstruation ceased six years ago, and for the last six years she has been ailing, chiefly from bronchitis. She likewise has mitral stenosis and irregularity of the cardiac action. Brachial systolic blood-pressure, 120 mm. Hg. By abdominal palpation no enlargement of the liver or spleen nor any other visceral abnormality can be detected. Blood examination (March, 1913): Hæmoglobin, 70 per cent.; red cells, 4,640,000, and white cells, 6,500, to the cubic millimetre of blood. The urine (specific gravity, 1020) is free from albumin and sugar. There is some enuresis, probably connected with the coughing. The thyroid gland cannot be distinctly seen or felt. By Röntgen-ray examination the pituitary fossa appears to be very small. Ophthalmoscopic examination shows nothing abnormal in either eye. There is no history of rheumatism, and until six years ago the patient seems to have enjoyed good health. There is chronic swelling of the subcutaneous tissue of the legs below the knees of one or two years' duration. This is symmetrical and in both legs tends to "bag" above the ankles (*see* illustration). It either does not "pit" at all, or it (sometimes) "pits" slightly on pressure, and it feels as if it were due to accumulation of loose subcutaneous fat. I believe that in similar cases the swelling has been supposed (in France) to be due to a chronic œdematous degenerative process in the subcutaneous fat cells. Similar cases may have been described as "pseudo-œdematous" or even as "pseudo-lipomatous" conditions. In the present patient there is a less marked and more elastic "puffiness" of the subcutaneous tissue of the forearms (symmetrical) about the wrists. Skiagrams of the lower extremities show no bony abnormality.

I have no doubt that the "pseudo-œdema" in the present and similar cases is, like the elastic symmetrical swelling of the subcutaneous tissue of the fingers[1] and hands, sometimes met with in younger women, in some way connected with a disorder of internal secretions, especially of the thyroid gland and ovaries. The condition is to be distinguished from the true chronic œdema of the lower extremities of doubtful origin (including some cases which have been classed as "rheumatic œdema," "gouty œdema," or "arthritic œdema") some-



To show the "baggy" subcutaneous fat above the ankles.

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[1] *See* illustration of Dr. Weber's case of chronic symmetrical swelling of the fingers in a young woman, aged 20. *Proc. Roy. Soc. Med.* (Clin. Sect.), 1909, ii, p. 126.

times met with in men past middle life independently of kidney disease or circulatory failure.[1]

Thyroid treatment has been tried in the present case, but only for a short time and with indefinite result. In a similar case under my care in 1905 thyroid treatment seemed to have no obvious (at least no rapid) effect on the swelling in the legs. The patient was an unmarried woman, aged 50, in whom the menopause had occurred three years previously. In that case the feet did not at all share in the swelling of the legs, which commenced below the knees and terminated by a fold just above the malleoli; this fold was not removed even by prolonged rest in bed. Sometimes there was slight "pitting" on pressure. There was no anæmia or cardiac or renal disease, and nothing abnormal was discovered in the pelvis or abdomen. Brachial systolic blood-pressure, 135 mm. Hg.

DISCUSSION.

Dr. GALLOWAY remarked that the case recalled to his mind a patient he had shown some ten or twelve years ago in whom a condition of œdema somewhat resembling Dr. Weber's patient had developed in association with Graves's disease. Dr. Galloway's patient was then a woman aged about 45, and had been a nurse. She came suffering from a very noticeable chronic firm œdema, affecting both the lower extremities and extending well up the thighs. It was most marked in both legs, the skin showing a deep fold at the ankle. The œdema was very firm and the skin was apt to suffer from superficial erythematous or even inflammatory attacks. The result was that over the lower portion of the leg the skin had assumed an elephantoid appearance. No local cause affecting the veins or lymphatics in the pelvis or the thigh could be found to account for this state, but it was noteworthy that for some time before the development of this chronic œdema the patient had developed symptoms of Graves's disease, and when she came under observation these symptoms, though not excessive, were quite characteristic. She showed enlargement of the thyroid, proptosis, tachycardia, and tremor. This patient was still under Dr. Galloway's observation, and in the process of years and as the result of treatment such as massage, the firm œdema of the lower extremities had almost gone. The symptoms of Graves's disease had also much diminished. With very little excitement, however, the pulse would increase in frequency, slight proptosis would occur, and the thyroid was still enlarged. Dr. Galloway said he had always considered in this case that the elephantoid condition of the extremities was associated with hyperthyroidism, and it might be suggested that in Dr. Weber's case also the condition of the subcutaneous tissue was due to an abnormality of the glands of internal secretion.

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[1] *See* F. P. Weber, "Chronic Œdema due to Local Degenerative Changes," *Med. Press*, Lond., 1909, cxxxviii, p. 243. Perhaps the condition of the eyelids known as "ptosis adiposa" or "blepharochalasis," when it occurs as a degenerative change in elderly persons, is allied to the fatty swelling in the lower extremities in the present case rather than the true chronic œdema of the lower extremities which I specially referred to in the above article in 1909.

Dr. PARKES WEBER, in reply to the Chairman, said that there seemed good reason for saying that the pituitary fossa was small. The first skiagram taken had been rejected because it was taken in a somewhat slanting direction. He had suggested the use of two little leaden plugs, one to be placed in each external auditory meatus before a skiagram of the base of the skull was taken. By that means it was easy to see whether a given skiagram had been taken in a slanting direction or not. He thought that the present case was an example of a group in which the patients were women at the period of the menopause or beyond it. In some cases the menopause was premature, and in some cases artifically induced by surgical operation or otherwise. Amongst the symptoms noticed in this group of cases in question was the occurrence of a puffy condition of the subcutaneous tissues in the lower extremities, best marked just above the ankles, but not due to ordinary œdema. In some cases also a similar puffy condition might, as in the present case, be likewise observed in the upper extremities, near the wrists. The condition seemed to be due to an abnormality in the subcutaneous fat, in some way connected with ovarian function. Possibly failure of the ovaries might constitute only the final determining factor in inducing a symptom-complex in reality due to a "polyglandular" insufficiency. Younger women might have a minor form of the same subcutaneous swelling, with less noticeable "bagginess" about the ankles.

Hypoplasia of the Right Limbs, of Cerebral Origin.

By F. PARKES WEBER, M.D.

THE patient, A. W., is a rather tall and thin young woman, aged 20. Her right upper and lower extremities are slightly shorter and decidedly weaker and smaller in regard to muscular development than her corresponding left limbs. The difference is least marked in the legs below the knees, the calf muscles of the right leg looking so much smaller than those of the left leg, as at first sight to suggest a result of old infantile paralysis. There is, however, no history of an attack of infantile paralysis or of any kind of paralysis whatever. Her right arm and leg have always been smaller than her left arm and leg as long as she can remember, and her mother noticed a difference in size between the two legs as soon as she commenced to walk, which was at about the age of 3. She is much stronger in the left hand than in the right, and she is left-handed, having always preferred to use her left hand for any work, though at school she was taught (with difficulty) to use her right hand for writing.

The right half of the thorax appears to be slightly smaller than the left half, but there is no obvious difference in development between the gluteal muscles on the two sides or between the two halves of the abdomen. The right shoulder muscles are decidedly smaller than the left. There is considerable facial asymmetry, but it is not easy to say which side of the face is the better developed (*see* fig. 1). The left eyelids are generally wider apart than the right eyelids, but when she shows her teeth the right side of the mouth is opened more than the left. She has a flat-foot on the right side with hallux extensus, or habitual over-extension (bending backwards) of the right great toe. An orthopædic operation was performed on that foot five years ago—namely, an attempt to produce arthrodesis—but with imperfect result.

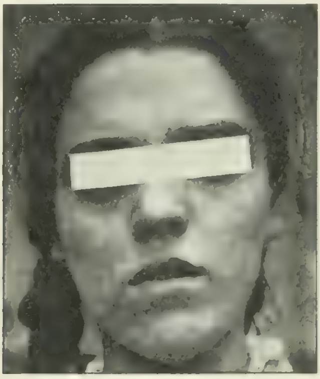

FIG. 1.

To show the facial asymmetry. In spite of obvious asymmetry it is not easy to decide which side is the better developed of the two.

The triceps and supinator reflexes are normal on both sides. The knee-jerks are both excessive, but the right one is specially exaggerated and is followed by clonus (the "trepidation type" of knee-jerk), and can be obtained by tapping over the periosteum of the upper part of the tibia as readily as over the patellar tendon itself. The plantar reflex cannot be obtained or is indefinite on the left side, but is active and of decided extensor type (Babinski's sign) on the right side. In regard to sensation there is hypo-æsthesia in the whole of the right upper extremity and in the right lower extremity below the knee. In front of a fire her right leg gets hot, she says, more readily than her left leg, and in cold weather her right leg more readily becomes cold and blue. She says that any scratches or cuts "fester" more readily if they are on her right hand or foot than if they are on her left hand or foot.

Measurements of the upper extremities: The length, with arm to the side, from the greater tuberosity of the humerus to the end of the middle finger, is 68·5 cm. on the right side, 74 cm. on the left side. The circumference at the middle of the upper arm is 20·5 cm. on the right side, 22·8 cm. on the left side.

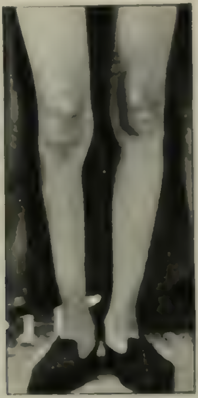

Fig. 2.

To show the smaller size of the muscles of the right leg.

Measurements of the lower extremities: The length from the anterior superior iliac spine to the tip of the external malleolus is 85⅓ cm. on the right side, 88⅓ cm. on the left side; to the tip of the internal malleolus it is 84⅓ cm. on the right side, 87½ cm. on the left side. The circumference of the thigh (15 cm. above the upper border of the patella) is 36 cm. on the right side, 37½ cm. on the left side. The circumference of the calf (15 cm. below the lower border of the patella) is 23 cm. on the right side, 28 cm. on the left side (*see* fig. 2).

The right half of the thorax at the nipple level measures 37 cm.; the left half measures 37·3 cm. The dynamometer grasp with the right hand is 16, with the left hand it is 30. By Röntgen-ray examination (Dr. N. S. Finzi) the head and hands do not appear to be abnormal

There is no decided difference between the hands in regard to the size of the bones, but the bones of the right foot are smaller than those of the left foot (*see* fig. 3).

In view of the difference in reflexes between the two lower extremities, especially the presence of a decided Babinski's sign on the right side, one must suppose that the right limbs, and not the left ones, are the abnormal ones—namely, that the condition is one of hypoplasia (or hypotrophy) of the right limbs and not of hyperplasia (hypertrophy) of the left limbs. One must further suppose that the hypoplasia of the right limbs, which especially affects the muscles, is of cerebral

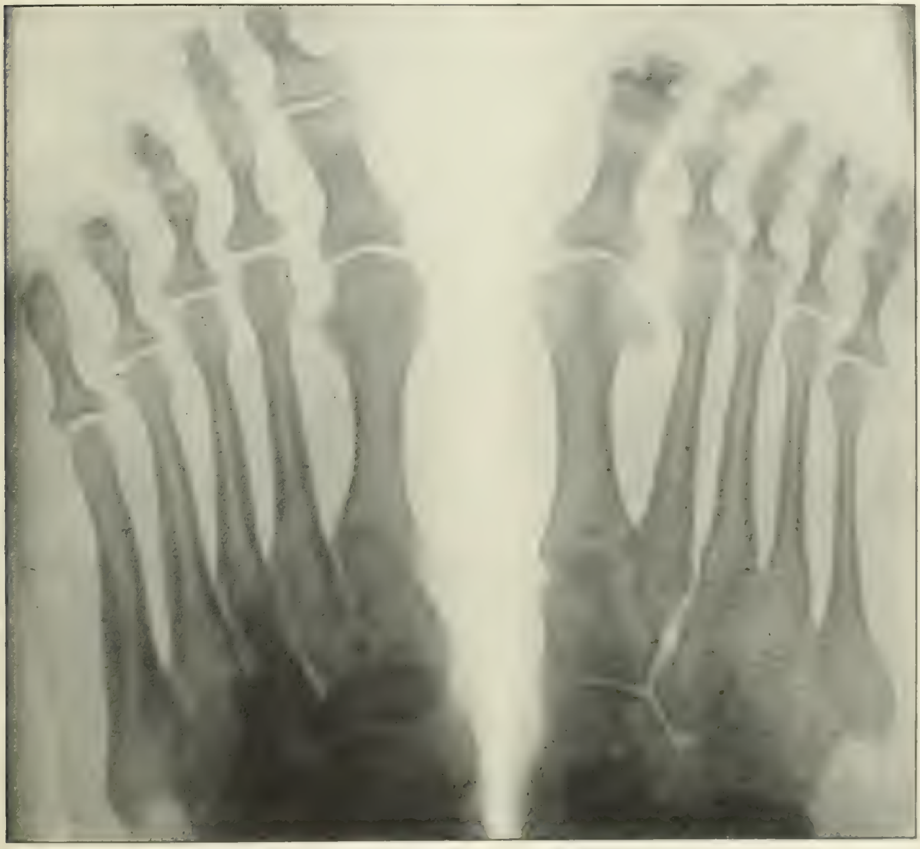

Fig. 3.

Skiagram of the feet, showing difference in size of bones. The apparent smallness of the terminal phalanx of the right big toe is, however, mainly due to over-extension.

origin, though there is no history of infantile hemiplegia of any kind. Disease or injury on one side of the brain in very early life may admittedly lead to diminished growth of the limbs on the opposite side of the body, and some pathological condition of the left side of the brain must be assumed to have been the cause of the weakness, the deficient growth, and the abnormal reflexes in the right limbs in the present case.[1]

In addition to what has been stated, the patient suffers from extreme chronic constipation and great enteroptosis. There is no pharyngeal reflex. The heart and lungs appear to be healthy, but the pulse is usually abnormally frequent. The general development of her body appears to be fairly good. Menstruation commenced at the age of 12 and is regular. There are no urinary troubles, and the urine shows nothing abnormal. Brachial systolic blood-pressure, 125 mm. Hg. In the hospital the patient has had occasional attacks, lasting half an hour or so, of "paroxysmal tachypnœa" (or "polypnœa"), if I may use the term employed by J. Pal, of Vienna, and S. West, of London; the respirations during the attacks become as frequent as 48 or 56 in the minute, but the patient, though looking excited, does not seem really ill. These attacks are doubtless functional (hysterical) in nature, and commence with a feeling of coldness and shivering. Previously she seems to have had occasional "fainting attacks," commencing in the same way, but accompanied, she says, by loss of consciousness, though not accompanied by biting her tongue or by the passage of urine or fæces. The patient's father was certainly addicted to alcohol before she was born. She has no sisters and only one brother, who is older than herself, and said to be healthy in every way.

The present case is of some interest as an example of partial right-sided hypoplasia (or hypotrophy) in a left-handed individual. E. Stier, of Berlin, who has made a special study of cases of hemiatrophy (which term, I presume, is generally used to include cases of congenital or developmental hemi-hypoplasia or hemi-hypotrophy) and hemi-hypertrophy, concludes that congenital hemi-hypertrophy or congenital over-development occurs in left-handed persons generally on the left side and in right-handed persons mostly on the right side; vice versa, I gather, in regard to hemiatrophy.[2]

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[1] The Babinski's sign may, however, be partly connected with the ankle-joint condition, for ankylosis of a knee-joint sometimes causes an alteration of the plantar reflex on the affected side.

[2] E. Stier, "Ueber Hemiatrophie und Hemihypertrophie, nebst einigen Bemerkungen über die laterale Lokalisation," *Deut. Zeitschr. f. Nervenheilk.*, Leipzig, 1912, xliv, pp. 21-64.

A question which arises in the present and some other cases of left-handedness is whether the left-handedness is merely a result of the weakness of the right limbs and of the cerebral condition on the left side of the brain, which has given rise to the weakness and hypoplasia in the right limbs; or whether the hypoplasia of the limbs is to be regarded as a pathological developmental process which has grafted itself on to a congenitally defective part of the body, the left-handedness being an expression of a congenitally defective condition of the left side of the brain (which may be regarded as a *locus minoris resistentiæ*).

Cases of unilateral hypoplasia of limbs due to cerebral lesions occurring before birth (cases of so-called "congenital hemiatrophy") or during childhood (before the development of the body is complete) must of course be distinguished from cases of more or less complete unilateral hypoplasia of spinal origin, due to infantile paralysis. They must also be separated from the more correctly termed cases of hemiatrophy (of the face and body or of the face only) in which the atrophy sometimes occurs after full development of the body. These latter I would divide into three provisional groups: (1) Cases of facial hemiatrophy due to a kind of local sclerodermia, in which the facial hemiatrophy constitutes part of the sclerodermatous process; (2) cases of hemiatrophy associated with, but not clearly due to, patches of morphœa or sclerodermia; (3) facial hemiatrophy or hemiatrophy of the whole of one side of the body, not due to, or accompanied by, any kind of sclerodermia. Cases of hemiatrophy and other local atrophy may likewise be divided into groups according to the depth of the atrophic process, whether only the fat and subcutaneous tissue are affected, or whether the subjacent muscles or the muscles and even the bones share in the atrophy.

Arthritis with Baker's Cysts.

By W. Essex Wynter, M.D.

C. L., aged 58, formerly a marine, exhibits cystic developments in connexion with three joints. Three years ago pain, stiffness and enlargement occurred after a twist of the left knee. The patella is enlarged and there is considerable thickening of the synovial membrane. Fluctuation can be elicited across the front of the joint, which shows limitation of movement, especially when the weight of the body is put upon it in walking. There are extensive cystic developments on the outer and posterior aspects which communicate with the joint. The external condyle of the femur is irregular in outline as viewed by X-rays. The knee and pupil reflexes are present. There are no nervous symptoms, but twice a trace of albumin has been found in the urine. About a year ago a swelling occurred in the left wrist after strain in digging. This has the appearance of a compound ganglion of the flexor sheath and there is also limitation of movement, extending to the little finger. Fluctuation can be appreciated between the palm and wrist. X-rays disclose thinning of the bones of the hand.

There is some wasting of the interosseous muscles, and slight ulnar deviation of the fingers. Six months ago pain and swelling occurred in the right elbow with limitation of movement, so that the hand cannot be carried to the mouth. The olecranon appears to be enlarged and there are extensive cystic swellings on the radial and ulnar side, which communicate through the joint. X-rays show considerable changes in the ulna with much thickening in front of the elbow.

Though these cystic developments in connexion with joints have been described in connexion with rheumatoid arthritis, this case shows none of the anaemia or polyarthritis commonly seen and the small joints are not affected. The character of the lesions does not resemble Charcot's joints, and there are no nervous symptoms. Wassermann's reaction is positive.

DISCUSSION.

The CHAIRMAN (Dr. A. E. Garrod, F.R.S.) expressed his belief that Dr. Wynter's case was a member of a well-defined group of infective lesions of joints, characterized by the multiple fluid swellings around the affected joints. In his experience these cases had proved very refractory to treatment, but had run a mild course without producing serious crippling. In the present case there appeared to be loose bodies in the elbow-joints. He did not think that these swellings were of the same nature as the large cysts described by the late Mr. Morrant Baker, in connexion with hip-joints, the seats of osteo-arthritic changes.

Dr. F. PARKES WEBER thought that such cases of multiple peri-articular cyst formation needed working out again in reference to their possible relation with syphilis. When they were originally studied, the connexion with syphilis had not been sufficiently considered. At that time, moreover, the connexion of syphilis with tabes dorsalis, general paralysis of the insane, Argyll-Robertson pupils, aortic aneurysms, &c., was not understood, and the Wassermann reaction for syphilis had, of course, not been discovered. Evidence of syphilis was, therefore, in many cases at that time more difficult to obtain than in similar cases nowadays. It had been pointed out to him that there were melon-seed bodies in a cyst near the patient's right elbow, and he would like to hear a satisfactory explanation of their presence. Possibly their presence in some such cases was a result of hæmorrhage into the synovial fluid of the cysts, giving rise to the formation of small fibrin lumps.

Dr. BATTY SHAW, referring to the question of puncturing the joint, so as to remove the fluid, said he had had this done several times, but always with a negative result. One must do more than remove and examine the fluid from such a joint; and even more than remove and examine the melon-seed bodies. It was necessary that the joint should be thoroughly cleaned out and the synovial membrane removed. On staining in situ, the organisms would be found in this toxin. He believed it would be agreed that bacteriological examination of the fluid was generally negative, but of the membranes, positive.

Dr. ESSEX WYNTER replied that he could not throw any further light on the case, but he would have some of the fluid withdrawn, to see if that would give any clue. There was a history that the patient had had gonorrhœa, but long before the onset of the joint trouble. Surgical operation he considered would have to be too extensive to be considered.

Progressive Muscular Atrophy associated with Primary Muscular Dystrophy in the Second Generation.

By ARTHUR F. HERTZ, M.D., and W. JOHNSON, M.D.

THE father, G. H., aged 41, a butcher, came under observation six months ago, complaining of weakness, chiefly in the left hand. He was a strongly developed man and had formerly great muscular power, being capable of carrying "a quarter of beef." His occupation necessitated this daily overuse of his muscles. He first noticed weakness of the left hand about twelve months ago, and he ascribed it to an injury to the left index-finger from a chopper. The whole of the left arm gradually became weak, and within six months of the onset the right hand and later the arm became affected. The legs also began to drag about this time, the left being the first involved. About a year after the onset, rather rapid wasting occurred in the hands, the weakness having previously been associated with only slight wasting. This continued for about four months, but no further wasting has occurred during the last two months. The legs have recently become rapidly weaker, and he now frequently falls in the street. Fibrillary tremor has been marked in the arm, leg and shoulder muscles. The thenar and hypothenar eminences are markedly wasted in both hands, and the interossei to a less extent. The opponens movement is almost absent, while all the muscles of the forearm and arm are deficient in power: they are also flabby and smaller than they were three or four years ago. The calf muscles do not appear to be wasted, but some weakness is present. The thigh muscles, especially those on the inner side, are definitely wasted. The abdominal and back muscles both show weakness. The strabismus which the patient exhibits has been present since birth. At times there is a tendency to nystagmus. The orbiculares palpebrarum are weak, and there is some tremor of the tongue. Examination of the eyes shows nothing abnormal. It is difficult to obtain any trace of tendon reflexes, with the exception of the triceps-jerk. Cremasteric and abdominal reflexes are not obtained; the plantar reflexes are flexor. There has never been any bladder or rectal trouble. Cutaneous sensation is normal. The Wassermann reaction of the blood is negative.

The case appears to be an example of the pure progressive muscular atrophy type of motor neuron disease. The rapid progress of the symptoms, however, is an unusual feature, and it is a remarkable fact that some improvement in general muscular power has occurred during the last few weeks.

S. H., aged 17, the eldest son of G. H., was a well-developed child at birth, and normal in every way up to the age of 8. It was then noticed that he walked with his back arched, and with his abdomen prominent. Soon his legs began to drag and he used to fall about a good deal. Within three years of the onset he was quite unable to walk. In raising himself from the floor "he climbed up his legs," and his power of walking upstairs was very early impaired. His face now has the myopathic character, but there is no marked facial weakness. There is much wasting of the scapular, humeral and thigh muscles, with a corresponding amount of weakness. The hamstrings are much contracted. The calf muscles are large and firm in comparison with the other muscles. The hand muscles are not wasted and the patient is able to make good use of them. The knee-jerks are absent, but the ankle-jerks are present. No arm-jerks could be obtained. He is clearly suffering from the pseudo-hypertrophic form of primary muscular dystrophy.

F. H., aged 5, another son of G. H., appears to exhibit a very early stage of pseudo-hypertrophic paralysis, his condition being similar to what was noticed at the onset of his brother's illness. He has the same tendency to arch his back and protrude his abdomen when walking. His calves are well developed and unusually firm. There is, however, no wasting present. In rising from the floor he shows a distinct tendency to climb up himself. His condition is still too early for a definite diagnosis to be made, but the association with the other cases makes it highly probable that it is an early stage of a primary muscular dystrophy.

There are two other children in the family—one a boy aged 15, and the other a girl aged 7, both of whom are perfectly normal. Inquiry into the history of relatives on both sides led to negative results, with the exception of a boy, J. H., aged 3, the son of the first patient's brother. For the last six months he has been noticed to be falling about. No wasting is present, but there is a tendency to lordosis of the spine, and to prominence of the abdomen when he walks, and the calves are unusually well developed and hard. It is possible that this boy may also be suffering from the earliest stage of a primary muscular dystrophy.

DISCUSSION.

Dr. GALLOWAY considered that it was much more probable that these patients, the father, the two sons and possibly the nephew, all suffered from the same disease—namely, muscular dystrophy. From what was known of the family inheritance in cases of muscular dystrophy, it was much more easy to associate the occurrence of even different types of the disease in successive generations than to defend the hypothesis that a member of one generation should develop progressive muscular atrophy, while members of the next generation developed primary muscular dystrophy. But on examining the father he could not avoid the conclusion that the atrophy of muscles in his case was of the type of a muscular dystrophy rather than of progressive muscular atrophy. There was not only the wasting of muscles of the hands, but the weakness of the muscles of the back, the buttocks, and of the shoulder girdle. He would like to draw attention especially to the fact of the very suggestive way in which the patient heaved himself up on attempting to stand, a movement characteristic of a patient suffering from muscular dystrophy affecting the trunk. It was true that it was unusual to find muscular dystrophy commencing so late in life as in the case of the father of this family, but this was by no means an insuperable objection to the diagnosis. Some time ago he had the opportunity of bringing before the Clinical Section[1] a case of undoubted muscular dystrophy, partly of the pseudo-hypertrophic variety and partly of the facio-scapulo-humeral type. In the case of that patient the symptoms did not develop till about the age of 21. Previous to this weakness, which first affected the arms and hands, the patient had been a good athlete, and an instructor in gymnastics in various schools. It seemed almost as if the excessive use of his muscles had found out the congenital weak spot in the patient's muscular development. It must not be forgotten that muscular dystrophy might arise in almost any group of the skeletal muscles. We perhaps got it too firmly impressed in our minds that there were two varieties of the disease, the well-known pseudo-hypertrophic type affecting the lower extremities, and the type affecting the muscles of the shoulder-girdle. There were mixed examples of these two varieties, and no doubt the disease might commence in still other muscular regions.

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[1] *Proceedings*, 1912, v, pp. 157-63.

Dr. F. PARKES WEBER said that he regarded the father as an almost certain example of primary muscular dystrophy, though the disease had commenced relatively late in life and was masked somewhat by the large amount of subcutaneous fat. The way in which the man moved his body was typical, and there was doubtless much atrophy of the trunk muscles and those of the hip joints. The father and his two sons were all suffering from the same disease, but the children were affected in earlier life than the father; a feature not rarely noticed in hereditary disease. A certain amount of the pseudo-hypertrophic type was often associated with the Landouzy-Déjerine type, and therefore the pseudo-hypertrophic appearance of the younger son's lower limbs was not very surprising; but the present group of cases (the father and his two sons) on the whole fitted in best with the Landouzy-Déjerine family groups. The nephew's symptoms were still doubtful, though perhaps he would develop decided muscular dystrophy later on.

Dr. HERTZ replied that Dr. Johnson and he had brought the four patients forward in order to obtain expressions of opinion as to which of various possibilities was the most likely to be correct. In the first place, the father might be regarded as a case of the pure progressive muscular atrophy type of motor neurone disease, and the two sons and nephew as cases of primary muscular dystrophy, of the pure pseudo-hypertrophic type in the two younger boys and of a mixed pseudo-hypertrophic and facio-scapulo-humeral type in the eldest boy, the former apparently being the primary condition, as the paralysis and atrophy of the facio-scapulo-humeral muscles was of comparatively recent development. If these diagnoses were accepted, then the most probable conclusion was that the association was accidental, as it was difficult to conceive how there could be any causal relationship between a primary spinal cord disease in one generation and a primary muscular disease in the next generation. On the other hand, it was possible, as Dr. Galloway and Dr. Parkes Weber had suggested, that the father also was suffering from a form of primary muscular dystrophy, though he could not agree with Dr. Parkes Weber that the case was an obvious and typical one of that condition.

It was very uncommon for a primary muscular dystrophy to develop at such a comparatively advanced age as 39; the transmission from father to son and to brother's son was very uncommon; the distribution of the atrophy was most unusual, the hands having been first affected and still being the parts most seriously involved; the progress was unusually rapid and the recent improvement was an unusual feature, though both of these points told equally against a diagnosis of progressive muscular atrophy. He was much interested in hearing of Dr. Galloway's case, as he himself had had a very similar one, a professional strong man, who at the age of 25 developed atrophy and paralysis of the muscles of his arms. But in both cases the patients were the only members of their family affected, so they could hardly be regarded as examples of what was commonly called primary muscular dystrophy. However, these cases suggested a possible explanation of the condition of the father of the family shown by Dr. Johnson and himself, as he, too, had used his arms excessively. Perhaps in all these cases there was an inherited tendency for muscular atrophy to occur, and the over-use was the exciting cause. The tendency in the patient G. H. was insufficient to cause atrophy without over-use, but in his sons and nephew the tendency was greater, as it often was in the second generation of inherited diseases, and consequently atrophy and paralysis occurred at an early age without any obvious exciting cause.

Case for Diagnosis.

By T. Jefferson Faulder, F.R.C.S.

Miss N. L., aged 17, has noticed a swelling low down in the neck for about three years. She states that it is increasing in size. It causes no symptoms. There is a tense, elastic swelling in the episternal notch. It does not move with the larynx on deglutition, and no respiratory or cardiac movements appear to be communicated; it therefore seems to be a tumour in "Burns's space"; at the same time it is possible that there is a pedicle passing down into the mediastinum.

Opinions are invited as to the nature of the tumour.

DISCUSSION.

Mr. Sidney Boyd considered that it was a dermoid cyst. They were not very common in that situation. There was a picture of such a case in Bland-Sutton's book; and he had seen a case in a child, aged 7, with a small, hard cystic tumour in the same situation, which he regarded as dermoid. If it were a caseous gland, he thought there would be more surrounding inflammation, and the swelling would be more fixed. The girl was able to work it well forward herself. He had had a case which illustrated the difficulty in diagnosing some of these swellings. It was in a boy, aged 15, who came with a swelling in the lower part of the neck going down behind the manubrium sterni; X-rays showed that it extended two-thirds of the way down the manubrium sterni. He cut down upon it with a transverse incision above the sternum, and found behind the sternohyoid and sternothyroid a tuberculous abscess, but no bone disease, no other glands in the neck, and no tubercle elsewhere. One must bear tuberculous abscess in mind when seeing a cystic swelling in the suprasternal space.

Mr. C. H. Fagge said that if Mr. Faulder meant that the suprasternal cyst was derived from the same internal cleft depression of the primitive pharynx as that from which the thymus was developed, and that, in fact, it was a true branchial cyst, he agreed with him. But he did not agree with the statement that the girl could drag the tumour above her sternum and isolate it. There seemed to be a fair area of diminished resonance behind the sternum, and he believed that operation would show that the cyst extended some distance into the mediastinum.

Mr. Maynard Heath asked whether the swelling was translucent; some dermoid cysts about the head and face were translucent. The presence of translucency would negative the diagnosis of tuberculous abscess.

Mr. Faulder thanked members for their opinions. His feeling was to cut down upon it to see what it was; but he would first do what had been suggested, and report later.

Cases of Pulmonary Tuberculosis before and after Gymnastic Treatment.

By Filip Sylvan, M.D.

Case I.—W. P., shown at the January meeting, is now quite cured.[1] Vital capacity, 5,650 c.c.

Case II.—H. A., aged 42. In February, 1909, he had right-sided pneumonia; during 1910 gradually developed cough. In November, 1910, he attended Mount Vernon Hospital, and tubercle bacilli were found. In April, 1911, he went to Frimley Sanatorium where he did graduated labour, which was frequently interrupted by fever. Was discharged about 1911. He felt stronger, but had still much cough and expectoration, and frequently rise of temperature. In January, 1912, he started work, but after three weeks he had to give it up. In May, 1912, he started gymnastic exercises, and his condition gradually improved and rise of temperature became seldom. Since September, 1912, he has never had fever. Under right clavicle from second to fourth rib cracked-pot sound indicated a cavity of the size of a lemon. In May, 1912, crepitation all over both lungs; now hardly any. He has now been at work for six weeks, and feels quite well. No cough to speak of.

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[1] *Proceedings*, p. 82.

Case III.—W. G., aged 24. At Easter, 1912, he got a very bad cold and cough, and expectoration increased gradually. During January, 1913, he got much worse and lost 1 st. in three weeks. Started gymnastic treatment on February 13, 1913. Left lung dullness on percussion and crepitation all over; right lung slight crepitation behind the shoulder-blade. Vital capacity, 440 c.c. March 8: Crepitation considerably less; vital capacity, 2,650 c.c.

Case of Myelitis treated with Gymnastic Exercises.

By Filip Sylvan, M.D.

F., aged 36. In January, 1910, he got a bad cold, and had severe pains in the back, lower thoracic region. After three weeks his legs began to drag, he gradually lost the use of both legs and had to stay in bed. He went into the Middlesex Hospital where he was treated for myelitis during six months. Soon after he went to the National Hospital in Queen Square, was treated as in-patient for three months, and as out-patient for eighteen months, and he improved a little. In May, 1912, he started gymnastic treatment. The adductor muscles were contracted to such a degree that he could not get his knees apart, and could walk only with great difficulty. He often had a painful cramp in the left foot. March 5, 1913: He can walk without difficulty; has no pain and no cramps; can separate his knees quite easily.

DISCUSSION.

Dr. **Essex Wynter** said that the patient was under his care two years ago, when the progressive paralysis appeared first in the left leg, and then gradually ascended and involved the right. After being six months in Middlesex Hospital the patient was discharged; at this time the paralysis had ceased to extend. He understood that the patient had been three months in the National Hospital, Queen Square, and since then had been much better. The improvement was striking after such a long period of paralysis.

Dr. **Sylvan** replied that the adductor muscles of both thighs were contracted, so that he could not separate his knees, and it was difficult to give exercises. The first thing was to loosen the contraction, and Professor

Sherrington had shown that excitation of the cortical centre for the biceps produced immediate relaxation of the triceps, and similarly with other antagonistic muscles. After working on that principle for some time the legs could be separated a little, making it possible to have the exercises.

Case for Diagnosis.

By P. MAYNARD HEATH, M.S.

J. M., MALE, aged 36, a motor mechanic. In September, 1912, he suffered from a "poisoned finger"—the right little finger, which was followed by a swelling in the axilla. These cleared up, but were followed by a scaly eruption below the right knee and over the lower third of the left shin, and by painful swellings on both shins, at the inner side of the left knee and on the right elbow.

The scaly eruption has now largely disappeared. The swelling in the right leg involves the anterior tibial group of muscles for the greater part of its length. The swelling is very ill defined, is firm, and the skin and subcutaneous tissues over it are œdematous, especially in the lower part just above the constriction caused by the boot. The swelling is said to get worse at night, and pressure over it causes severe pain. The swelling in the left leg appears to be confined to the skin and subcutaneous tissues, though it lies over the subcutaneous surface of the tibia. There is an irregular tender nodule in the subcutaneous tissue at the inner side of the left knee, just above the inner condyle of the femur. The nodule about the right elbow has now disappeared, though the patient still complains of tenderness in this situation.

The patient is fairly healthy looking, but he states that his nose has become red and swollen during his present illness. Well-marked pyorrhœa alveolaris is present. No changes can be found in the blood. The man denies infection with syphilis, and the Wassermann reaction has been negative on several occasions. Potassium iodide up to 100 gr. a day has had no effect on the swellings. The swelling on the right leg was incised on January 9. The skin and subcutaneous tissues were œdematous and the muscles were found to be converted into a firm, greyish, homogeneous-looking material. Sections of a portion removed are shown. No soft spot was found, and cultures from the wound proved to be sterile.

When first seen X-ray photographs showed nothing wrong with the bones, but more recent ones show deposits of new bone on tibiæ and fibulæ.

DISCUSSION.

Mr. MAYNARD HEATH added that clinically the case was obviously syphilitic. The microscopic section of the portion removed from the right leg showed inflammation of muscle, the cellular infiltration tending to follow the course of the blood-vessels. In places there were masses of inflammatory cells, often round the blood-vessels, and the vessel walls were slightly thickened. The section stained by Pappenheim's method showed some larger cells with oval nuclei in the inflammatory masses, but no definite plasma cells.

Dr. F. PARKES WEBER said that the appearance of the right leg suggested the possibility of a form of blastomycosis. Owing to such a possibility, a further biopsy examination could perhaps be undertaken with advantage.

Mr. C. H. FAGGE suggested that full doses of mercury should be given. He had seen several cases which clinically were syphilitic, but which did not yield to iodide of potassium, and yet cleared up on full doses of mercury. His opinion was that the slide under the microscope showed appearances more like gummatous deposits than any other localized inflammatory change. He was sorry Mr. Heath did not discuss the possibility of osteitis deformans, the early stage of which was, in regard to the osseous changes alone, often difficult to differentiate from syphilitic bone disease.

Mr. HEATH, in reply, said that he proposed to give neo-salvarsan. A very thorough biopsy was done, and he did not see the need for another. He did not know much about the early stages of osteitis deformans, but he would not expect to find any affection of the skin and subcutaneous tissues, nor would he expect the X-ray photographs to show merely a slight subperiosteal thickening of all the bones of the legs as in the present case.

Bastedo's Sign: a New Symptom of Chronic Appendicitis.

By ARTHUR F. HERTZ, M.D.

THE frequent occurrence of chronic appendicitis, often unaccompanied by acute attacks, is becoming more and more widely recognized. At first only cases in which chronic pain in the right iliac fossa was present were regarded as examples of this condition, but it is now known that the pain may be situated in the epigastrium alone, although in many cases it is characterized by radiating downwards and to the right. In most instances the pain simulates some gastric or duodenal disorder, as it develops soon after meals. I have, however, seen a number of cases in which the disease manifested itself as recurrent attacks of acute abdominal pain, colicky in nature, and situated in the neighbourhood of the umbilicus; the pain was quite independent of meals, was not associated with any rise in temperature, and its onset and disappearance from half an hour to five or six hours later were generally sudden. Sometimes even in the absence of spontaneous pain in the right iliac fossa there is distinct tenderness in this situation, which may be as great as, or greater than, that in the epigastrium. A characteristic symptom sometimes observed is the production by pressure in the right iliac fossa of epigastric pain, which simulates exactly the spontaneous pain from which the patient suffers.

In the majority of cases, however, a doubt must still be present as to the correct diagnosis, and any new sign which experience proves to be reliable must clearly be very welcome. Such a sign would also prove of great value in those cases in which doubt arises as to whether pain in the lower part of the right side of the abdomen is due to chronic appendicitis or to some disease of the right Fallopian tube or ovary, or to a stone in the right ureter, and, when it is situated at a somewhat higher level, whether appendicitis or disease of the right kidney or the biliary passages is present.

In a paper read before the Medical Society of the County of Richmond, U.S.A., in March, 1909, Bastedo of New York described a sign which he believes is of great value in the diagnosis of chronic appendicitis under the conditions just described. Two years later he published a further communication on the subject [1], and during the last few months papers have appeared by Rost [3], Dreyer [2], and Slawinski [4] in Germany confirming his results. I have now employed the test in a large number of cases, and have found it of such great value that I am anxious that it should become more widely recognized, as it is simple to carry out, causes very little inconvenience to the patient, and is in my experience more reliable than any sign hitherto described.

The test depends upon the production of pain and tenderness in the right iliac fossa on inflation of the colon with air. For this purpose I use an ordinary rubber rectal flatus tube, which is connected by a short piece of glass to a pump, such as that used with the sigmoidoscope. Bastedo recommends that the tube should be inserted 11 or 12 in. into the rectum, but, as the experiments of Goodhart, myself and others have shown that a tube can very rarely be passed beyond the pelvirectal flexure, which is never more than 6 in. from the anus, there is no advantage in introducing it farther than just within the ampulla of the rectum about 1½ in. from the anus. After the tube has been inserted, the patient lies flat on his back, and the pump is brought up between his legs. On now slowly pumping air through the tube, the colon is seen gradually to distend, and after a certain quantity has been introduced, an individual who is not suffering from appendicitis feels a diffuse discomfort in the lower part of the abdomen, but there is no pain unless an excessive quantity of air is introduced, in which case it is not more marked on one side than the other. There is also no tenderness.

Patients suffering from appendicitis, however, generally experience pain in the right iliac fossa, even if the pain has hitherto been confined to the epigastrium or the neighbourhood of the umbilicus. In one of my cases, in which a diseased appendix was subsequently removed, pain was only felt some hours later. Whenever pain is produced, and in some cases in which no pain has been felt, well-marked tenderness is found in the neighbourhood of McBurney's point. When tenderness has already been observed in this situation it is always much increased by inflation, but it is often found in cases in which no tenderness had hitherto been noticed in spite of frequent examinations. In a number of instances I have observed a further exceedingly characteristic symptom; the pain is referred to the epigastrium when pressure is exerted in the right iliac fossa after inflation, the epigastric pain being identical in character with that which formed the chief symptom of which the patient complained.

The test known as Rovsing's sign, although it had been employed for some years before Rovsing's first publication on the subject by Dr. Lauriston E. Shaw, in which pain is felt in the right iliac fossa on exerting pressure over the descending colon, has probably a similar significance to Bastedo's sign, as it appears to depend upon gas being pressed from the distal into the proximal part of the colon. It is, however, of very limited use, as it is comparatively rare for sufficient gas to be present in the descending colon. After the colon has been inflated in the carrying out of Bastedo's test and the pain produced has disappeared, it can often be caused to return by pressing upwards along the descending colon, as in Rovsing's test.

In my own experience, I have only obtained a positive Bastedo's sign in appendicitis, the appendix having always been found diseased at the subsequent operation, with the possible exception of one case, which is still too recent to form a judgment as to whether the removal of an apparently normal appendix will cause the symptoms to disappear. The only other case so far recorded, in which appendicitis was not present, was one in which Rost found that inflation of the colon gave rise to pain in the right iliac fossa, although the appendix had previously been removed. At the operation, which was subsequently performed, a band was found passing from the side of the cæcum, which was otherwise abnormally movable; in all probability the pain resulted from the pull of this band upon the distended and movable cæcum when the colon was inflated. In numerous cases, in which the colon has been inflated and no pain or tenderness produced, the appendix has later been found to be healthy, and in some cases disease has been discovered in the female pelvic organs, the gall-bladder or the kidney. In only two cases so far recorded has a negative test apparently been at fault, Bastedo having had two patients in whom typical attacks of appendicitis occurred within six months of his test having been tried with a negative result.

I have only employed the test in cases of suspected chronic appendicitis and in patients who have recently recovered from a doubtful attack of acute appendicitis. Mr. Eric W. Sheaf, of Guildford, tells me, however, that in two cases of acute appendicitis, in which he was in doubt as to the diagnosis, he employed the test and found that the introduction of a very small quantity of air at once increased the pain. He would, of course, not have done this had he not been prepared to operate at once, and in both cases he operated within a few minutes and removed an acutely inflamed appendix. Inflation in such cases is naturally contra-indicated unless it is possible to operate at once, if the test proves positive, and great care must also be taken to introduce only just sufficient air to produce a slight increase in the pain, as otherwise there would be a danger of tearing adhesions or causing a diseased appendix to perforate.

In my Goulstonian Lectures I showed that the only stimulus to visceral pain is distension. On inflating the colon the pain eventually produced occurs no sooner in the appendix than in the rest of the colon, so long as the former is not diseased, but if it is inflamed pain is much more readily produced, and consequently local pain and tenderness are observed in the right iliac fossa. It is not yet clear to what extent adhesions binding down the appendix may be concerned in the production of the pain, but in some of my cases there were no adhesions, and consequently it is clear that this cannot be the sole factor.

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[3] Rost, F. *Münch. med. Wochenschr.*, 1912, lix, p. 2055.
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Clinical Section.

April 11, 1913.

Sir WILLIAM WATSON CHEYNE, Bt., C.B., F.R.S., Vice-President of the Section, in the Chair.

Case of Chronic Bone Disease.

By E. M. CORNER, M.C., and W. J. PETTY, M.B.

THIS girl was shown at the Clinical Society on October 26, 1906. The disease of the bone was then regarded as syphilitic. Later the bony enlargements, confined to one limb and the corresponding half of the pelvis, were shown to be enchondromata. Finally the disease was regarded as a series of developmental errors whose distribution suggested the idea that they were dependent on a dystrophy due to some nervous lesion, possibly congenital.

Since 1906 the patient broke her leg and came to the hospital because union was slow and unsatisfactory. The shortening of the limb in 1906 was 2½ in.; it is now 5¾ in., the greater part (3½ in.) being due to differences in the femora.

Case of Ossification in the Brachialis Anticus.

By E. M. CORNER, M.C.

THE patient is a young man who fell on his hand a few months ago, fracturing the lower end of his radius. There was no complaint of any injury or pain in the neighbourhood of the elbow-joint. Later the patient discovered a painless lump in front of the elbow. A skiagram showed that this lump was due to some ossification in the brachialis anticus muscle. Presumably this is the result of a partial rupture of the muscle, done unconsciously at the same accident.

Case of Recovery after Severe Electric Burns.

By E. M. Corner, M.C., and W. M. Oakden.

G. R., male, aged 32. Five years ago had an attack of sciatica. The patient is employed by the London Electric Supply Corporation, and on September 7 was standing on the top of the transformer, a distance of 4 ft. from the ground, with his left leg pressed against the runner bar, cleaning the bus bars. In the process of his work his left palm came in contact with the terminals, the current having not been previously disconnected. The current was an alternating one, with a strength of 10,000 volts. He immediately felt a shock through his left hand and was thrown to the ground, where he lay unconscious for a period of about two minutes. On rising he was slightly dazed, but had no pain, and was unaware of the presence of the burns till he saw them. He was brought to St. Thomas's Hospital within half an hour, when his condition was as follows:—

Temperature 99·4° F. Pulse 112, of poor volume and tension. Respiration shallow and slow. Considerable shock present. Burnt areas: (1) Left upper extremity—A patch, the size of a shilling, over the inner part of the antecubital fossa to the fourth degree. The forearm and the palmar aspect of the hand to the second and third degree. (2) Right foot—The outer half of the dorsum to the second and third degree. Fourth and fifth toes to the fourth degree. (3) Left lower extremity—A patch, the size of half-a-crown, about the middle of the inner aspect of the leg to the fourth and fifth degree. Two small patches on the inner border of the foot to the second and third degree.

The temperature remained intermittent for six weeks, varying between 100° and 102° F. The inflammatory reaction in each area was well marked, but most so in the left calf, where there was considerable necrosis and sloughing, portions of the inner border of the tendo Achillis and inner head of the gastrocnemius separating. The superficial area reached the size of a large saucer before healthy granulations appeared. This large area was grafted on September 30, but without success. On October 29 another attempt was made, and the result was extremely satisfactory in that practically all the grafts took. The fourth and fifth toes of the right foot became dry and shrivelled up, so recourse to amputation was necessary on September 25. Pain was conspicuous by its absence throughout, and even when the wounds were dressed it was very slight.

The patient complained of loss of sensation in the left hand about the middle of October. On November 15, when the arm had practically healed, except for a small and rather deep focus at the elbow, the peripheral nerves were examined and the following condition found: Sensory—The shaded pencil areas (fig. 1) indicate the loss of protopathic sensibility, the area bounded by the ink line the loss of epicritic. Trophic—The skin was cold, blue, dry, and smooth over above areas. Motor—There is marked wasting of the interossei, the muscles of the thenar and hypothenar eminences, and also of the flexors of the forearm to a lesser degree. Flexion of the wrist and fingers, adduction and abduction of the hand, adduction and abduction of the fingers, and thumb movements are not possible. The terminal phalanx of the thumb cannot be flexed. Muscles do not react to faradic stimulation, but there is a slight reaction to galvanic.

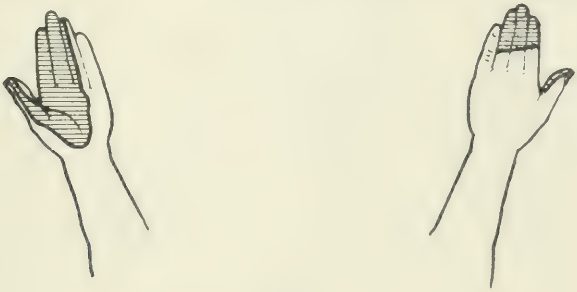

FIG. 1.

As the elbow wound had firmly healed by the middle of December, the median nerve was exposed in the antecubital fossa, as it was thought that the suppuration might have extended deeply and implicated the nerve. The nerve, however, was found to be perfectly normal, except that the perineurium was very lax. On stimulating the nerve with sterilized electrodes there was a weak but definite contraction of the muscles supplied by it.

Treatment by massage was instituted in the middle of November, and there has been a slow but steady and continuous improvement since, as will be seen from the following reports:—

(1) January 15, 1913: Sensory—The diagrams (fig. 2) show the

loss of protopathic and epicritic sensibility. Trophic—The skin is of normal colour and temperature, but remains soft and smooth. Motor—The flexor muscles of the forearm have increased considerably in bulk, but there is no obvious change in the interossei and the muscles of the thenar and hypothenar eminences. Flexion of the wrist and fingers and adduction and abduction of the hand can be effected weakly. The index-finger lags a little behind on flexion. The terminal phalanx of the thumb can be flexed. Abduction and adduction of the fingers are impossible and the thumb movements are very slight. The flexors react to both forms of electrical stimulation, but the interossei and the muscles of the thenar and hypothenar eminences only react to the galvanic current.

(2) February 25, 1913: Sensory—The diagrams (fig. 3) show the loss of histopathic and epicritic sensibility. Trophic—The skin is still soft and smooth. Motor—The flexor muscles of the forearm more nearly approximate those of fellow-arm in bulk. Wasting is not quite so marked in the interossei and the muscles of the thenar and hypothenar eminences. Flexion of the wrist and fingers is much stronger, the index now conforming to the other fingers in its range of movement. Abduction and adduction of the fingers can be effected to about half the normal amplitude.

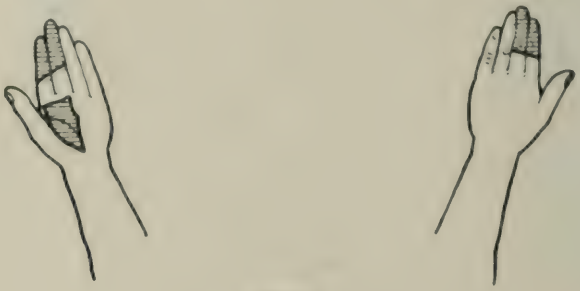

FIG. 2.

POINTS.

Burnt areas: The left leg being pressed against the runner bar would account for the burn at this site. Its depth, as opposed to the superficial nature of the other areas, depends on the fact that the resistance of the body varies according to the nature of the tissue, the moister the tissue the less the resistance, and in consequence, the deeper the burn. There was money in both trousers pockets, and it is interesting why no burn ensued at these sites. No explanation can be given why the elbow should sustain a more severe burn than the hand, seeing that it was not in contact with any metal. The marked absence of pain throughout is somewhat unusual, as burns of this nature are said to be invariably characterized by severe pain. This was a prominent feature in Clement Lucas's case. The slowness of the healing processes and the absence of contraction in the scar are noteworthy.

Associated paralysis: The paralysis corresponds to the peripheral distribution of the median and ulnar nerves. The loss of sensation to the median nerve. It is peculiar that there should be complete loss of median sensation, without a corresponding loss for the ulnar nerve, seeing that the motor fibres of both nerves sustained the same degree of injury.

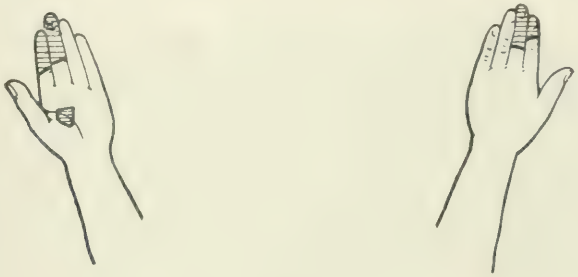

Fig. 3.

Nerve palsy due to severe electrical stimulation appears to be extremely rare, as after an extensive search I can find no trace of a similar condition in the literature on this subject. Clement Lucas[1] describes a case in which a 2,500 volt terminal was touched, with inhibition of the function of the nerves of the arm for several days. The injury must have arisen in the terminal ramification of the nerves, causing subsequent degeneration which ascended for a considerable distance, seeing that the flexor muscles of the forearm were paralysed to a lesser degree.

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[1] R. Clement Lucas's "Case of Accidental Electrocution," *Trans. Clin. Soc. Lond.*, 1905, xxxviii, pp. 86-92.

DISCUSSION.

Mr. CORNER added that it was unusual for recovery to take place after the passage of so large a current through the body. This was attended by absolute painlessness and unconsciousness, falling "on sleep," a very interesting fact in reference to the electrocutions that took place in America.

Dr. ESSEX WYNTER said he had had experience of cases of minor electric burns while applying the galvanic current. Although the application of the current was disagreeable or painful, the ulcer resulting was devoid of pain, but very indolent in healing—one the size of a sixpence might take as long as three months. An interesting fact in reference to the abolition of minor deformities such as moles and nævi was, that healing of an electrically produced slough or ulcer occurred without contraction; hence it was opposite to the general rule, that the slower the healing the greater was the cicatricial contraction.

Case of Myeloma of the Head of the Tibia Seven Years after Enucleation.

By C. H. FAGGE, M.S.

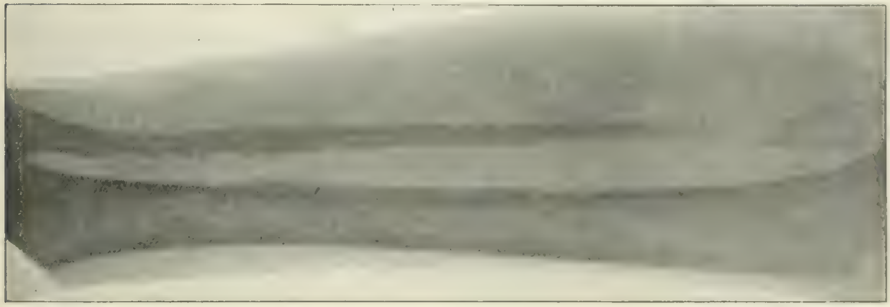
R. C., AGED 11, first noticed a swelling over the head of the left tibia about January, 1905. The pain made her limp, and she came up to Guy's Hospital, where, in April, 1905, the tibia was explored on the suspicion of abscess or myeloma; nothing abnormal was found even after free exposure and drilling of the bone. Pain continued in spite of iodides, and swelling increased, particularly on the lateral aspect of the bone. X-rays showed the upper end of the tibia expanded, and the original diagnosis of myeloma was reverted to; there was no albumosuria. A year after the first operation the bone was again exposed, and in separating off the periosteum the rougine entered a large cavity in the bone filled with maroon-coloured growth. The opening was enlarged by cutting away the compact layer with a gouge, when the upper third of the bone was found expanded and hollowed out. Posteriorly over a large area the exploring finger came in contact with bare periosteum. The soft growth was carefully scraped away with a Volkmann's spoon, the cavity washed out with saline and packed with gauze.

Microscopically the growth shows general myxomatous degeneration, but in one part are mingled round and spindle cells with several myeloid cells scattered among them.

Re-formation of Fibula after Complete Removal of Diaphysis for Acute Infective Periostitis.

By C. H. Fagge, M.S.

W. H., aged 11, was admitted into Guy's Hospital in December, 1911, with an acute inflammatory swelling of the outer side of the right leg; the knee- and ankle-joints were not involved. Incision disclosed the shaft of the fibula lying bare in a large subperiosteal abscess; its lower limit was the lower epiphyseal line, and through this the bone was divided with a chisel, when the diaphysis came away, having been spontaneously separated at the upper epiphyseal line. The cavity was packed. A second abscess over the outer side of the knee was incised; pus from both cavities contained *Staphylococcus aureus*. Saline was infused subcutaneously into both axillæ, and two days later both areas of infusion became infected, and pneumonia developed at the right base. Two pectoral abscesses were opened and anti-staphylococcal serum was given. Skiagrams taken at intervals since January, 1912, show a gradually darkened shadow in the position of the fibula, which is now completely osseous except just below the upper epiphyseal line. Section of the removed diaphysis showed a pyogenic focus in the medulla from which a sinus led through the compact layer.



Re-formation of fibula after complete removal of diaphysis.

Case of Re-formation of Tibia after Resection of Diaphysis for Chronic Osteo-myelitis. (?) Syphilitic.

By LIONEL E. C. NORBURY, F.R.C.S.

J. W., MALE, aged 10, was admitted to the Belgrave Hospital on July 25, 1912. History of swelling of left tibia, four years. Sanatorium treatment for past six weeks. Previous history of eye trouble. Ulcer on right leg two years ago. Father said to have had a chancre. Mother said to have suffered from tuberculous ulcers of leg and shoulder.

On examination: Thin, sallow boy. Enlargement and bowing forward of shaft of left tibia. Thickening of bone extends from just below tibial tubercle to 2½ in. above ankle-joint. Nodes on tibia, one tender. Skiagram shows diffuse sclerosis of bone, with several light areas, suggesting collections of pus. Small node on right shin. Scar of old ulcer on right leg. Diagnosis—(?) syphilitic, (?) tuberculous. Wassermann reaction negative. No previous anti-syphilitic treatment. Treated with mercury and potassium iodide; also with mercurial inunctions. No apparent change locally.

August 16: Subperiosteal resection of 5 in. of shaft of tibia. Periosteum adherent to bone in two places and was button-holed at these spots; openings closed with catgut sutures. Periosteal sleeve closed with interrupted catgut sutures. Wound healed by first intention.

Plaster of Paris splint twenty-four days after operation and reapplied several times since.

Skiagram now shows a considerable degree of re-formation of the shaft, which, however, is not yet complete.

Microscopical report on decalcified section of bone removed: Chronic osteitis. No evidence of tubercle. Evidence of syphilis indefinite also, but there is one area showing a mild endo-arteritis.

DISCUSSION.

Mr. NORBURY asked for opinions as to the diagnosis. It was difficult to be certain whether the disease was tuberculous, or syphilitic, or some other chronic bone trouble. Was one justified in operating? He thought he was, because the disease was beginning to involve and spread through the periosteum. Anti-syphilitic treatment was tried for only a short time, and he thought it was better to attack the disease early, and remove it altogether.

Mr. LAWRIE MCGAVIN congratulated Mr. Norbury on the result, but it was difficult to answer his question in regard to diagnosis, without having seen the original condition. The result seemed to afford ample justification for saying that the correct method was probably adopted. He asked what was the experience of any surgeons present with regard to the implantation of the fibula into the upper and lower ends of the tibia in cases like this, where the whole diaphysis had been removed. Some cases were reported in the *Annals of Surgery*[1] a few years ago in which the fibula had been transplanted between the separated ends of the tibia, and the result seemed to have been satisfactory: the fibula hypertrophied to such an extent that it became almost the same thickness as the tibia had originally been. He was interested in the slight amount of shortening which had occurred; and it was curious that it should have been so, considering that, as Mr. Norbury told him, no special effort had been made to keep the two ends of the tibia apart during the healing process. One would have thought there would have been a tendency for the foot to turn into a position of varus, and that there would have been considerable shortening if not treated by a splint. If any member had had experience of the treatment, he would like to know whether the result was the same. If so, it would seem to suggest that the larger operation of transplanting the fibula was more or less unnecessary. A quarter of an inch of shortening was very small after such an amount of bone destruction.

Ateleiosis in a Man, aged 45.

By F. PARKES WEBER, M.D.

THE patient, F. B. H., of English parentage, is an infantile dwarf, aged just 45, whose height without boots or shoes is 120·5 cm. (47½ in.) and who at present weighs 26·5 kilogrammes (58$\frac{3}{10}$ lb.) without clothes. He has heard his parents say that he was much like other children up to the age of 9, but that his growth and development then ceased. He is said to have had "water on the brain" as a baby, and, as a child, to have had two or three falls on the head. He cannot himself remember having had any serious illness. There is no history of chronic diarrhœa or intestinal steatorrhœa. His head is rather large for the diminutive size of his body. The shape of his trunk, the undeveloped state of his sexual organs, the appearance of his neck (owing to the want of projection of the "pomum Adami"), and the high pitch of his voice, are those of a child, but his expression, the wrinkles on his face, his attitude, his manner of speaking, and his general behaviour, are somewhat more those of an adult. His mental development is childish, but otherwise not abnormal. He cannot read or write, but, apparently "on account of weakness," the doctor would not let him go to school as a child. He earns a little money by light work in a glass factory. Dr. Reginald Langdon-Down has kindly examined the patient's mental development by the Binet tests (April, 1913), and he reports as follows:—

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[1] Huntington, *Ann. of Surg.*, Philad., 1905, xli, p. 249. Codman, *ibid.*, 1909, xlix, p. 820.

"This patient is interesting, as he was deliberately deprived of school instruction on medical advice, and, on the other hand, owing to his mature years has long been subject to the influences of everyday life in town. The result of this environment has been to produce a superficial sharpness in one or two directions, which might readily mislead one in judging of his intellectual development. In grading him the modified scale of the Binet tests has been used, which avoids as far as possible tests which depend upon the results of instruction. The examination shows that he has reached an intellectual development such as is reached by the average child aged 8."

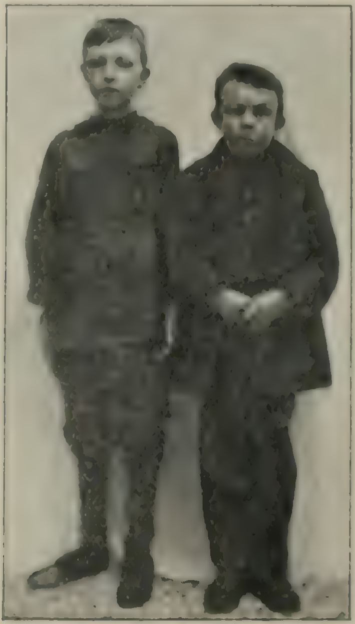

FIG. 1.

Photograph of the patient F. B. H. (on the right), together with a boy, aged 8¼ years, of normal size and development.

His father and mother were both born in 1832, and lived to about the ages of 56 and 50 respectively. There is no history of any other dwarfism or infantilism in the family.

I showed the case before the Society three years ago,[1] when the patient's age was 42, and since then there has been little change. He has as yet no grey hair or baldness on his scalp. There is no hair on his face or pubes or in his axillæ. The penis is exceedingly small. No testicle can be felt on the right side; the left testicle, of about the size of a small cherry, is incompletely descended. Nothing abnormal can be discovered in the heart, lungs, or blood-vessels, or in the abdomen (by palpation), or by examination of the urine. Pulse, 100 per minute.

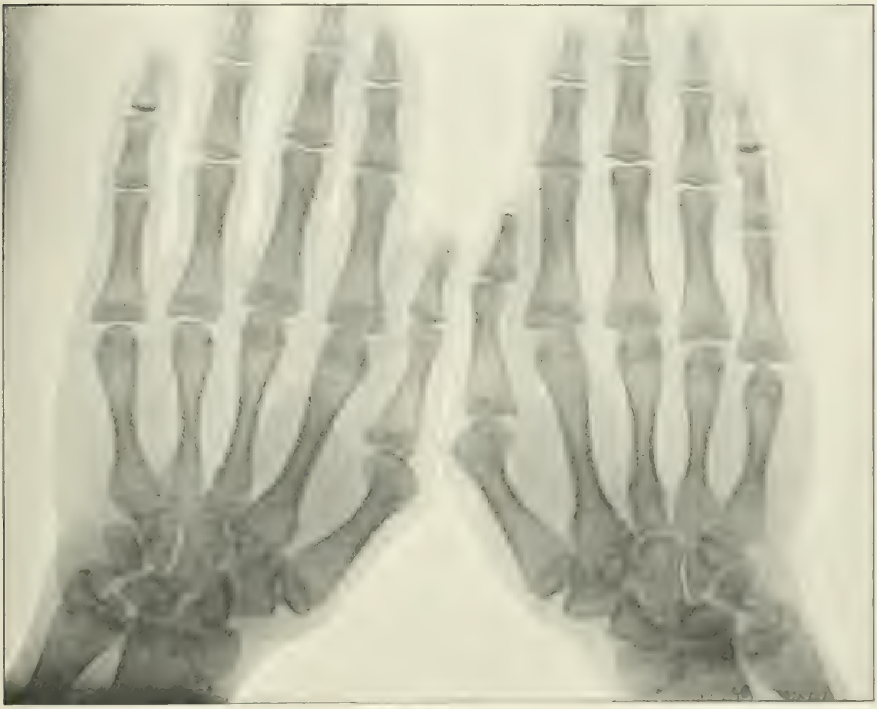

Fig. 2.

Skiagram (1913) of the hands of F. B. H., aged 45, showing persistence of some of the epiphyseal cartilages, which are bordered by deep shadows, apparently due to sclerosed bone.

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[1] *Proceedings*, 1910, iii (Child. Sect.), p. 143.

Brachial systolic blood-pressure, 145 mm. Hg. Blood examination (Dr Bauch, April, 1913): Hæmoglobin, 80 per cent.; red cells, 5,040,000, and white cells, 15,900, to the cubic millimetre of blood. The abdomen is prominent so as to form a regular "alderman's paunch," and the development of the subcutaneous fat about the back of the neck and the front of the thorax is of somewhat eunuchoid type. He apparently has never had penile erections or sexual desire, but has had nocturnal emissions occasionally. The texture of the skin, the facial complexion, the extreme fineness of the hairs of the scalp, and the small size of his thyroid gland, all suggest that there may be an element of hypothyroidism combined with the ateleiosis, but these features may be merely a part of the general infantilism.

Examination of the eyes (Dr. R. Gruber, April, 1913): The pupillary reactions are normal. In each eye there is a little horizontal nystagmus on exact fixation of objects. In each eye there is a central corneal macula (from old inflammation), with a little pigment on the anterior capsule of the lens. The optic disks are both normal. In the left eye one and a half disk diameters above the optic disk, is a well-defined circular area (resulting from old retino-choroiditis), a little larger than the optic disk, over which the retinal pigment has disappeared (with the exception of some small irregular specks), so that the choroidal vessels are clearly visible. At the inner upper peripheral portion of this area there is a small spot of shining silvery-white appearance, lying around a retinal artery, which it partly covers. The visual acuity for the right eye is $\frac{6}{36}$, and for the left eye about $\frac{6}{24}$. With the perimeter the fields of vision cannot be exactly traced out, but there is no temporal hemianopsia. There is no colour-blindness. The atrophic retino-choroidal patch in the left eye is similar in appearance to patches supposed to be seen after recovery from miliary tubercles of the choroid. The impairment of vision and the nystagmus are probably due to the old corneal maculæ. Examination of the ears (Mr. G. J. Jenkins) shows nothing abnormal, excepting that the external auditory meatus is of the infantile type. It may be here mentioned that the patient's knee-jerks are very difficult to obtain, but are not altogether absent; there is no ankle clonus; the plantar reflexes are of the normal flexor type.

In regard to the condition of the skeleton, there is slight spinal scoliosis. Skiagrams (Dr. N. S. Finzi) of the extremities show persistence of some of the epiphyseal cartilages, but the amount of union of the epiphyses with the diaphyses varies considerably in different bones. Dr. Finzi points out that, in the skiagrams of the hands, those epiphyses which are not yet joined to the diaphyses, and some of the diaphyses themselves, are seen to be bordered by a very deep shadow, as if they were bordered by sclerosed bone. The epiphysis of the terminal phalanx of each little finger is represented by a single very black line, as if it entirely consisted of very much sclerosed bone. In the same skiagrams the carpal bones seem to be relatively more developed than the bones of the metacarpus and fingers. When I previously showed the case I thought that skiagrams of the skull showed nothing special. Fresh skiagrams of the sella turcica region recently taken by Dr. Finzi, seem to show that the posterior clinoid process is very thick, but slight variations in the position of the head may make considerable differences in the skiagraphic appearances at the base of the skull. The pituitary fossa is, however, almost certainly rather small.

Ateleiosis in a Woman, aged 20, with a Slight Congenital Malformation of the Hands and Feet.

By F. Parkes Weber, M.D.

The patient, E. B., of Polish Jewish descent, born in June, 1892, is small and infantile in development. Height, 135 cm.; weight, 34 kilogrammes. According to the mother's account she was born at full term without instrumental aid. She learned to talk and walk like other children. She was always small and delicate, but has hardly increased in height or general development since the age of 12. She has never menstruated. She has been subject to psoriasis since the age of 10, and has chronic middle-ear disease on the right side, which apparently followed scarlet fever at 5 years of age. She has likewise had measles, mumps, several attacks of tonsillitis, and (at 7 years of age) severe pneumonia. For a considerable time between the scarlet fever at 5 years of age and the pneumonia at 7 years of age, she suffered from more or less baldness of the scalp. In 1907 she was for a short time an in-patient under my care for an attack of diarrhœa with dysentery-like symptoms. In 1910 she had swelling of the right knee for two or three weeks without fever. Two years ago she was treated at a hospital for lateral curvature of the spine. She is the eldest child of healthy parents. Of her five brothers and sisters four are living and one (the fifth of the family) died as a baby of convulsions and pneumonia. The mother has had no miscarriages or abortions. There seems to have been no dwarfism, infantilism, or congenital malformation of hands and feet in any other members of the patient's family.

Present condition: There is a slight congenital malformation of the hands and feet, consisting in decided relative shortness of the fourth finger (ring-finger) of each hand, and the fourth toe of each foot. Röntgen-ray examination of the hands (*see* the skiagrams) and feet shows this deformity to be due to abnormal shortness of the fourth metacarpal and fourth metatarsal bones respectively. The patient has a fresh complexion and, as already stated, suffers from chronic psoriasis and chronic right middle-ear disease. The lower jaw is relatively somewhat small and the chin receding. The teeth are fairly well developed. The thyroid gland seems very small. The breasts are quite infantile, and there is complete absence of pubic and axillary hair. A gynæcological examination made under general anæsthesia by my colleague Dr. E. Michels, at the desire of the patient and her mother, showed that,

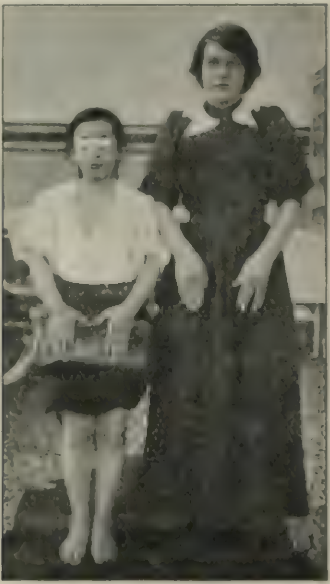

FIG. 3.

Photograph of the patient E.B. (on the left), showing abnormal shortness of the fourth finger of each hand and fourth toe of each foot. On the right is a normal woman of about the patient's age, for comparison in regard to size.

although a small intravaginal portion of the cervix uteri was present, the body of the uterus seemed almost absent, and no ovaries could be felt. Nothing abnormal can be detected by ordinary or X-ray examination of the thoracic viscera, or by palpation of the abdomen. The liver and spleen are not enlarged. The pulse is rather frequent (104 to 116 per minute), and the brachial systolic blood-pressure rather low (100 mm. Hg.). Blood examination (April 3, 1913): Red cells, 4,200,000 to the cubic millimetre of blood; white cells, 8,000; hæmoglobin, 65 per cent. The urine (quantity not excessive) is free from albumin and sugar, but on one occasion recently was found temporarily to contain 0·33 per cent. of sugar. In a case of so-called "mitral infantilism," I recently observed similar occurrence of temporary glycosuria. The condition of the bowels seems to be normal.

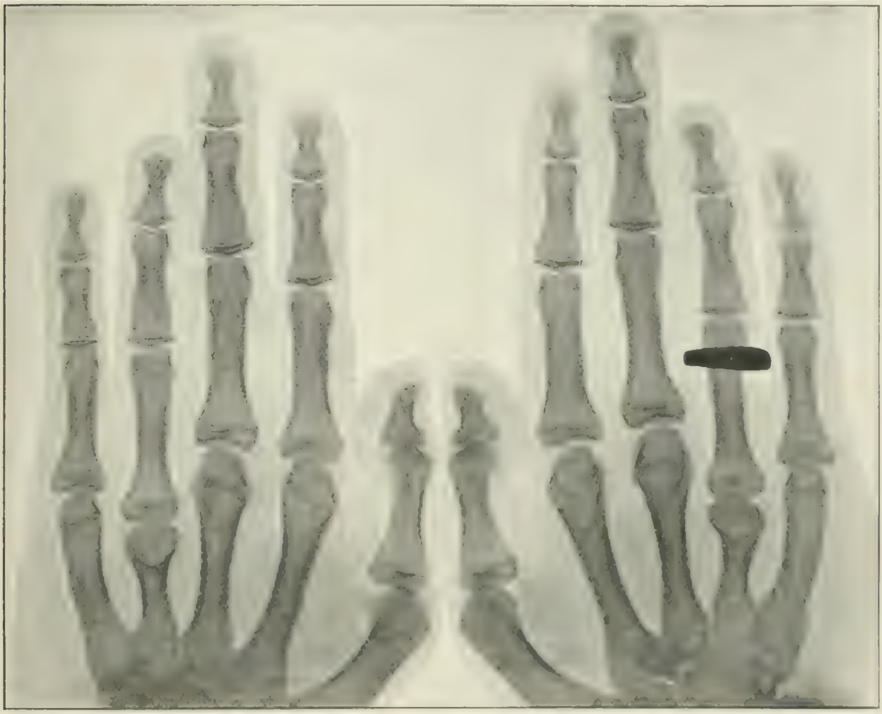

FIG. 4.

Skiagram of the hands of E. B., aged 20, showing abnormal shortness of the fourth metacarpal bones and abnormal persistence of epiphyseal cartilages.

Ophthalmoscopic and perimeter examinations (Dr. C. Markus) show nothing abnormal; the visual acuity is $\frac{6}{6}$ for each eye. The pupils react naturally to light and accommodation. The patient has, as already stated, old middle-ear disease, and she is slightly deaf. The knee-jerks are excessive (as in some neurasthenic subjects); Achilles reflexes active; no ankle clonus. In proportion to the bodily development the patient's mental development seems to be fairly good and she is able to earn a little money at fur-machining. Dr. Reginald Langdon-Down has kindly examined the patient's mental development by the Binet tests (April, 1913), and he reports as follows:—

"This patient suffers from the drawback of a slight defect of hearing, which probably affected her response to some of the tests to a certain degree, for which allowance has been made. The examination shows that she has an intellectual development equivalent to that reached by the average child aged about 13 or 14. It is admitted that the differentiation of natural capacity towards the upper limit of the scale is more difficult, even with the revised Binet's tests, or their modification by Goddard, and these are at present in a tentative stage and do not attempt a closer approximation over 12 than a test for age 15, and one for ages more than 15. This case seems to fall just short of the 15 standard. It is obvious that social status and environment may exercise considerable influence in these higher levels, and that standardization becomes more difficult owing to increasing complexity of development and individual divergences."

In regard to the osseous system, there is slight spinal scoliosis. Skiagrams (Dr. N. S. Finzi) show abnormal persistence of epiphyseal cartilages, and the congenital abnormality of the metacarpal and metatarsal bones already referred to. In those taken of the hands (*see* figure) most of the epiphyses are seen to be bordered by a very deep shadow, as if by sclerosed bone. Skiagrams of the region of the sella turcica at the base of the skull seem to show a rather small pituitary fossa.

I think the present case should be included amongst the "ateleiosis" group according to the descriptions of Hastings Gilford, the originator of the term.

Pseudo-hypertrophy of the Breast.

By P. MAYNARD HEATH, M.S.

M. A., GIRL, aged 16. In March, 1912, she noticed that the right breast was growing bigger than the left. It continued to grow until November, 1912, since when there has not been much increase. At no time has there been any pain. The breasts began to develop at the age of 14. Menstruation started at the age of 15 and has been slightly irregular, the intervals between the periods being sometimes five or six weeks. It is stated that the breast gets slightly larger just before the period and decreases after it. The right breast is considerably larger than the left. There are some superficial enlarged veins, but the skin is normal. The whole breast tissue is lumpy and appears to be divided up into more or less separate lobules. There is no tumour which can be separated from the breast tissue. The breast is not adherent to the chest wall. There are a few enlarged glands under cover of pectoralis major. The left breast is slightly lumpy. There are no glands in the left axilla.

The girl's general health is said to be indifferent and she has a cough, but there are no signs of pulmonary tuberculosis, from which disease her eldest sister, aged 21, is suffering.

The breast has remained in its present condition for the last four months in spite of continuous support and pressure with Scott's dressing.

Mr. MAYNARD HEATH said he had called it pseudo-hypertrophy because it did not seem to him to be a true hypertrophy. It differed in being unilateral, although he thought there was an indication that the left breast was undergoing a similar, but slighter, change, and in not being uniform; there were great masses of breast tissue, which felt as if they had undergone chronic inflammation, and there were glands in the axilla; indeed, they were now well marked in both axillæ. This condition had remained in practically the same state for four months, and if it had been tuberculous he thought it would have broken down by now. Von Pirquet's test was negative, but that was not strong evidence. He would like suggestions as to treatment, as nothing he had done had improved her; should any operation be done? It had been suggested that he should excise the glands, to see if they showed evidence of tuberculosis.

Congenital Cyst in Parotid Region.

By P. MAYNARD HEATH, M.S.

E. G., GIRL, aged 16. A swelling in front of the right ear was noticed soon after birth. At the present time, when the patient's head is erect, there is a distinct hollow in front of the tragus. At the bottom of this hollow is a hard nodule about the size of a millet seed, which is tender on pressure. The skin over the hollow is normal in appearance. When the patient bends her head downwards and to the right the hollow is gradually filled up and is replaced by a rounded swelling, which projects about ½ in. above the surface of the cheek. The skin over the swelling then shows a faint blue tinge. The swelling is cystic and can be made to disappear by pressure. It is not translucent and no pulsation or impulse can be detected in it. There is no abnormality in the auricle or meatus.

DISCUSSION.

Mr. MAYNARD HEATH said he regarded it as a nævoid condition, possibly in connexion with the first branchial cleft. He did not regard his title as a good one; it was not a cyst in the true sense of the word. It was rather a nævus or lymphangioma. Probably the former, on account of the bluish colour. He did not propose to do anything to it.

Dr. F. PARKES WEBER, owing to its feel and position, thought that the swelling was probably a cystic lymphangioma. He acknowledged that there was a slightly bluish appearance of the stretched skin over it.

Case of Chronic Hydrarthrosis.

By LAWRIE MCGAVIN, F.R.C.S.

T. L., AGED 32, labourer, on June 5, 1912, fell a distance of 40 ft., and was admitted to Seamen's Hospital with simple fracture of both bones of the right leg. On June 11 Mr. McGavin operated. Fibula plated; four-screw plate. Tibia not plated. On July 25 discharged apparently cured. On August 12 readmitted with considerable swelling of the right ankle. On August 23 plate removed. On September 24 discharged as "relieved"; still swelling of the right ankle. On February 14, 1913, readmitted with swelling of the left knee-joint. This swelling subsided under strapping with Scott's dressing, but has reappeared each time that he has been allowed to use the joint for walking. On February 18 the knee-joint was washed out with 1 in 5,000 perchloride solution. At present still some swelling of, and fluid in, the joint, and much grating on passive movement. Slight grating in the right knee-joint also. X-rays show no bony change present.

Swelling of the Lower Extremity following Bassini's Operation.

By Lawrie McGavin, F.R.C.S.

S. H., aged 38, waterman. Admitted to Seamen's Hospital, November 11, 1912, suffering from double hernia. On November 14 Mr. McGavin operated. Stovaine. Bassini's operation done on both sides. Linen thread was used throughout. On February 19 clips removed. Right side slightly red. Left side apparently perfectly all right. On February 21 complained of pain in, and swelling of the left calf. Internal saphenous vein felt thrombosed on the inner side of the left thigh. Leg wrapped up in cotton-wool and slung from a cradle. At mid-thigh circumference of limb was 2 in. greater on the left side than on the right; calf of left leg 2½ in. greater than right. By March 10 the limb had returned to its normal size. When patient was allowed out of bed there immediately reappeared some swelling of the limb. This has not yet entirely subsided, although the patient has been confined to bed since the reappearance of the swelling.

Cystic Tumour in Submaxillary Region.

By Lawrie McGavin, F.R.C.S.

G. S., aged 63, fitter. Swelling in left submaxillary region; gradual onset during last four months; painless. Swelling smooth, fairly soft, semi-fluctuating, slightly softer at the lower part; not fixed to jaw. Mass the size of a large tangerine orange. Mouth and pharynx show nothing abnormal. Enlarged glands at bifurcation of left common carotid. Needle inserted drew off yellowish-brown, oily fluid, containing cholesterin. Upper part of the swelling then found to be nodular and hard. Mixed tumour of the left submaxillary salivary gland?

DISCUSSION.

Mr. McGavin said, in regard to the second case, that the swelling was not accompanied by tenderness or pain, and there was no suppuration, or anything to account for it. Nothing which he had done for the man seemed to make it better. He thought it would progress until there was elephantiasis. Whether it was venous or lymphatic he did not know, but he invited suggestions. Apparently the man was in good health, but when the man got on to his legs, this leg began to swell. In the third case, the swelling did not contain pus, a brownish material was drawn off, and it was found to contain cholesterin. A hard mass was found in the neighbourhood of the submaxillary gland, and there was a chain of enlarged glands high up. There were no means of ascertaining where it started. Possibly the man had got a malignant focus deep in the neck, and the upper cervical glands were secondarily affected and were now extending forwards underneath the submaxillary gland, but apparently the larynx and œsophagus were normal. He asked for an expression of opinion as to whether the case was malignant, or if it was malignant whether it should be operated upon. He was inclined to leave it alone.

Dr. Essex Wynter, in regard to the second case, in which there was thought to be thrombosis, said that in a number of cases in recent years he had found striking benefit from the application of a leech in the early stage, when thrombosis was commencing, the application being made not to withdraw blood, but because it altered its degree of viscosity. Hæmorrhages had broken out after the application of a leech, from a wound which had been sealed some hours. He had been impressed with the result of this treatment in typhoid fever, in association with which thrombosis was not uncommon. He thought this abortive treatment of thrombosis deserved to be more widely known.

Mr. McGavin replied that he was interested in Dr. Wynter's remarks. In operating upon patients who gave a history of slight thromboses, one was anxious whether anything serious, such as pulmonary embolism, might develop. He asked if Dr. Wynter had had experience of attempts made to minimize the danger of such an accident occurring after operation, by producing artificial liquefaction of blood, such as by giving large doses of citric acid. He had recently had two cases of pulmonary embolism in which it seemed to him some such treatment would have done good. One patient had had varices, and the operation was done for uncomplicated appendicitis. Five days after the removal of the appendix the patient suddenly died of pulmonary thrombosis. Another case, three years ago, was also one of appendicitis, in which a concretion was found. There was no suppuration, and the concretion was removed, but the patient died about the tenth day of pulmonary embolism. Would citric acid or the application of leeches be likely to obviate such a calamity?

Dr. ESSEX WYNTER replied that he thought one could control the coagulation of the blood by immediately applying leeches. The question was whether it was worth while applying such an unpleasant remedy in a hundred cases to prevent one instance of thrombosis. His suggestion had reference to preventing the spread of thrombosis which there was reason to infer had already commenced. When he did it he always had a bandage put on the leg, in case a separate clot might travel. He had now an unusual case, in which he thought there was sclerosis of the retroperitoneal tissue involving the vena cava: the patient had already had thrombosis in the right leg. A leech was applied and the œdema disappeared in two or three days. A week later similar symptoms, pain and swelling, occurred in the left leg and subsided in the same way after a leech had been applied.

Case of Ulcerative Colitis, terminating Fatally.

By P. LOCKHART MUMMERY, F.R.C.S.

F. R., A MAN, aged 49, a baker by trade, had diarrhœa and bleeding from the rectum for the last three years, but became worse during the last three months before his admission to the hospital. He also had vague abdominal pains, felt very weak and ill, and had lost weight to a considerable extent. The stools varied from eight to ten in twenty-four hours, and were usually blood-stained. He was admitted to St. Mark's Hospital on January 25 this year. He then had a slight chronic cough, and on examination there were slight signs of tubercle of both apices. He had well-marked pyorrhœa alveolaris. He was very thin, tender over the colon, and was passing a considerable number of stools during the day containing much mucus and blood. A sigmoidoscope examination revealed extensive ulceration of the bowel as far up as could be seen. On January 27 the abdomen was opened in the middle line and the colon thoroughly examined. It was found to be thickened, and numerous ulcers could be felt, but no other lesion. The appendix was brought out through a stab puncture in the right iliac fossa, and an appendicostomy opening established. From this time the bowel was washed out repeatedly with water and solutions of argyrol, 1 per cent. There was some improvement for a time, and the wounds healed by first intention. Soon, however, the skin round the appendicostomy opening became ulcerated, and a slow form of ulceration gradually progressed in this situation, and ate away a large portion of the skin, and exposed the muscles and fascia in the abdominal wall. Nothing that could be done would prevent this ulceration from spreading, and soon there was an ulcer as large as half-a-crown round the opening. The patient's condition varied a good deal during the subsequent weeks. At the beginning of March he was certainly picking up, putting on flesh, and improving generally; but on March 5 he complained of pain in the chest, and on examination there was dullness at the left base. His pulse became rapid, signs of bronchial pneumonia set in, and he died.

At the post-mortem examination there was well-marked bronchial pneumonia at both bases and scattered areas of consolidation. The liver, spleen, and kidneys were normal. On opening the peritoneal cavity there were no signs of chronic peritonitis, no adhesions, no enlarged glands; the whole of the ileum was perfectly normal. The colon was cut out, including the cæcum and rectum, and specimens are exhibited here this evening.

Very extensive ulceration was found throughout the colon, much more marked in the lower half. Over large areas the mucous membrane was entirely destroyed down to the elastic coat, with only a few islands of mucous membrane which stood up like polypi. Other sections showed well-marked, irregular-shaped ulcers of the most characteristic variety. In many parts of the specimen the appearance at first sight is that of multiple polypi of the bowel. This is, however, an artificial effect produced by the complete erosion of the mucous membrane, leaving small islands of mucosa, usually of a circular shape, and standing up exactly like polypi from the surrounding ulcerated area. It is noticeable that towards the cæcal end of the colon there is marked evidence of healing of many of the ulcers, no doubt due to the fact that this part of the colon, owing to the appendicostomy opening, was enabled to be kept much cleaner than the remainder; in fact, the ascending colon and cæcum show very little ulceration, although there are numerous scars of healed ulcers. The ulcerated condition in many parts of the colon is so extreme that it would appear to be impossible for the patient to have recovered.

As regards the bacteriology of the condition, owing to the large number of organisms usually found in the stools in these cases it is seldom possible to ascertain with any certainty the particular micro-organism which is producing the lesion. In this case, however, I think we can be fairly certain of the particular offending micro-organism, although we were not able to ascertain this by the ordinary culture methods. The nurse who was looking after this patient contracted a bad whitlow of the finger, and was for some days threatened with septicæmia. She was found to be suffering from a pure infection of *Staphylococcus aureus*, and it seems to me that there is every reason to suppose that this was the micro-organism causing the ulceration in the colon of the patient. Examination of the fæces for tubercle bacilli failed to produce evidence of the ulceration being tubercular.

This case appears to me to be of interest chiefly for two reasons. It is one of the very few cases I have met with in which the patient has not recovered from ulcerative colitis after an appendicostomy. Had the operation been performed earlier I believe this patient would have recovered. As a rule such cases, even where there is very extensive ulceration, rapidly recover when the bowel is kept washed out, and it is obvious that in this case there was a very marked tendency to healing, although the ulceration was too severe for it to be complete. The other reason why I have ventured to bring forward this case is because the specimen in places so closely imitates the condition of multiple polypi of the colon, although its pathology is of quite a different nature. At first sight one might easily suppose that the specimen was one of multiple polypi unless one examined microscopic sections.

I may say that the patient was never out of England, and that this is a typical instance of acute ulcerative colitis.

DISCUSSION.

Mr. LAWRIE McGAVIN said that one of the points which interested him was the occurrence of the ulceration around the appendicostomy opening. Five years ago a case was admitted to the Seamen's Hospital, Greenwich, not with ulcerative colitis, but with a dysenteric abscess of the liver, and the patient had had dysenteric ulceration of the rectum. The abscess of the liver was apparently being drained satisfactorily, when suddenly the same kind of ulceration as Mr. Mummery mentioned in his case took place. It was the worst condition of the kind he had ever seen; nothing stopped it; it ate away the skin and the fascia and laid the muscles bare. It reached from the nipple in front to the spine behind, and the area involved was of the size of an average breakfast plate. Sections were taken and cultivations made, but no cause was discovered. Various micro-organisms were found, among them the *Staphylococcus aureus*, but the condition could not be attributed to any particular one. The patient died from asthenia more than from anything else. Mr. Mummery mentioned that his patient had a slight chronic cough, and on examination both apices were found to be tuberculous, and he asked Mr. Mummery if he found that cases of pulmonary tuberculosis were common in association with ulcerative colitis, death from this cause having occurred in a case of his own some years previously.

Mr. MAYNARD HEATH said he had had a similar case, but when he did the appendicostomy the patient recovered and went to a convalescent home. She seemed so well that the appendicostomy was allowed to close. She left the home, but came back as she had a relapse. He reopened the appendicostomy, but this time the case went in the same way as Mr. Mummery's: the disease progressed, there was ulceration round the appendicostomy opening and into the ischio-rectal fossæ. But his patient had not pyorrhœa alveolaris. Mr. Mummery did not say whether he had anything done to the mouth of his patient, but he supposed he had the teeth extracted so as to stop the supply of micro-organisms to the bowel. That would be quite an important part of the treatment.

Fleet-Surgeon BASSETT-SMITH remarked that Mr. Mummery spoke of the large number of organisms found in the intestinal canal. That was always so. But in cases of this kind with ulceration there were always septic pathogenic organisms present in variety, as well as the one which actually caused the disease, which was possibly Flexner's bacillus. He thought it probable that *Staphylococcus aureus* caused the serious infection of the nurse's finger, but probably the first outbreak in the case was due to some other organism of the colon group.

Mr. LOCKHART MUMMERY, in reply, said the only other instance of ulceration of this type he had seen was in a case of liver abscess, in which ulceration started round the drainage-tube. The way the ulceration in this case began and continued made him think that the skin was being digested by gastric or pancreatic juice. An attempt was made to correct this by altering the reaction of the wound, but without effect, so it seemed probable that it must have been due to a specific micro-organism. Everything which could be thought of was done to stop the ulceration, but without success. It was difficult to say whether the primary lesion was tuberculous or not; sometimes such a condition did occur in people who had phthisis, due to the patient swallowing sputum loaded with tubercle bacilli. Later the colon bacillus, or some form of staphylococci, invaded the tissues, already injured by tubercle, and the tubercle bacilli were killed off. He did not doubt that many of these cases were tuberculous at the start. In answer to Mr. Heath, he said that everything was done to correct the patient's pyorrhœa; the bad teeth were removed, and the mouth carefully cleansed.

Clinical Section.

May 2, 1913.

Mr. W. G. SPENCER, Vice-President of the Section, in the Chair.

Old Quiescent Zoniform Sclerodermia of the Right Lower Extremity.

By F. PARKES WEBER, M.D.

THE patient, A. J., a German married woman, aged 44, well developed but rather thin, has a hard depressed band, feeling as if due to a cicatricial change in the subcutaneous tissue, situated lengthwise on the right thigh; the surface of the skin over the band is somewhat shiny, but of natural colour, and there are no telangiectases. This band, from its point of commencement above the great trochanter (between the great trochanter and the iliac crest), passes inwards, just below and parallel to Poupart's ligament, and then extends in a straight line downwards on the anterior inner aspect of the thigh to end close above the knee (*see* diagram). The case is a typical one of localized zoniform sclerodermia (the "morphœa herpetiformis" of Sir Jonathan Hutchinson) of characteristic band-like distribution ("sclerodermia in stripes"), resembling that of zona (herpes zoster), when one of the limbs instead of the trunk is affected. The parts below the knee in the present case are not involved, but in the loin of the right side there is a depressed area of sclerosed subcutaneous tissue of the size of a five-shilling piece close to the spinal column, above the iliac crest and just below the false ribs. The sclerodermia in this case was apparently first noticed at the age of 11 or 12, and, according to the patient, has not progressed since she was aged 18. On the contrary, it has perhaps undergone some involution. It may be mentioned that when the patient was aged 18, about the time when the sclerodermia ceased to progress, she underwent an operation for some diseased bone in the right tibia, just above the ankle.

In May, 1911, a gastro-enterostomy was performed on account of carcinoma of the pylorus and mesentery, which was too extensive for operative removal. From May to September she was treated for her abdominal malignant disease, with great benefit, by X-rays (Dr. G. Dorner). Unfortunately, some X-ray burning occurred on the front of the abdomen in connexion with the otherwise successful treatment. This soon healed up, but in March, 1913 (after an interval of half a year), whilst the pelvic portion of the abdomen was being subjected to X-rays, a patch of very painful chronic induration, with telangiectases and superficial necrosis (apparently a kind of "late X-ray burn"), gradually developed in the anterior abdominal wall, near the site of the previous X-ray burning.

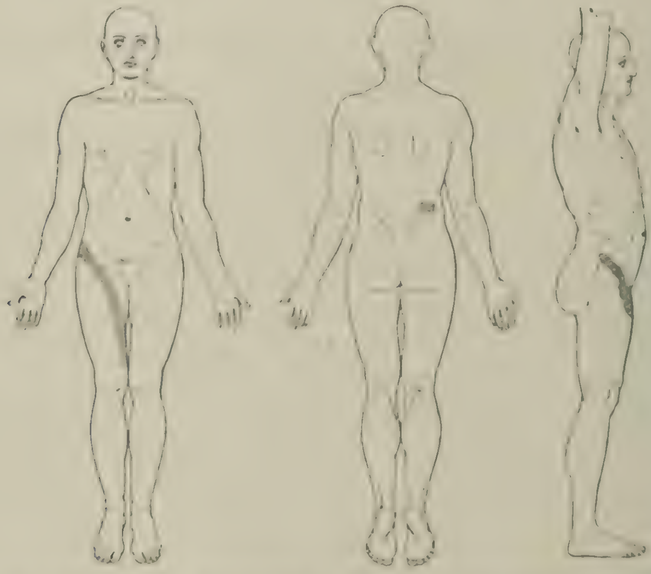

Diagram illustrating the distribution of the zoniform sclerodermia in the case of A. J. The shaded portions show the sclerodermatous areas.

DISCUSSION.

Dr. GALLOWAY said that he agreed with Dr. Weber's diagnosis, though the case was an unusual one. In the type of sclerodermia in which the atrophic lesions of the skin were distributed in stripes round the body or on the extremities, like zoster, the amount of recovery was usually very slight. In the type of disease, however, which is also known by the name of sclerodermia, in which the skin is more or less universally affected with swelling of the tissues, rather than of atrophy, the amount of recovery was often very considerable. Both classes of patients, especially the latter, benefited very much from carefully regulated massage. The X-ray burn in this case complicated treatment and served as a warning, which seemed to require frequent reiteration, of the dangers following X-ray exposure.

Mr. MIDELTON asked whether Dr. Weber had noticed a more rapid involution since the X-rays were used.

Dr. WEBER replied that he had never used X-rays for the sclerodermia.

Intermittent Claudication of the Right Lower Extremity in a Young Man whose Business has been to Work a Treadle Machine.

By F. PARKES WEBER, M.D.

IN this case the disease giving rise to the arterial obstruction and the consequent "intermittent claudication" is doubtless the same (non-syphilitic arteritis obliterans, "thrombo-angeitis obliterans" of Leo Buerger) as in the two other patients with intermittent claudication whose cases I have already demonstrated this year at this Section.[1] The subject of the disease this time is, as usual, of the male sex, in the first half of life, a Russian Jew and a cigarette smoker, and, as usual, there is no evidence of previous syphilis, but the present case is specially interesting on account of the almost "ultra-typical" early onset of the disease, its characteristically slow progress, and on account of the constant working of a treadle having possibly acted as a determining factor.

The patient, L. K., a tailor, aged 24¾, is of Russian Jewish parentage and was born in Moscow, but was only aged 2½ when he was brought from Russia to London. In regard to this patient, therefore, as he left Russia at such an early age, the cold winters and dietetic conditions of his childhood in Russia can hardly be invoked, as they sometimes have been in the case of other patients, to explain the special tendency of poor Russian and Polish Jews to fall victims to the early arterial disease in question. He is a well-developed man of moderate general nutrition and rather dark and sallow complexion. His height is 5 ft. 6 in., and his weight 9 st. 12¾ lb. in ordinary clothes. His only complaint is the trouble with the right lower extremity. When walking he has to stop every 25 yards or so on account of cramp-like pain in the muscles of the foot and calf of the affected limb. The pain goes off immediately when he rests, and he can then walk another 25 yards before he has to stop again on account of fresh pain. If he rests again for a moment he can walk a little farther, and so on. He has sometimes tried to "out-walk" the pain, but on each occasion when he has tried the pain has got the better of him and forced him to stop. The right foot always becomes pale and cold when he walks, or forcibly exercises the ankle-joint in any way, but when he allows it to hang down in a dependent position it always shows a red flush as compared to the left (normal) foot. The right foot, moreover, often feels colder than the left foot, even when he is resting. Sometimes he wakes up in the night with a cramp-like pain in the toes and whole right foot, and he says that on such occasions he has been able to obtain relief by allowing the right leg to hang out over the side of the bed from the knee downwards. There is no pulsation to be felt in the right arteria dorsalis pedis, though there is good pulsation in the left one. Pulsation in both femoral arteries at the groin is good. The right calf is a little smaller than the left calf; on measurement a difference of ¼ in. in girth can be detected. No calcification in the arteries of the right leg can be detected by Röntgen ray skiagrams. But the "Moszkowicz test" confirms the other signs of arterial obstruction in the right leg.[1]

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[1] See *Proceedings*, p. 72 and p. 162.

When the left lower extremity has been rendered anæmic by Esmarch's apparatus, the hyperæmic reaction in the foot follows almost instantaneously after removal of the apparatus, but on the right side the return of blood after removal of the apparatus is slower and irregular (blotchy), the wax-like pallor lasting two minutes at some spots of the right foot before giving place to the redness of cutaneous hyperæmia.

No evidence of vascular disease in other parts of the body can be detected. Brachial systolic blood-pressure, 125 mm. Hg. Examination of the mouth and the thoracic and abdominal viscera, and of the urine, shows nothing abnormal. The thyroid gland is of natural size. There is no enlargement of liver or spleen, or of superficial lymphatic glands. The grasp in both hands is very good. The patellar and Achilles reflexes are normal on both sides, and so also are the plantar, cremasteric and abdominal reflexes. The pupils are equal and react naturally to light and accommodation. Ophthalmoscopic examination (Dr. C. Markus) shows nothing abnormal in either eye. Blood examination (Dr. Bauch): Red cells 5,200,000 and white cells 10,500 to the cubic millimetre of blood; hæmoglobin 80 per cent. The blood serum (May, 1913) gives a negative Wassermann reaction for syphilis.

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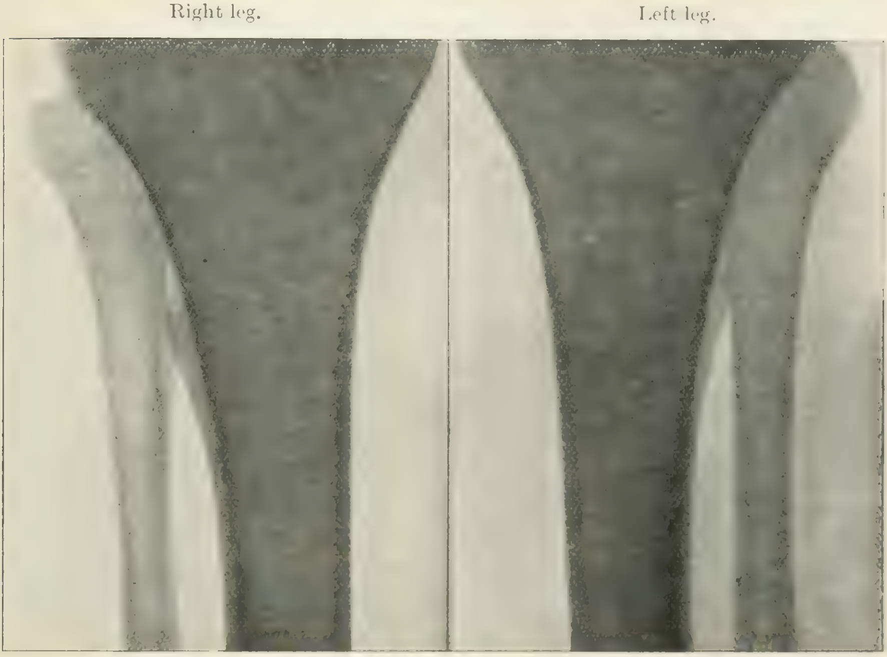
[1] *See* Ludwig Moszkowicz, "Die Diagnose des Arterienverschlusses," *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, Jena, 1907, xvii. p. 216.

There is nothing special in the family history, and in regard to his own past history the patient says he has had no other illnesses, except one attack of gonorrhœa, five years ago. He married two and a half years ago, and has one child, living and healthy. His wife has had no abortions or miscarriages. The beginning of the present illness was two and a quarter years ago, when, one day, on coming home from work, he felt a cramp-like pain in the calf of the right leg. Since then the symptoms of intermittent claudication have gradually become worse. For the first year the pains were limited to the calf, but then pains in



Skiagrams (May, 1913) of the legs in the case of L. K. They seem to show considerable periosteal thickening in the upper angle (just below the knee) between the tibia and fibula, proceeding from both bones in the right leg, but, in the left leg, from the tibia only. In all probability the condition causing this very unusual skiagraphic appearance is not due to disease. There is no evidence on either side of any arterial calcification.

the foot occurred as well, and he used to notice the already described blanching of the feet on exercise. A year ago he could walk 300 yards without having to rest, but now he can only walk 25 yards without stopping. He smokes cigarettes only, and for the last nine years has been accustomed to smoke about twelve daily. There is nothing specially noteworthy about his diet, and he has always been moderate in regard to alcohol. In his business he formerly always worked with a treadle (sewing) machine, but after nine and a half years he had to give it up (fifteen months ago) on account of his present disease. He now does tailoring work with his hands only.

Purpura Hæmorrhagica, a Fulminating Case; Gelatine Treatment.

By F. Parkes Weber, M.D.

The case is a fulminating one, though not an example of Henoch's fatal type of "purpura fulminans." The patient, L. S., a well-built boy, aged 13, was admitted under my care at the German Hospital, on April 17, 1913, with the history that he had enjoyed good health until three days previously, when the present trouble commenced. On admission the boy was covered with petechiæ and ecchymoses varying greatly in size and from red to blue-black in colour. The darker petechiæ and larger ecchymoses were especially numerous on the lower extremities. There were many submucous petechiæ and ecchymoses in the mouth and pharynx; and there was a sub-conjunctival hæmorrhage over the sclerotic of his left eye. The urine passed on admission looked like slightly diluted blood with a little brownish colouring matter superadded; the specific gravity was 1022, and the reaction neutral; microscopical examination showed nothing special except blood corpuscles. There was blood in the fæces (melæna), when examined on April 18. Examination of capillary blood (Dr. Bauch) on admission: Red cells 2,360,000 and white cells 7,000 to the cubic millimetre of blood. Differential count of white cells: Lymphocytes, 26·1 per cent; monocytes, 1·9 per cent; polymorphonuclear neutrophiles, 69·8 per cent; eosinophiles, 1·1 per cent; no mast cells; myelocytes, 1·1 per cent. During the count of white cells two nucleated red cells (both normoblasts) were seen. There was no poikilocytosis. The erythrocytes appeared natural with the exception of very slight anisocytosis and polychromatophilia. By examination of the heart and lungs, and by palpation of the abdomen, nothing abnormal was discovered. There was no enlargement of the liver, spleen, or superficial lymphatic glands.

On admission I gave him a subcutaneous injection of gelatine (16 c.c. of Merck's "gelatina sterilizata"), and he was ordered to have milk, fruit, fresh vegetables, eggs, fish, &c., as well as jelly made from gelatine in his diet. The improvement in his condition was rapid and striking. On April 19 there was much less hæmaturia, and the urine passed on the afternoon of the following day was quite free from blood and albumin. The subcutaneous hæmorrhages had in great part disappeared by April 27, and hardly a trace of the petechiæ and ecchymoses in the mouth remained. It should be mentioned, however, that though no retinal hæmorrhages were seen on admission (Dr. C. Markus), some appeared on April 20, one large one on the inner side of the optic disk of the left eye giving rise to loss of the outer part of the visual field in that eye. These retinal hæmorrhages are likewise clearing up and his vision is now practically normal in every way in each eye. The red cells in his blood (May 2) have already risen to 4,000,000 in the cubic millimetre; hæmoglobin 55 per cent. Dr. G. R. Ward, who has kindly examined a blood film, regards the appearances as characteristic of the condition usually met with some time after hæmorrhage, the variations of the erythrocytes in shape and size, and staining property, being greater than in normal blood. The patient's temperature, from his admission on April 17 to April 22, was often up to about 100° F., but since that date there has been no fever.

Case of Pulmonary Tuberculosis after Gymnastic Treatment.

By Filip Sylvan, M.D.

W. G., aged 24. Shown at the March meeting.[1] The whole left lung was affected, and there was a cavity below the clavicle. The upper part of the right lung was affected. After ten weeks' treatment, the activity of the disease had, in Dr. Sylvan's opinion, ceased and the patient was fit for work. Vital capacity when starting treatment, 440 c.c.; now 2,650 c.c.

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[1] See *Proceedings*, p. 183.

DISCUSSION.

Dr. SYLVAN said that, judging from similar cases that he had had, the patient would remain able to work. He had increased in weight 2½ lb., though it was not usual for the patients to gain much weight during the treatment. Some had gained more at the conclusion of the treatment, but they had all remained well and fit for work.

The CHAIRMAN (Mr. W. G. Spencer) said that as there was a serious difference of opinion, he would ask Dr. Sylvan to report later on on the further progress of the case.

Functional Aphasia.

By HECTOR MACKENZIE, M.D.

THE history is as follows: Her husband was admitted to St. Thomas's Hospital on April 20 with a right-sided hemiplegia and complete motor aphasia of two hours' history; since then she had worried considerably and had not slept. On April 23, on getting up, she found she could not speak at all. I saw her on April 25, when there was complete motor aphasia. On April 27 she was better, and could talk fairly well; on April 28 she was talking normally, but in the evening was upset at home, and the aphasia again became complete. I have seen her to-day and she is talking a little, and the friend with her tells me that when outside the hospital she is talking quite normally. On eight or nine previous occasions, the first being two years and the last two weeks ago, she has similarly been aphasic, but never for more than a few hours at a time, the attacks always occurring after some special worry. She seems of a very nervous disposition, but her past history does not otherwise present any special features. She is aged 35, and has been married fifteen years, and has one child, aged 15, and had three miscarriages since.

DISCUSSION.

Dr. LANGMEAD recalled an instance of the condition in a man, in which case it was also brought on by shock. The patient was working with a fellow-labourer, who fell off a scaffold, whom he conveyed to the hospital. On arrival it was found that the injured man had died. The patient on hearing the news was literally "struck dumb," making only inarticulate sounds on attempting to speak. The condition lasted about three weeks.

Dr. F. Parkes Weber asked, in view of the patient having had three miscarriages, whether a Wassermann reaction for syphilis had been tried, and the reply was in the negative. Dr. Weber therefore suggested that syphilis should as far as possible be excluded before the diagnosis of "functional aphasia" was definitely arrived at. Temporary, and sometimes recurrent, attacks of aphasia might occur as premonitory symptoms of grave, and even fatal, syphilitic disease of the blood-vessels of the brain. He alluded to the case of a young man in whom an attack of temporary aphasia was followed some months later by fatal syphilitic thrombosis of the basilar artery.

Case of Complete Heart Block.

By G. H. Hunt, M.B.

The patient, an unmarried woman, aged 19, had typhoid when she was aged 6, but gave no history of rheumatism. All her life she seemed to have been quite free from any attacks of syncope or other symptoms of heart disease. She was admitted into Guy's Hospital for an operation for hammer-toe. On admission she was a well-developed, healthy-looking woman; she was in no respiratory distress, and her appearance did not suggest the presence of any cardiac lesion. The pulse was regular and of good volume and normal tension, and the artery was not thickened. The apex beat was in the fourth space in the nipple line, and there was a mitral systolic murmur. There were no physical signs of disease in the lungs. The urine was normal and the liver was not enlarged. There was no anasarca. The pulse-rate while she was in bed was usually about 50. Electrocardiograms were taken and showed that complete heart block was present; the auricles and ventricles were both beating quite regularly and independently, the auricular rate being 120 per minute, the ventricular 64.

The case presents some unusual features: In the first place, it is interesting that the condition should have been discovered quite accidentally. Careful inquiries were made into the patient's previous history, but the condition of her heart does not seem to have given rise to any symptoms whatever; this is particularly remarkable, since she is suffering from a form of heart disease usually considered to be of a serious nature. Secondly, the pulse-rate is considerably faster than that commonly encountered in cases of complete heart block; the usual rate is about 30 per minute, although rates as high as 80 have been recorded. This rapid ventricular rhythm would seem to point to a favourable prognosis. The epileptiform seizures of Stokes-Adams disease are probably due to temporary cerebral anæmia produced by the slow ventricular action, and consequently patients with a comparatively rapid ventricular rate are less liable to these attacks. Thirdly, the ætiology in this particular case is not quite certain. Rheumatism is the only common cause of heart block in patients of this age, and the absence of any history of joint pains does not exclude this origin. On the other hand, this patient has had typhoid fever, and there is a case of heart block recorded occurring in this disease.

The operation on the hammer-toe was performed under local anæsthesia.

The patient was under the care of Sir Cooper Perry, and I wish to express my thanks to him for permission to describe her case.

Dr. GALLOWAY remarked that with the exception of a case shown two and a half years ago,[1] he thought that this was the youngest case of heart block which had been shown before this Section. The case to which he referred occurred in a boy aged 18, with an apparent radial pulse-rate of about 32. It was interesting to recall the fact that he had been asked to see this patient, because he was supposed to have *petit mal*. The attacks of fainting had occurred frequently, especially on even slight exertion. At the discussion which followed on this case the possibility that the auriculo-ventricular connecting bands had been damaged by syphilitic lesions of congenital origin had been suggested, and the patient had been carefully treated by means of iodide of potassium. The boy improved greatly afterwards, and he understood had been able to return to his occupation. Dr. Galloway suggested that it would be very advisable to have Dr. Hunt's case investigated from this point of view, and that at any rate treatment by iodide of potassium, if not by mercury or arsenic, should be used.

Case illustrating the Prognosis of Extrasystoles.

By G. H. HUNT, M.B.

THE patient is a medical student, aged 23. He has been under the care of Dr. Fawcett, and I must thank him for permission to describe his case. The patient gives no history of rheumatism. He has been in the habit of taking vigorous exercise all his life; he used to row and play Rugby football fairly regularly. He is not a heavy smoker. Except for minor ailments he enjoyed good health until April, 1911, when he was advised to give up rowing because his heart was irregular. A year later he consulted Dr. Fawcett for indigestion.

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[1] "Stokes-Adams Symptoms in a Lad, aged 18," James Galloway, M.D., and W. J. Fenton, M.D., *Proc. Roy. Soc. Med.*, 1911, iv (Clin. Sect.), p. 39.

On examination it was found that his heart was dilated, the apex being in the fourth space in the nipple line. There were no murmurs, but the first sound was prolonged. The pulse was of good volume, but very irregular. Radial and jugular tracings showed that this irregularity was due to the presence of extrasystoles of ventricular origin, and this was subsequently confirmed by electrocardiographic examination. There were no physical signs of disease in any of the other organs. The dyspepsia was treated, and the patient was instructed to lead a quiet life and to avoid all violent exertion.

In July, 1912, he was examined again, and it was found that the heart had diminished in size, but that the pulse was still irregular. At this time the extrasystoles were occurring after every fourth or fifth beat. During the next few months he took rather more exercise, and occasionally played a game of rackets. Tracings taken directly after a game, while the pulse-rate was still about 120, did not show the presence of extrasystoles, but they reappeared a few minutes later when the pulse-rate had fallen. This amount of exercise did not cause the heart to dilate again. Tracings were taken on several occasions during this period and the frequency of the extrasystoles did not seem to be appreciably affected. During December the patient trained for Rugby football, and started playing in January, and continued to do so regularly for two and a half months. Towards the end of January he was examined again; the heart was of normal size and the extrasystoles were still present. By the middle of March, however, the pulse had become absolutely regular, all the extrasystoles having disappeared. There was no dilatation, and the heart appeared to be absolutely normal.

The case is of interest because of its bearing on the treatment and prognosis of this form of irregularity. It has been recognized for some time that in the absence of other signs of heart disease extrasystoles are not of any serious significance and are no contra-indication to a certain amount of exercise. On the other hand, their presence does indicate that the heart is not acting quite normally, and in such cases there is naturally some hesitation in subjecting the heart to severe strain. This is particularly the case with Rugby football, as this game probably entails greater muscular exertion than any other, and not infrequently causes cardiac trouble in men whose hearts were perfectly healthy beforehand. In the case under consideration, however, the heart does not appear to have been in any way injured; indeed, it seems as if the regular training has had a beneficial effect.

Dr. F. PARKES WEBER said there were many cases in which an ordinarily irregular action of the heart became regular on suitable muscular exercise of the body. Dr. James Mackenzie, in particular, had drawn attention to the whole subject. He (Dr. Weber) thought those cases might be compared roughly to cases of functional nervous disease in which some organic disease arose. In such cases sometimes the functional nervous phenomena, for the time, vanished completely. It was a very old observation that in such cases "morbus dissipat spasmos," the organic disease dispels or disperses the functional. The demand made by acute febrile diseases on the body often for a time completely banished functional nervous irregularities of various kinds. So, in psychical life, the necessity for thorough and immediate action often banished morbid doubts and irregularities.

Renal Calculi in both Kidneys.

By PHILIP TURNER, M.S.

E. G., AGED 42, was admitted on January 18, 1913, for pain in both loins and pyuria. The trouble was first noticed eighteen years ago when she passed two small stones and pus was found in the urine. Four years after this she was admitted to the St. Albans Hospital, where another stone was passed. She then remained well for four years, when another stone the size of a date-stone was passed. The patient then continued in good health until August, 1912, since when she has had pain, worse on the left side, pyuria, and hæmaturia. On admission both kidneys were much enlarged, extending downwards into the iliac fossæ. On the left side a very large calculus could be distinctly felt, while on palpating the right kidney numerous stones could be detected and a sensation like crepitus was readily obtained. The urine was acid in reaction and contained much pus, blood, and albumin. She was very thin, pale, and cachectic. A radiographic examination showed numerous shadows in the right kidney and a large dark area in the left. On January 23 the left kidney was exposed and a large calculus, weighing, when dried, 4 oz., and several smaller ones were removed; two large abscesses containing foul pus were also opened and drained. She stood the operation well and no serious symptoms followed. Four weeks later the right kidney was exposed and seventeen calculi and a number of small ones were removed, their total weight being 4 oz. The pelvis of the kidney was distended with foul turbid urine. The patient made good progress, and was discharged on March 13.

Though still a bad colour the patient has put on flesh, and is much better and stronger. The urine is now acid in reaction, contains no blood and only a trace of albumin. The wounds are firmly healed and the kidneys appear to be about normal in size.

DISCUSSION.

Mr. CHARTERS SYMONDS congratulated Mr. Turner on the successful issue in this case. Bilateral nephrotomy had been done before, but he was not acquainted with a case where such large calculi had been removed. These very large calculi were found sometimes accidentally in autopsies on people who died from other causes, and it was remarkable how people could go about with calculi, and yet enjoy perfect health. He was reminded of a case in a lady, aged 65, who had a great deal of pain on the left side, with pyuria, and although she was in very poor health, he cut down on the left kidney, and found an enormous stone, much larger than either of the specimens now exhibited. Knowing, from the character of that stone, that there was probably one on the opposite side, he intended to remove the calculus, but on account of the severe hæmorrhage from a large vessel which was accidentally torn, he was compelled to remove the kidney. From that operation she recovered, but symptoms arose on the other side, which had hitherto been free from pain, and death ensued from renal failure.

The CHAIRMAN (Mr. W. G. Spencer) said the case was very important, as showing the care which the surgeon must exercise when operating on one kidney, unless he was quite certain as to the condition of its fellow. It had often been proved that the opposite kidney was even more diseased. He had a case in which the second kidney was not affected with such advanced disease as in this case, but the patient had for twenty years been treated on mistaken lines. He was supposed to have cyclical albuminuria, probably because the urine had not been centrifugalized. Hence there had been a difficulty in regard to insuring the patient's life; the policy had been refused. He had continual pain in the right kidney region, and it was conjectured that he had appendicitis. Twenty years afterwards he was suddenly seized with what was supposed to be intestinal obstruction. But fortunately another doctor, reviewing this long illness, had a skiagram taken, and this showed that there was a stone in the left kidney, blocking the pelvis, and threatening suppression of urine, while the opposite kidney was filled with a stone the size of that in one of Mr. Turner's cases. He (the speaker) was forced to operate first on the better kidney, to take the stone away. The patient was now alive, several years afterwards. But, as in Mr. Turner's case, the urine remained deficient in quantity, although the patient's health seemed to be preserved. Many of these cases did not pass the normal quantity of urine. Recently his colleague, Mr. Arthur Evans, was operating on a case for what he thought was malignant disease of the kidney, and it seemed as if he could remove the kidney; but before doing so he passed his hand across the abdomen, and a careful search failed to reveal another kidney. It proved to be malignant disease in a patient who had only one kidney, but by not removing it the patient had two or three months more of life.

Mr. C. H. FAGGE asked whether Mr. Turner opened the pelvis to remove these stones, or whether he incised the kidney along its outer convex surface through the cortex. Nephrotomy, which was the method formerly in vogue, seemed now to be supplanted by pyelotomy, as the latter did no damage to renal tissue. This advantage would appear to be of some importance when there is little active renal tissue. Some years ago he did nephrolithotomy on a case in which subsequent hæmorrhage from the incised kidney necessitated his colleague, Mr. Steward, removing the kidney. Mr. Gilbert Barling had recorded two similar cases. Though in Mr. Turner's case the renal cortex was so thin as to be probably nothing more than fibrous tissue, one should do everything possible to conserve that amount of secreting substance which still persisted. He therefore asked whether the kidney cortex was incised in this case, and, if it was not, whether it would have been possible to incise the pelvis.

Congenital Lipoma of the Sole of the Foot with Hypertrophy of the Second and Third Toes.

By J. G. SANER, F.R.C.S.

H. C., AGED 2, was seen at Guy's Hospital by Mr. Rowlands, when 6 weeks old. The condition had been noticed since birth. The tumour appears to have grown at the same rate as the foot. Operative interference was postponed owing to ill-health.

Congenital Hypertrophy of the Right Leg—Scoliosis.

By J. G. SANER, F.R.C.S.

GIRL, aged 13, shows the right leg generally larger than the left. The hypertrophy appears to affect all the tissues, and gives the child an adult appearance on the affected side. There is a general nævoid condition of the skin. It has been tentatively suggested that a resection of some inches of the shaft of the right femur, followed by plating, should be tried. Further suggestions are invited.

DISCUSSION.

Mr. SANER added, in reference to the first case, that the question was one of operative interference, whether it would be possible to remove that lipoma completely, or whether it was likely to recur. It would be difficult to remove all the tumour tissue as the muscular planes were infiltrated. With regard to the second case, he did not know that the diagnosis was quite certain, but he would be glad of opinions.

Mr. SYMONDS said it would add to the elucidation of the second case if a more careful comparison were made between the two femora. The disparity in length on the two sides might be due to alteration on the left, rather than to enlargement of the right side. He suggested measuring the bones, and having the comparison noted in the statement of the case.

Dr. F. PARKES WEBER said he had seen several cases of congenital hypertrophy of a limb of the same kind as Mr. Saner's second case. He did not doubt that the hypertrophy in Mr. Saner's case was a real hypertrophy, and that it involved the bones. This type of hypertrophy, which involved the bones, had to be differentiated chiefly from two other conditions, in one of which (cases of so-called "trophœdema") there was enlargement of the soft parts of the whole limb, or of one section of the limb, but no enlargement of the bones, as evidenced by X-ray examination. The other condition was true "gigantism," in which the bony enlargement was very great and generally accompanied by irregular bony projections or bosses. In regard to the present case shown by Mr. Saner, Dr. Weber preferred the term "congenital hæmangiectatic hypertrophy of a limb," because the kind of hypertrophy in question (in which the bones *always* shared) was nearly always accompanied by some form of local exuberance of blood-vessels, shown by the presence of capillary or venous angiomata or, more rarely, by excessive size of the main arteries of the limb or by the presence of so-called "congenital or early developmental varicose veins." Another good term for the same kind of hypertrophied limb was "Congenital or developmental hypertrophy of a limb *of the hemi-hypertrophy type*"; for this kind of hypertrophy was undoubtedly very similar to that affecting the whole of one side of the body in cases of hemi-hypertrophy. Hæmangiomata, it might be recalled, were notoriously frequently present in the subjects of hemi-hypertrophy.[1]

Dr. LANGMEAD said he had seen a family of three, with congenital lipoma of the sole of the foot, and he asked whether there was a family history of the condition in this case. One of the patients had been thrice operated upon, an evidence that the condition recurred. The condition was a very interesting one, especially prone to recur, and often requiring very extensive operative procedure to eradicate it. Amputation away from the tumour was called for.

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[1] *See* F. P. Weber, "Angioma Formation in connexion with Hypertrophy of Limbs and Hemi-hypertrophy," *Brit. Journ. Derm.*, 1907, xix, p. 231.

Mr. C. H. FAGGE said that some years ago he operated at the Evelina Hospital on a child with a similar condition on the inner side of the hand. He had recently seen the child, ten years after the operation, for a minute nævoid patch in the scar, but there had been no recurrence of the subcutaneous tumour. Two years ago he was asked to see a child at a suburban hospital who had an enormous great toe, which was obviously due to increase of subcutaneous tissue: the skiagram showed the bones were normal. He advised amputation of the big toe, and this was carried out by the doctor in charge. Three months ago he was again asked to see the child with a very large second toe, and the lipomatous mass had extended into the sole of the foot. There was also a large area over the tibialis anticus in which the same change had taken place. He treated it by amputation of the second toe, with removal of the mass in the sole. He also did an extensive operation on the front of the leg, resecting the skin and subcutaneous swelling so extensively that he failed by $1\frac{1}{2}$ in. to get the skin together. There was some difficulty in defining the limits of the fatty tumour, though perhaps the abnormal fat was more pink than normal owing to excessive vascularity. He supposed that amputation through the knee-joint would probably be the final operation in this case; but it seemed almost criminal to do this now when the child had a useful ankle and leg. These cases showed with Mr. Saner's a pathology which was probably common to all these fatty tumours of congenital origin—they showed overgrowth of blood-vessels, fat and lymph vessels, and usually the lymphangiomatous change was the most important and the most difficult to eradicate.

The CHAIRMAN said he could confirm what Mr. Fagge said, that the determining factor in many of these cases was the lymphangiectasis, which tended to extend. Some time ago at hospital he did a Pirogoff's amputation in a case of almost exactly similar condition, and the disease seemed to be arrested. In a second case some time afterwards there was a more advanced condition in the foot. The parents brought the child up at intervals, but they continued to refuse amputation. It gradually spread, becoming a solid growth, and the child died with a small-celled sarcoma, involving the pelvis. He suggested to Mr. Saner that there was some lymphangiectasis, which if left would gradually spread.

Mr. SANER replied that, in the first case, there was no history of other members of the family being affected with the same condition. With regard to the second case, in answer to Mr. Symonds, he stated that the skiagram showed a line of importance in cases where the neck of the femur was in question. The upper border of the obturator foramen and the lower border of the neck of the femur formed the arc of a circle in normal limbs. If there was any distortion of the neck, the arc was incomplete or broken. In this case the arc was perfect on the left side though smaller than that on the right. The right bones seemed larger in the X-ray. The femora and tibiæ had been measured, and the measurements showed that both bones were larger on the right side. The right limb was distinctly warmer than the left. It had the appearance one would expect to find in a young woman rather than in a girl before puberty. He believed that the line he had referred to was first pointed out by Mr. Shenton.

Case of Thyro-glossal Fistula.

By W. G. Spencer, M.S.

The patient was a girl with a thyroglossal fistula, which had been operated upon at other hospitals eight times. Mr. Spencer showed the excised fistula, including a piece of skin, which represented the flattened, stretched scar in the neck. A fistulous tract ran up close behind the hyoid bone, of which he cut out a centimetre. Guided by an injection of methylene blue, he had followed up the fistula nearly to the foramen cæcum. It healed by first intention and the ugly broad scar was reduced to a linear vertical one. Two or three years ago he had a case of similar kind which had been operated upon elsewhere four times. There also he followed the fistula up and excised a piece of hyoid bone, and that, too, was cured. There were other cases, equally difficult, which seemed to pass in front of the hyoid bone. The removal of a piece of hyoid bone did not seem to have harmed the patient, and she took to swallowing directly afterwards, and could speak well. Unless some such measure was taken, these fistulæ tended to re-form.

Arsenic Cancer, with description of a Case.

By R. J. Pye-Smith, F.R.C.S.

On November 7, 1910, I first saw Mrs. T., aged 29, in consultation with Dr. W. Harwood Nutt, who gave me the following history of her case: About eighteen months previously the patient had first noticed a little thickening of the skin under her wedding ring, and eight or nine months later ulceration had commenced there. When Dr. Nutt first saw her, in February, 1910, he found a deep ulcer on the radial side of the first internode of the left ring-finger. He treated it for a month with X-rays and then with zinc ionization. No improvement taking place, the patient submitted, on August 26, to the ring-finger being amputated by Dr. Nutt, at the metacarpo-phalangeal joint. The ulcerated part was examined by Professor J. M. Beattie, at the University of Sheffield, and reported to be malignant, being regarded at first as probably an endothelioma. At the operation it was noticed that there was a little scaly, dry patch of skin at the base of the left middle finger where it had been in contact with the ulcer on the ring-finger. At this part of the amputation wound no healing took place and the wound soon took on the appearance of the old ulcer. A month later the patient showed Dr. Nutt an ulcer on the pubes, which she thought had commenced seven months previously, at about which date she had become pregnant for the first time, though married seven years. Ulceration was also found about the labia, and, on the suspicion that the lesions might be syphilitic, iodide of potassium was now prescribed. As no improvement took place in a month, a piece of the edge of the ulcer on the pubes was excised on October 20, and on examination by Professor Beattie was pronounced to be definitely epitheliomatous.

When I saw the patient the appearance of her hand at once reminded me of the coloured plate in Sir Jonathan Hutchinson's "Archives of Surgery," representing Dr. James C. White's first case of arsenic cancer. On inquiring of the patient's mother, who was present at our examination, we ascertained that the patient had nearly all her life had a very dry skin, and that from the age of 7 to 14 she had been under treatment for that condition. The patient also stated that for the last six years her skin had been getting worse, a number of small, hard, warty specks having come on her hands and elsewhere, and latterly the skin of the palms having become very thick. For the last few years also the scalp had been very dry, whilst dry, white patches of rough skin had formed there and on other parts of the body, one of the earliest being at the site of the present ulcer on the pubes.

The general appearance of the patient was healthy. The left hand had lost its ring-finger and over the palmar aspect of the head of the corresponding metacarpal bone there was a deep ulcer, destitute of granulations, but with thick, red, and tender edges. Among the hair on the pubes there was a raised ulcer measuring 2 in. transversely and 1¾ in. vertically, with thick, but not hard, edges and with a smooth, moist, and red base. On both labia minora there were soft and moist strawberry-like growths, that on the left side being nearly ½ in. in diameter. On the scalp and on various parts of the body there were a few dry, slightly raised red patches, from ¼ in. to 1 in. in diameter, covered with whitish scales or crusts, suggestive of seborrhœa sicca. but no doubt part of a general keratosis. On the upper limbs were several spots of, or like, psoriasis. The cuticle of the palms and soles, except in their centres, was very thick. Scattered over the hands, to a less extent over the forearms and feet, and only sparsely over the trunk and lower limbs, were small lumps with thick and hard epidermis, those on the palms being apparently small corns, but those on the backs of the hands looking like warts and being red at their bases. No enlarged lymph glands could be felt anywhere.

As the patient was expecting her confinement in a fortnight, we advised her to have the left hand amputated immediately and to leave the vulval lesions to be dealt with after the puerperium. Accordingly, I performed amputation 2 in. above the wrist on November 9, and three days later the patient gave birth to a healthy child. She made a good recovery from both events. The ulcer on the hand was examined and was reported to be epitheliomatous.

The medical attendant of the patient in her girlhood was not now living, but on inquiry of his successor, who had formerly been his assistant, the case was remembered as one of psoriasis, and, although the prescriptions could not be found, no doubt was felt that they had contained arsenic.

A month after the patient's confinement X-rays were again applied to the pubic ulcer, to the palms and soles, and to the spots on the forearms, whilst carbon dioxide snow was applied to several of the larger warts and corns. The ulcer continued to spread, but the other lesions steadily improved under these applications for two months. The patient then consented to undergo a further operation, and on February 21, 1911, I excised the pubic ulcer, which now measured 3 in. by 2 in., cleared out the glands and fat from both groins, and removed the growths on the labia. The portions removed were found, on examination, to be epitheliomatous. After healing, X-rays were again applied over the scars for some weeks. The patient then enjoyed good health for about six months, when she began to be increasingly short of breath, and early in January, 1912, severe headaches came on, which were soon accompanied by giddiness and occasional vomiting. The optic disks were examined, but were found normal.

On January 22, I again saw the patient with Dr. Nutt. She was lying in bed, cyanosed and sweating; pulse 120, small; respiration 18; temperature normal. The right side of the chest moved very little in respiration. It was dull on percussion anteriorly. The breathing was bronchial and the vocal resonance increased. The patient was too ill to be raised for examination of the back. The left side of the chest and the heart appeared normal. There were no signs of recurrence at the sites of any of the parts operated on, nor were any lumps to be felt on the scalp, neck, or elsewhere, except one small nodule at the axillary border of the right breast. The skin generally was much more natural than a year previously, only a few small corns and warts remaining, and some slight remnants of the callosities on the soles.

A week later the patient became comatose, and she died on January 30. Every effort was made to obtain a post-mortem examination, but without success.

REMARKS.

It is mainly owing to the teaching of Sir Jonathan Hutchinson that arsenic has now been generally accepted by dermatologists as a causative factor in certain cases of cancer of the skin. Including the case just narrated, I have been able to tabulate thirty cases of so-called arsenic cancer. In almost all of these there was a history of the administration of arsenic for years, followed by keratosis, especially of the palms and soles. In two-thirds of the cases psoriasis was the complaint for which the drug had been prescribed, but rarely, if ever, did an epithelioma develop on a patch of psoriasis. In half the cases the epitheliomata were multiple. One-fourth of the cases occurred in women, and in one-fourth the subjects were under 35 years of age.

As in many cases where injury by mechanical, chemical, X-ray or atmospheric causes plays a part in the production of epithelioma of the skin, so in these arsenic cancer cases the onset of malignancy is commonly preceded by a local condition of hyperkeratosis, often accompanied by desquamation and pigmentation, and followed by fissure and ulceration, or by ulceration without fissure. It looks as if arsenic induces keratosis of the skin, the keratosis mechanically causing fissures, which readily become infected with bacteria and then ulcerate, the ulcers finally becoming cancerous.

Sir Jonathan Hutchinson, however, has repeatedly suggested that arsenic may possibly be a causative factor in forms of cancer in which it has not hitherto been suspected; and so widely is this metal distributed in Nature as to lend countenance to the suggestion, for its occasional ingestion in food or water must be possible to nearly all races of men and to many animals. Its presence in soot has been held to be the cause of the influence of that material in the production of chimney-sweep's cancer, and its presence in coal-tar and pitch may perhaps be held to account for the similar effects of those substances on the skin of briquette makers and paraffin refiners. The occurrence of cancer of the lungs among workers in smaltine mines has been thought to be due to the inhalation of the dust (arsenide of cobalt), and cancer of the bladder among aniline workers has been attributed to the effects of the excretion of arsenic inhaled as vapour in the process of manufacture. I have not met with any definite statement as to the frequency of cancer among the arsenic eaters of Styria. Probably their number is small, as compared with the sufferers from psoriasis in Europe who have been for years together treated with Fowler's solution.

The pathological action of arsenic in causing hyperkeratosis, and its therapeutic effect in psoriasis, point also to a special influence on the skin, for which several hypotheses have been advanced. It appears to depend in part on the chemical affinity that exists between arsenic and keratin.

Various considerations, however, seem opposed to the supposition that arsenic is at all generally a factor in the causation of epithelioma. One of these is the low cancer-mortality of coalminers, in spite of the presence of arsenic in coal dust. Another is the existence of cases closely resembling the one just narrated, but without any history, sign, or apparent probability of arsenic having been present. In many of these cases mechanical, atmospheric, X-ray and other injuries or irritations provide such obvious causes as to exclude the suggestion of arsenic in the absence of definite evidence. Again, the multiplicity of the lesions commonly found in cases of arsenic cancer as in those of X-ray cancer, sailors' cancer and xerodermia pigmentosa, seems to separate all these in ætiology from the more ordinary cases of single epithelioma of the skin. Lastly, where hyperkeratosis is present we already have a condition which is well recognized as one which is frequently pre-cancerous, though only exceptionally referable to the effect of arsenic.

It seems, therefore, reasonable to conclude that, whilst arsenic plays a definite part as an ætiological factor in the small group of cases of so-called arsenic cancer, there is no probability of its being of ætiological importance in the great majority of cases of epithelioma of the skin, and still less in cancer generally. Arsenic cannot be regarded as the essential and efficient cause, without which cancer cannot occur, but it may well be one of many predisposing or contributory causes. The real cause of cancer is yet to seek.

Among the latest hypotheses on the subject there is one which offers a biochemical explanation of the cancer-inducing properties of soot, tar, pitch, &c., other than their containing arsenic. If the researches into induced cell reproduction and cancer, by Mr. H. C. Ross and his coadjutors, should be confirmed, they would seem likely to elucidate the mode of action of many reputed factors in the causation of cancer.

Whether or not the existence of this group of cases is capable of throwing any light on the problem of the cause of cancer, its recognition may at least serve to warn us of a danger attaching to the prolonged administration of arsenic.

DISCUSSION.

The CHAIRMAN (Mr. W. G. Spencer) remarked that this paper confirmed what Sir Jonathan Hutchinson had stated in his Archives. Mr. Pye-Smith had pointed out that the arsenical lesions in this case, as in others previously recorded, did not start on the original psoriasis patches, but were independent lesions pointing to the separate action of the arsenic itself. Now that arsenic was being given more largely, in an organic combination, the subject became of increased importance. He had been told of a case of arsenical epithelioma traceable to salvarsan given to a syphilitic patient. In the treatment the arsenic should be stopped when nodules and keratosis appeared.

Mr. H. C. Ross remarked that Mr. Pye-Smith had mentioned the researches made by him (the speaker) and his colleagues at the Lister Institute, on the subject of pitch cancer. It seemed from the inquiry in connexion with pitch cancer that arsenic caused a predisposing condition similar to that caused by aniline dyes; both caused localized cell death, and this cell death set free certain agents, which caused the predisposing conditions. In pitch cancer, which was like chimney-sweep's cancer, there was a different condition, in that the pitch and tar contained a definite auxetic. Watery extracts of pitch and tar contained these substances which were derived from the decomposition of the proteins, and from the decaying vegetable matter from which coal was derived. Those substances were soluble in water, but did not contain arsenic. The researches at the Lister Institute had arrived at an interesting stage. The auxetics (amino-bodies) seemed to cause conditions predisposing to epithelioma in the workmen employed in the pitch factories. Pitch was derived from tar, and tar from coal, and in the briquette factories in South Wales the employees suffered from the condition. In animals they had been able to produce conditions resembling epithelioma by the action, not only of the watery extracts of pitch, tar and coal, but also of aniline dyes. But animal experimentation was not sufficient to prove the point. Therefore there was about to be made a series of experiments on a vast scale, as three companies of tar distillers were arranging to apply the process of treating the tar with a certain quantity of formaldehyde, which latter would destroy the amino-bodies. These companies would agree to make between them about 30,000 tons of tar during the next six months, treated with formaldehyde. One of the briquette companies would agree to use nothing but the treated tar, and the other briquette companies would act as controls. Before the experiment started a commission of medical men would be sent to the briquette company who would use the treated tar, and would compare the skin conditions in this company's employees with those of the men in the other factories. After the experiment had been continued for some months the commission would again report. Thus he hoped that in the next few months there would be a very practical proof as to whether the experiments which had been done were right. The outcome of this inquiry might be to take this industry off the schedule of dangerous trades. There would also be strong evidence as to the part played by these nitrogenous auxetics in the production of these predisposing conditions of epithelioma among briquette makers.

Dr. JAMES GALLOWAY said that it would be in the recollection of many present that when they were in the custom of seeing out-patients at their various hospitals some years ago, many cases of prolonged treatment by means of arsenic were in attendance. These were the patients who remained from the days when regular treatment by arsenic for many morbid conditions was a matter of routine. These patients frequently showed the different forms of arsenical degeneration of the skin. The usual features were the peculiar form of pigmentation and the changes in the epithelium resulting in hyperkeratosis. This modification of the horny epithelium was clearly degenerative in type, and frequently led on to the development of the peculiar warts and sometimes to the superficial form of epithelioma described by Mr. Pye-Smith. Hyperkeratosis was a change of degenerative character, and clearly the epitheliomatous change resulted from similar degenerative influences. This was a matter of long-standing clinical observation which was receiving clearer explanation at the present time. In dealing with these warty lesions it seemed that the best scars were formed by the careful use of solid carbonic dioxide. The use of X-rays should certainly be avoided. The scar left after freezing by carbonic acid was very superficial, supple, and often very satisfactory. All such cases should be at the same time carefully treated from the point of view of improving the general condition of health. He thought it was a very prudent course to sound a note of warning that the use of salvarsan, in large quantities and by repeated injections, might very well be followed by the same type of degenerative epithelial lesions to which he had alluded following the indiscriminate use of arsenic in days not long past.

Mr. MIDELTON remarked that arsenic was a very powerful drug, and one should think twice about using it if it was liable to cause cancer. He had substituted continuous counter-irritation, following acupuncture, which was originated in Germany. He did not know why it was not practised more in

England. The results were excellent, and it was quite safe. He had cases of both dermatitis herpetiformis and psoriasis which had done very well by this means. An ingenious instrument carrying thirty-three needles was employed, and a ring of punctures the size of a shilling was made. Over this area was painted a solution of cantharides, croton oil, and acetic acid. In two days the eruption had an alarming appearance, but a few days later there only remained some slight irritation.

The CHAIRMAN asked whether it was true that on the Continent, where there were aniline dye works, the workers still suffered as in the early days from cancer of the bladder, and if so, was it due to the dye or to the arsenic in the dye?

Mr. ROSS said it was true there were a larger number of cases of epithelioma of the bladder amongst aniline dye workers. He believed it was due to their swallowing aniline dyes. Dr. Legge, of the Home Office, drew his attention to that, and an effort was being made to get some more facts on the subject from Germany, but they had not yet arrived. It had been known for years that with aniline dyes epithelial tumours could be produced which looked like cancer. The dyes were known to cause cell division, but only indirectly by causing cell death. And it was probable that this was the manner in which arsenic also caused some pathological conditions.

Mr. PYE-SMITH, in reply, said that this girl was treated with arsenic, apparently, from the age of 7 to 14. At the latter age she had ceased taking it. When he first saw her she was aged 29, and she had been seen for the cancerous condition eighteen months previously to that, and had then noticed the keratosis for six years. Thus the interval between ceasing the arsenic and the onset of the keratosis was about eight years.

PROCEEDINGS

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COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Dermatological Section.

October 17, 1912.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Three Cases of Subcutaneous "Sarcoid" (or Hypodermic Tuberculide) of Darier and Roussy.

By H. G. ADAMSON, M.D.

THE patients were a woman, aged 45, and two girls. All three patients appeared to be in good health but for the skin affection. The skin affection consisted of circumscribed mauve-coloured somewhat infiltrated patches of the size of a florin to areas of two or more inches in diameter. In the substance of these patches there were scattered reddish-brown nodules of the size of a pea, some of which were more easily felt than seen; while others projected slightly above the surface of the skin and were evident to the sight as nodules. These three cases corresponded very closely to the description given by Darier and Roussy of the eruption which Darier called "sarcoid," and which the two observers afterwards recognized as forms of hypodermic tuberculides nearly allied to Bazin's erythema induratum scrofulosorum. The case of the woman was in all respects like the cases recorded by Darier and others. The other two cases differed in the age of the patients (the affection being generally met with in middle-aged women). They were also notable in that the eruption began in both instances as patches of purplish mottling associated with a more general condition of livedo and with severe joints pains.

Case I.—The first patient, R. B., was a married woman, aged 45. She was somewhat stout, and except for the eruption appeared to be in robust health. The eruption had begun two and a half years ago as a red spot 1½ in. above the right nipple. Then a second patch and a third had appeared on the right arm and on the left foot, and the margins of the ears had become red and swollen. The patches rapidly developed into irregularly shaped disk-like areas from one to several inches in diameter. They were of bluish-red colour, slightly raised and infiltrated, and embossed with reddish-brown nodules of the size of a pea, which could be more readily felt than seen. This case had been shown to the Section in February, 1910,[1] as a case for diagnosis, and the condition had not altered to any marked extent since then. An injection of old tuberculin, 0·005 c.c., had been given in May, 1910, and this had been followed by a general reaction, with the temperature rising to 101·2° F. and the production of a large erythematous node at the seat of injection in the back. A second injection in the arm of 0·004 c.c. of old tuberculin in January, 1911, also gives a marked local reaction. The diagnosis of hypodermic tuberculide, or "sarcoid" of Darier, was then made.

Case II.—The next patient was a healthy-looking, rather stout little girl, L. G., aged 10. The eruption had been present with varying intensity for five months. It first appeared as painful and tender patches on arms and knees, like bruises, after she had been in bed for two weeks with pains in the limbs and joints. On July 5 (three months ago) the skin infection consisted of (1) patches of deep purple mottling, having a reticular pattern which corresponded in some measure with the network of livedo annularis. These lesions were situated on the outer side of the right arm and forearm, on the left arm, on the front of the right knee, and on the right calf. There was no feeling of induration in these patches, and they disappeared on firm pressure, except for a little staining which remained. (2) A general tendency to livedo annularis. On September 4 there appeared fresh lesions on the thighs and buttocks, and those upon the arms became more pronounced. These fresh lesions were in the form of large dusky-red areas, in which there were several pea-sized red-brown nodules of the size of a small pea, distinct and firm to the touch, and apparently situated in the deeper part of the skin. The reticular character of the patches was pronounced, and the nodules appeared to occupy the lines of the network. The patient appeared otherwise in good health, and a thorough physical examination had failed to reveal any sign of tuberculosis. A von Pirquet test gave a positive, though not very marked reaction. A microscopical section of one of the nodules showed a round cell and epithelioid cell infiltration around the blood-vessels of the deeper parts of the corium and in subcutaneous tissue.

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[1] *Brit. Journ. Derm.*, 1910, xxii, p. 89.

Case III.—The third case was that of M. McC., aged 12, healthy-looking and well nourished. She has been under the care of Dr. H. Morley Fletcher, who had kindly allowed Dr. Adamson to show the case here. The eruption appeared one year ago, together with pains in the joints and slight attacks of vomiting. She was admitted to the ward. No visceral lesions were discovered, and although some of the larger joints were tender there was no swelling. Two weeks after admission she had an evening rise of temperature (101° to 102° F.), with rapid pulse at times, which continued for about a fortnight. This was accompanied by fresh joint pains and fresh eruption on the skin and slight sore throat. The left knee became tender and slightly swollen. The *eruption* consisted of an irregular mottling of dusky-red colour upon the arms and legs and buttocks, and less marked on the cheeks. The mottling was in patches, each patch having a broken reticular aspect. The dusky-red patches were slightly raised and very slightly infiltrated. To-day the eruption was much the same, but with the addition on the left cheek of a large circumscribed dusky-red infiltrated patch studded with reddish-brown pea-sized nodules. The diagnosis made some months ago was that of an unusual form of Bazin's disease or erythema induratum scrofulosorum. To-day the patch on the cheek led the exhibitor to call it "sarcoid" of Darier. No "tuberculin" test has been used in this case.

DISCUSSION.

Dr. ADAMSON, in answer to a question that had been asked by Sir Malcolm Morris, said that the "sarcoid" of Darier differed from "erythema induratum scrofulosorum" in the grouping of the nodules, in having less tendency to break down and ulcerate, and in their distribution on the face, trunk, or extremities.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) said he had seen marked improvement in some of these cases from the administration of thyroid. He found that tuberculin was uncertain in its action, and referred to a case in one young lady in which tuberculin had had no effect at all. He did not think the fact of a patient having the condition five years and not developing tuberculosis meant that the lesion was not tuberculous, as it was so slow-growing.

Dr. GALLOWAY said that he was glad to observe that Dr. Adamson hesitated in stating that the lesions in these cases were of tuberculous origin. He believed that the name "tuberculide," so commonly applied at the present time to various necrosing lesions of the skin, was a very unhappy one. In many of the cases so described the evidence brought forward of tuberculous infection of the lesions themselves or even elsewhere in the body was exceedingly slight, or entirely absent. The so-called "tuberculide" might as well be called X or Y or Z. In addition, the term suggested by analogy the close relation between a syphilide and syphilis; obviously no such close relationship existed in the case of tuberculides. He had just completed the investigation of a case in which there occurred in a woman aged about 40 indurated and necrosing lesions, mainly of the lower extremities, which might well be taken for the erythema induratum of Bazin, though it was noteworthy that there was little or no tendency to extensive softening or ulceration of the lesions. Microscopic examination showed that the affected areas consisted of an infiltration with certain resemblances to the granuloma of tuberculosis. Multinucleated giant cells were present as well as large mononuclear cells. So far as the histological examination was concerned the superficial resemblance to tubercle might well appeal to an inexperienced observer. Yet no evidence of tuberculosis existed in this patient, though she had been carefully watched for a period of nearly five years, and careful inoculation of the material obtained from one of the lesions into a guinea-pig gave an entirely negative result. It was especially interesting to note that this woman had come under observation nearly five years ago suffering from severe anaemia of what appeared to be the chlorotic type, and during this period the indurating and necrosing lesions of the skin commenced to develop. They had rapidly improved when she was able to rest in bed and be properly cared for. Many of the cases described as "tuberculides" occurred in patients with a stagnating peripheral circulation, and it appeared to be possible that thrombotic lesions of cutaneous arterioles and venules, not necessarily of tuberculous origin in any sense of the word, were the real cause of these necrosing cutaneous diseases. He would very much like to hear Dr. Adamson say that he would go still a step forward and declare that the relationship of these necrosing lesions to tubercle was very doubtful, and that in many cases there was no such connexion.

Dr. PERNET said he had examined some lesions of acne agminata and concluded they were not tuberculous.[1] As to Bazin's disease, the full name, it must be remembered, was "erythème induré des jeunes filles."

Dr. WHITFIELD said he believed one could, with great care, get rid of these lesions by means of tuberculin in very small doses. He had a girl under his care who came with lesions which had always burst, and there were the scars of them on her legs. There was one lesion when the tuberculin treatment was commenced, three weeks ago, and that had not burst. He began with $\frac{1}{5000}$ mg. of old tuberculin. She had had no reaction except the usual one at the site of the needle puncture.

Dr. WHITFIELD, answering Dr. Galloway, said he considered the only test was by tuberculin, because in many cases inoculation of the material did not kill a guinea-pig. One could not claim that a lesion was not tuberculous unless that were done.

Dr. ADAMSON, in reply, said he thought that there was abundant clinical evidence that the more familiar "tuberculides" were really of a tuberculous origin: that they had been so regarded for many years before the introduction of the term "tuberculide" was shown by their older names of "lichen scrofulosorum," "acne scrofulosorum," and "erythema induratum scrofulosorum." That the type of case shown to-day under the name of "sarcoid" of Darier was really a toxi-tuberculide was, perhaps, not so clear, but there was abundant reason for regarding it as a toxic eruption.

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[1] Vide Radcliffe-Crocker's "Diseases of the Skin," 1903, 3rd ed., ii, p. 1097.

Case of Paget's Disease of the Nipple.

By HALDIN DAVIS, F.R.C.S.

THE patient, a woman, aged 58, exhibited on the left breast a circular excoriated area about 2 in. in diameter, which had totally destroyed the nipple, where the patient said that the ulceration had started. The ulcerated area was smooth, glazed, and sharply defined. There was some induration in the region of the nipple, but no definite mass of malignant disease. There was no enlargement of the glands of the axilla. As the patient refused operation, it was proposed to treat her with radium.

DISCUSSION.

The PRESIDENT, discussing Dr. Davis's case, said that in two cases which absolutely refused operation an excellent result had ensued from the use of radium; there had been no recurrence, and the breasts felt normal afterwards. He considered that thin women with small breasts had a worse outlook in the condition than those with larger breasts. He admitted that there were limitations to the use of radium in the condition.

Dr. MACLEOD considered that it was advisable in every case of Paget's disease of the nipples to resort to free excision. He had seen marked benefit, if not cure, in two cases from treatment with radium, but in view of the serious microscopic changes which were present as soon as the affection could be recognized clinically, he considered that to delay surgical procedures was unwarranted.

Dr. SEQUEIRA recalled a case of his in which there was Paget's disease of the glans penis in a man, aged 82, who was shown before the Section. In this case an operation was performed, the glans penis being excised. The patient died from uræmia, and at the autopsy a rounded mass of carcinoma was found in the bulbus urethræ.

Dr. WHITFIELD said he had seen in the laboratory specimens from a case of the kind, where the lesion, before the shrinkage, was no bigger than a shilling, yet there were blocked gland-ducts lying in the deepest part of the mammary gland close to the pectoral muscle. He therefore thought that the whole breast should be removed in any surgical treatment of Paget's disease.

Cases of Inherited Alopecia.

By Haldin Davis, F.R.C.S.

The patients shown were the mother, aged 26, and her daughter, aged 2. The baby had been born with a normal amount of hair upon the scalp, but at the age of 4 months it fell off, leaving a fringe round the occiput and a single tuft of fine hair on the vertex of the skull. With these exceptions it was perfectly bald. The scalp was thin and atrophic and the blood-vessels were plainly visible through it; in fact, the whole appearance of the scalp was similar to that seen in senile alopecia—very different to that seen in alopecia areata, where the scalp remains thick and fleshy even although totally bald.

The mother, who wore a wig, gave a history exactly similar to that of her baby. Her hair had fallen when she was a few months old, and with the exception of a fringe round the occiput she had remained bald ever since. Her eyebrows were unaffected, but she had no hair on her body. The nails were normal in both mother and baby.

Dr. Graham Little said that Sabouraud, in a recent series of 100 consecutive cases in his clinic, found that of the eighty-one cases in which there was a history 22 per cent. had hereditary transmission of the alopecia. On looking at some of his own cases he had been struck with the frequency with which that was so, even in ordinary common alopecia.

Chancre of the Lower Lip in a Woman, aged 26.

By S. E. Dore, M.D.

When the patient was first seen, three weeks ago, there was a small hard nodule the size of a large pea below the red margin of the lower lip. There was very little inflammation of the skin apparent, and the lesion looked like a small cyst. The following week the small tumour had increased in size, and an enlarged gland could be felt under the chin. Material from the lesion showed the presence of the *Spirochæta pallida*, and the Wassermann test gave a positive result. When the case was shown there was a prominent growth about the size of a sixpenny-piece and a large submental gland. The patient was a married woman and had four healthy children. She had noticed no eruption on her skin nor had she suffered from sore throat, and, with the exception of the lesions already mentioned and some enlargement of the posterior cervical glands, there was no clinical evidence of syphilis.

Two Cases for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

Case I.—The patient was a gentleman, aged 48, retired from business. He had had several nervous "breakdowns"; was inclined to be shaky, was losing memory, and sleeping badly. Six months ago there began to appear upon the glans penis two small circinate lesions, one of which had disappeared entirely, the other persisting. The present lesion, situated on the left segment of the glans, was the size of a threepenny-piece and nearly completely circular; the margin, slightly raised and enclosing them, somewhat different in colour from the ordinary. There were, and had been, no lesions on any other part of the body. The area affected was moderately itchy and very slightly scaly. There was some itching about the anal region, which had resulted in some excoriation of the part.

The exhibitor offered the diagnosis of lichen planus, and recalled a similar case shown to the Section by Dr. J. M. H. MacLeod.

Case II.—Girl, aged about 10. On the dorsum of the left hand just above the wrist there was a single lesion which constituted the eruption. This was, when first seen, ring-shaped, with the edge more prominent than the centre, of a livid red, and showed considerable induration. It was rather larger than a sixpenny piece and had persisted for about eighteen months, slowly increasing in size. She had been under treatment for about three months with salicylic acid plasters and the lesion had somewhat flattened and become more uniform, so that the ring had been almost replaced by a nummular or disk-like hard swelling, not unlike a keloid. Upon firm pressure with a glass slide the colour was completely expelled. There were no subjective sensations connected with the lesion. The child was otherwise well. There was no history of tuberculosis.

The livid red colour and the persistence of the lesion under treatment for several months with strong salicylic acid plasters seemed, to the exhibitor, to negative the diagnosis of granuloma annulare. He regarded it as falling into line with the group which Dr. Colcott Fox still regarded as a separate entity and called erythema elevatum diutinum.

Dr. GRAY said he saw a girl, aged 10, with somewhat similar patches on the knuckles, associated with marked rheumatic nodules in the neighbourhood of the interphalangeal joints and elbows. The condition cleared up entirely by the use of salicylic acid externally and salicylate of soda internally.

Case of Œdema Neonatorum.

By E. G. GRAHAM LITTLE, M.D.

THE distinction between œdema and sclerema is carefully made and insisted upon by Radcliffe-Crocker, often confused by other authors. The variety now exhibited was difficult to class, for it was essentially an œdema, but there was no lividity, and the skin was waxy-white and smooth. The process, however, was transient in duration, and when the swelling was over the skin seemed but little altered, slight desquamation being present. The patient was a female, aged 6 weeks. The œdematous change had been noticed at first in the feet and legs, then in the forearms and hands, but had not become generalized. The child when shown had a white and solid œdema of both the forearms, which ceased abruptly at the elbow, where there was a definite ridge separating the healthy tissue from the diseased. The legs had by now regained their usual size and aspect. The child, seen four days later, showed no signs of œdema of the forearm or hands, and seemed to be otherwise in fairly good health. There was no reason to suspect syphilis, and she had not had diarrhœa. Previous cases seen by the exhibitor had invariably ended fatally, but the prognosis in this case seemed favourable.

The PRESIDENT suggested giving small quantities of thyroid through the mother if she was suckling the child.

The Life-cycle of the Organism of Syphilis, illustrated by a Series of Preparations under the Microscope.

By J. E. R. McDONAGH, F.R.C.S.

PROBABLY no discovery of an organism was more readily accepted than that of the *Spirochæta pallida* made by Schaudinn and Hoffmann in 1905. Those who laboured to find the cause of syphilis were legion, and, prior to the birth of the *Spirochæta pallida*, the research which received most attention was that of Siegel with his *Cytorrhyctes luis*. So firm has been the belief in the *Spirochæta pallida* that that organism is taken for granted as being the sole agent of everything syphilitic. Now let us, for a moment, ask ourselves two questions:—

(1) Why is the incubation period of syphilis so long?

(2) Why do not one or two injections of salvarsan cure every case?

If syphilis is conveyed by the passage of spirochætæ from one person to another, ought not the initial lesion to begin to show itself two or three days after intercourse, as is more or less the rule with bacterial infections—viz., ulcus molle, gonorrhœa, diphtheria, &c.? The diseases which have a long incubation period are nearly all due to protozoa. The incubation period is long because the infective organism has to go through a cycle of changes before it can give rise to symptoms. Since the *Spirochæta pallida* is a protozoön—an assumption which one may safely make, owing to its rapid destruction under salvarsan—is it not possible that it is only one of the phases in the life-cycle of the syphilitic parasite?

The action that salvarsan has on spirochætæ in general is phenomenal. No spirochætæ are found in films made from the discharge from a chancre, after forty-eight hours, following a single injection. If the action is so marked, even on the surface of a lesion, how much more pronounced must it be in the deeper parts? In fact, all the spirochætæ must be, and no doubt are, killed by the first or second injection. In spite of this, recurrences occur again and again. In relapsing fever, for instance, and in yaws, one or two injections of salvarsan at the most are all that are required to cure every case. On the other hand, in sleeping sickness the trypanosomes vanish almost immediately after an injection, but they soon return into the general blood-stream. Trypanosomiasis and syphilis are therefore not unlike, and the frequent recurrence of symptoms met with in both diseases is no doubt dependent on the cycle which the parasite goes through in the body of the host. Many of the bodies about to be described I have also seen in the lymphatic glands of rats infected with *Trypanosoma Lewisi*.

These are a few of the points which have puzzled me so much of late, and have stimulated me to inquire a little more closely into the life-history of the *Spirochæta pallida*, since I thought it was quite possible that the syphilitic organism went through a series of changes common to protozoa.

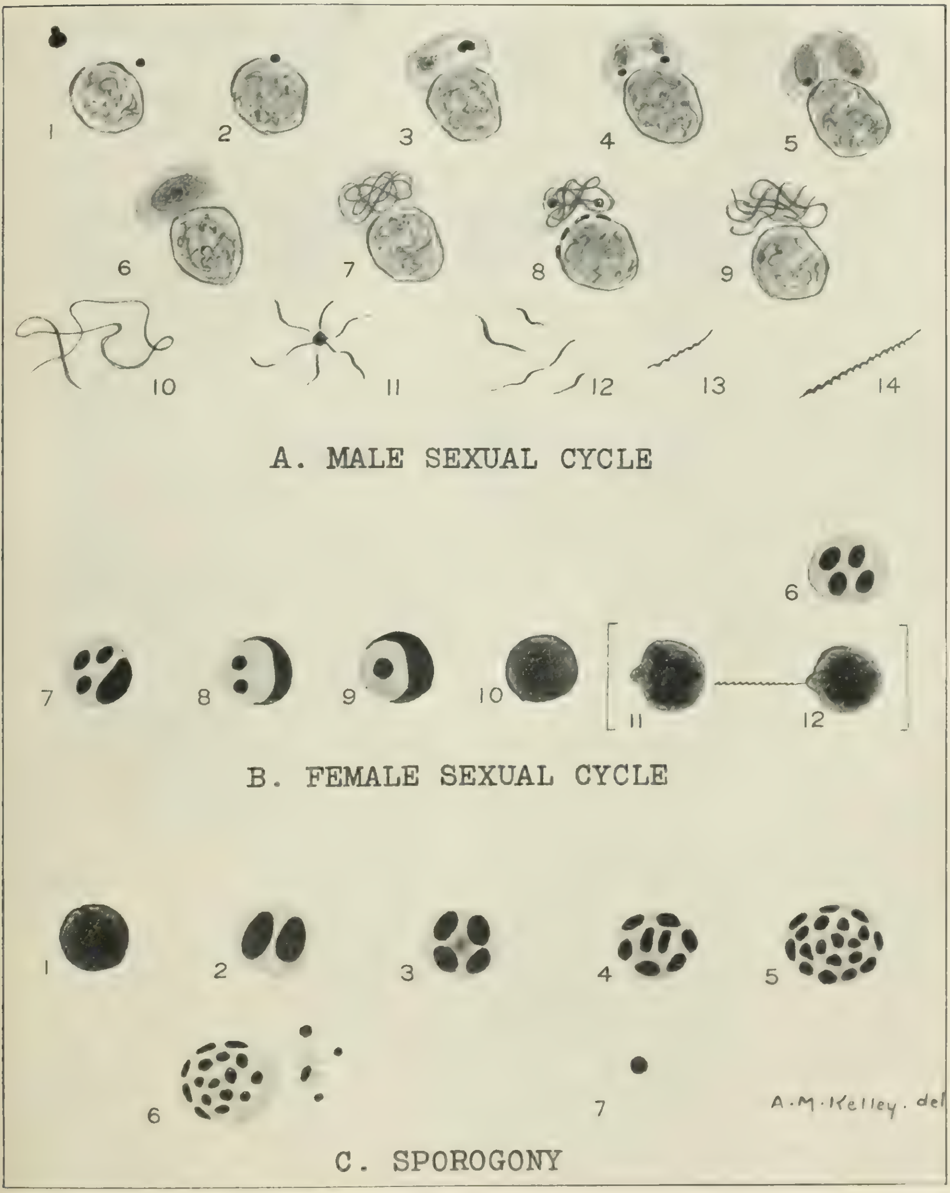
I started off with the idea that the *Spirochæta pallida* was the male gamete of some unknown protozoön. The reason for thinking that it was an end-phase of some cycle was due to the fact that I had never seen a *Spirochæta pallida* divide. If all syphilitic lesions are due to the *Spirochæta pallida*, and if the infection is a direct one of that body from one person to another, it is surely odd that we do not see division in every film we look at. This point, in itself, must suggest that the *Spirochæta pallida* is not to blame for all. It is doubtful whether the *Spirochæta pallida* divides at all. Has it any need to? In my opinion, in the body, probably no; what it does in culture is a different thing.

The variation in the sizes of the different spirochætæ, which is often explained as being the result of transverse fission, is probably dependent upon their development. The start is an undifferentiated coil, concerning which some remarks will be made later; this coil breaks up into spirochætæ, which are short, thick, and vary from being almost straight to slightly twisted. In the next stage the spirochæta is fine and evenly coiled, but short; and, finally, one gets a perfect *Spirochæta pallida* with about fifteen coils. All variations in the number of coils from about nine to fifteen or more are found. It is not uncommon to find a spirochæta with a bulbous extremity at one or both ends, or even in the centre. Opinions differ as to the nature of the swelling. Some observers see it in the spore stage of the spirochæta, and they assert that the swellings are formed when the spirochæta is in the act of dying; while others hold that they play some part in the multiplication of the organisms.

I have watched specimens alive for hours by the dark-ground illumination method, and I cannot help thinking that the swellings which are highly refractile are granules from the leucocytes which have been attracted by the spirochætæ. The granules have their origin in the leucocytes, leave them, and float about in the plasma; they are motile, and can, therefore, wander about easily. I have seen these bright bodies attach themselves to the spirochæta, and when they do so along the length of the organism, and not at its extremities, it appears as if the granule were lying on the spirochæta, rather than in the middle or body of the parasite. It has been mooted for some time past that the *Spirochæta pallida* had a resting stage; in fact, Schaudinn was at work on this very point just prior to his untimely death.

Prowazek was under the impression that the *Spirochæta pallida* rolled up into a ball. Similar circular bodies were also found by Hoffmann in the spleen of a congenital syphilitic, and intermediate stages in unrolling and rolling up were described by him. The resting stage, as just described, was held responsible for the long incubation period of syphilis. Krzysztalowicz and Siedlecki described a sexual development. In that, short, thick, nucleated bodies, which they looked upon as macrogametes, gave rise, by a process of division, to microgametes; but these authors afterwards altered their views.

For my research I cut sections of chancres, lymphatic glands and various forms of syphilitic skin lesions, and examined films obtained from their juice, both *in vivo*, by the borax methylene blue film method, and by fixed specimens. I paid most attention to the lymphatic



A. MALE SEXUAL CYCLE

B. FEMALE SEXUAL CYCLE

C. SPOROGONY

FIG. 1.

glands, as I thought the cycle was more likely to take place there than anywhere else, owing to the fact that in spite of vigorous syphilitic treatment the adenitis takes months to subside. I examined seventeen lymphatic glands, both before and after salvarsan, ten chancres, in various stages, from an early erosion to the slight induration left by a healed primary sore, and over twenty various skin lesions, both before and after salvarsan. For controls I examined four lymphatic glands enlarged by gonorrhœa, three enlarged by soft sores, three normal glands, and various glands enlarged as a result of tuberculosis, lymphadenoma and malignant disease, also five soft sores and skin lesions consisting of a plasmoma, or granuloma, viz., lupus, sporotrichosis, &c. In most instances in the syphilitic group (some of the skin lesions excepted), and in no instance in the control group, did I find any of the bodies which I am about to describe; and as these bodies were found exclusively in syphilitic tissue, I venture to suggest that they may be phases in the life-cycle of the syphilitic organism. These bodies I have seen in different films, and the interpretation I put upon them is the one that appears to me to be the most likely. The commencement of the cycle is with a sporozoite or infective granule, and by means of its progression, since it is motile (it appears to have flagella), the sporozoite reaches a cell and enters it; the cell is usually a large mononuclear leucocyte. Having entered a mononuclear, the sporozoite becomes motionless, increases in size, and develops a distinct mantle of protoplasm around itself. I have also seen parasitic bodies in small mononuclears, but in these cells they do not appear to develop. The further development goes on within and at the expense of the protoplasm of the mononuclear cell, the nucleus of the cell always remaining intact, and the degenerated protoplasm forms an envelope around the sporozoite, which has to be gradually increased in size according to the growth of the parasite. The sporozoite in some cases steadily increases in size, while in others it appears to divide. In the case where there is no division the development goes on until spirochætæ are formed—the male sexual cycle. In the case where there is division one half runs the course of the male sexual cycle, while the other runs the course of the female sexual cycle; the latter at this stage seems to leave the lymphocyte. It is possible that there are both male and female sporozoites, and that each enters a mononuclear on its own account, but the development of both male and female up to a certain point is so similar, and the female leaves the cell so early, that it is difficult to be absolutely certain whether a sporozoite is either male or female, or made up of both sexes.

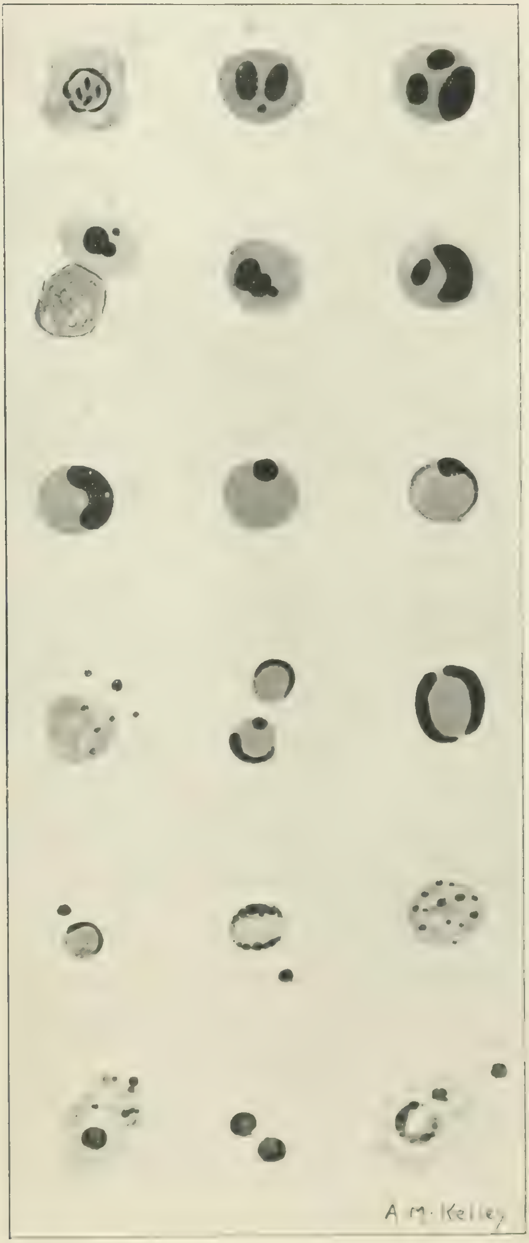


FIG. 2.

Bodies seen in sections.

Male Sexual Cycle.—The large trophozoite or merozoite becomes differentiated and later transformed into an irregular coil. The coil later is found to be thicker in some parts than others, and there sometimes appear to be some small, round, nuclear-like bodies in it. At the base of the coil, lying just above the nucleus of the cell, but in the protoplasm of the mononuclear, are some rod-shaped bodies, which stain better than the strands of the coil. I cannot make out the use of these rod-shaped bodies. The coil, which now appears to be extracellular, because the protoplasm of the mononuclear is so degenerated, becomes broken up into irregular, short, stout, and wavy bodies, and from these the delicate long and corkscrew-shaped organism, the *Spirochæta pallida*, develops. This is the microgamete, or adult male gamete, and it probably has no more necessity to divide than a spermatozoön. It sometimes happens that circular bodies with spirochætæ coming off, like the spokes of a wheel from the axle, are found outside the cell, and are probably the same as the small round bodies which are sometimes seen in the coil.

Female Sexual Cycle.—This is the same as the male up to a certain point. The female merozoite takes a circular form, and in the circle there appear to be four or five distinct pear-shaped bodies; at this stage the merozoite becomes extracellular. Two of these pear-shaped bodies then fuse and become crescentic; the remainder, one by one, join in until a spherical mass is produced. This is, in my opinion, the adult female or macrogamete.

The act of fertilization I have never seen, but it is highly probable that the spherical body forms a "cone of reception," which is touched by the microgamete; the microgamete must then penetrate the macrogamete until its pronucleus reaches the female pronucleus.

Spore Stage.—After fertilization a zygote is formed. The zygote then appears to divide into two and to subdivide into four. These four masses are the sporoblasts, and they are arranged around the circumference of the zygote; this outlying circular arrangement is kept up until the spores have been liberated and there is no further need for a casing. Each sporoblast divides and subdivides until numerous sporozoites are formed—a spore-cyst; when examined *in vivo* the cell appears to be alive with the movement of all the sporozoites. The cell then bursts, and the sporozoites are set free to start the sexual cycles again. This completes the sporogony. There appears to me to be also an important asexual development similar to that seen in the flagellata.

If what I have described is correct, the syphilitic parasite belongs to the Order Sporozoa and to the Sub-class Telosporidia, since the spores are formed at the end of the cycle. The Order is probably the Coccidiidea and the Species Leucocytozoon, hence the name of the syphilitic parasite should be *Leucocytozoon syphilis.* The infection is probably conveyed by the sporozoite and not by the *Spirochæta pallida.*

The bodies visible in sections of syphilitic tissues are no doubt types of all the stages above mentioned, and the drawing (fig. 2) will give the reader a much better idea of their appearance than a lengthy verbal account. There is no doubt that these bodies can still be found after treatment with salvarsan, and it is yet impossible to say what action anti-syphilitic treatment has upon them. Whether it is desirable, or even necessary, to give as many injections of salvarsan as is now the rule, and whether our aim to obtain a negative Wassermann reaction is a right one, are points which must remain *sub judice*, as our interpretation of the Wassermann reaction may be altered as the result of this discovery.

From the point of view of diagnosis the discovery of the life-cycle of the syphilitic parasite means a distinct gain, because bodies other than, and easier to find than, the *Spirochæta pallida* can be demonstrated with ease, and the old difficulty of being unable to distinguish one granuloma from another in section is partly done away with.

The best method of examining specimens *in vivo* is to take a fat-free slide and make a film on it of borax methylene blue; having allowed the stain to dry, a drop or two of secretion from a lymphatic gland, for instance, is put on to a cover-slip, and the cover-slip pressed down on to the slide. By this means the cells are metachromatically stained, all the organisms are stained, and even the delicate *Spirochæta pallida* can be seen moving about with ease. For fixed films Giemsa's stain is the best, and for sections I prefer Pappenheim's stain (pyronin and methyl green) as described in *Archiv für Dermatologie und Syphilis*, 1911, cix, p. 447 (McDonagh, "Ueber einige Transformationsformen der Plasmazellen"). In sections the bodies stain a brilliant red, they are most abundantly found in a giant cell formation (in the centre of a regional lymphatic gland) if there is one, or in the centre of a group of plasma cells. They are frequently found in the walls of vessels and in the vessels themselves among the red blood corpuscles I have seen free sporozoites. The bodies are also seen free in the lymphatic spaces.

For assistance in obtaining material I wish to thank my house surgeons at the London Lock Hospitals, who have kept me supplied since January, 1912, which was the time when I commenced my research.

Multiple Lupus Vulgaris following Measles in a Girl, aged 5.

By J. M. H. MacLeod, M.D.

The child had had measles two years previously, and in the convalescent stages of fever an eruption of reddish-brown spots had appeared, which, when first noticed, were about the size of a split-pea and were situated on both arms. These lesions had gradually grown, until at present they varied in size from a sixpence to a florin. They were confined to the arms, there being nine on one arm and ten on the other. The lesions were of the lupus type only; they had been considerably inflamed by some irritating ointment which had been applied, and several of them had become impetiginized. The child was one of a family of twelve, and there was no history of tuberculosis among them, nor, as far as could be gathered, in the antecedents. A physical examination of the child failed to reveal any other stigmata of tuberculosis.

Cases of this kind were by no means uncommon, and had been demonstrated at the Society by Dr. Adamson, the exhibitor, and others. They suggested the possibility that they were the result of an embolic shower of tubercle bacilli, emanating from some broken-down focus of tuberculosis, such as a mesenteric or bronchial gland, which had reached the cutaneous capillaries and there produced the tubercular lesions.

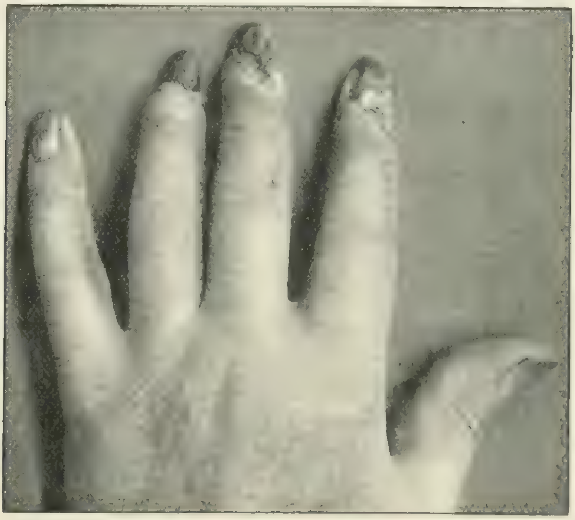
Dr. Adamson said that it was not uncommon for these cases of multiple lupus after measles to develop tuberculous lesions in other parts. He could recall one case in which there was subsequently a post-pharyngeal abscess, two others had hip-disease, and another tuberculous glands in the neck.

Case of Necrosis of the Terminal Phalanges of the Left Hand.

By J. H. Sequeira, M.D.

The patient, a florid man, aged 70, who had been a cabinet-maker, gave the following history: Eight years ago a small "sore" appeared on the left thumb near the nail, and this spread until, ultimately, it involved the nail-bed. The nail separated and was shed. A similar process affected all the other nails of both hands, with the exception of the little finger of the left hand and the right thumb. The nails separated by a moist process, but when the separation had occurred the lesions dried up and healed and the nails grew again. The patient stated that he was "gouty," and that he had picked out small pieces of chalk from the upper part of the pinna of the ears. Since the fingers were first affected the skin has always been of a livid purplish colour, and shooting pains had been felt in the fingers in frosty weather.

The present attack began in February, 1912. He first experienced shooting pains in the fingers of the left hand; these fingers became swollen, and suppuration occurred round the nails of the index, middle, and ring fingers. The nails again separated by a moist process, and the inflammation was attended with intense pain and tenderness. After the nails separated the ends of the fingers dried up, leaving brownish-black, dry, conical stumps, as shown in the accompanying photograph. The nail of the index finger has not yet separated. The middle toe of the right foot is somewhat similarly affected, but the nail has not yet separated. The urine was of specific gravity 1012. There was neither albumin nor sugar present. There were no tophi on the ears. The Wassermann reaction was negative. The neck was radiographed, but there was no evidence of cervical rib. There was no alteration in sensation to heat or cold, but during the acute attacks there was intense hyperæsthesia of the fingers and back of the hand.



Necrosis of the terminal phalanges.

Under simple tonic treatment and antiseptic dressings the ulceration dried up, and this appeared to be the natural course of the affection.

The case aroused great interest. It was difficult to see how it could be classified except with the condition known as Raynaud's disease, but the asymmetry of the lesions was an unusual feature.

Case of Lichen Planus of the Palms.

By J. H. SEQUEIRA, M.D.

THE patient, a corpulent, dyspnœic man, aged 42, with congested, swollen face, had suffered for the past five weeks from "pimples" on the hands and wrists. The eruption itched intensely. He was sent to Dr. Sequeira as a case of "cheiropompholyx," and, at first sight, the palms appeared to be studded with vesicles. A little closer examination showed that the eruption was really papular, the palms and the palmar surfaces of the fingers being covered with closely packed, rounded, flat swellings, varying in size from a pin's head to a hempseed. These papules were of the same colour as the rest of the skin. On the anterior surfaces of each wrist there were several characteristic papules of lichen planus, of a lilac tint and smooth, burnished surface. There were a few patches of eruption of the normal type in the front of each ankle. The most interesting feature was, perhaps, the extraordinary eruption on the buccal mucosa, palate, and tongue, where the surfaces were covered with closely set white spots the size of a pin's head. On the tongue the eruption had coalesced into flat white patches. The urine was acid, specific gravity 1020. There was no albumin and no sugar.

Dr. GRAY said he had a case a little more extensive than this, in which the condition was not confined to the palms, but also involved the flexor aspect of the forearms. He had treated one palm with two-thirds of a pastille dose of X-rays and it had reacted severely, and, in point of fact, took longer to clear up than the untreated palm.

Two Cases illustrating the Relation of Heart Disease to Skin Lesions.

By DAVID WALSH, M.D.

Case I.—The first patient was a woman, aged 36, who came in January, 1911, complaining of an irritating lesion on the left leg, it having begun as a spot three months before. There was a bad family history, and the lesion now shown was chronic eczema, which was moist at times. The mother died at the age of 60 of Graves's disease, and the father died at 40 of general paralysis. One brother suffered from rheumatism, and one sister from palpitation; one brother died of bronchitis, and an uncle had rheumatic fever. This patient had palpitation, shortness of breath, dyspepsia, varicose veins, and the hair was falling out. There was a malar flush, and she suffered from cold hands and chilblains. Dr. Halls Dally reported that there was an aortic systolic blowing murmur conducted upwards in the vessels of the neck, with an accentuated second sound and a mitral systolic murmur. He had found all kinds of skin diseases connected with unsuspected cardiac defects, some of them myocardial, in regard to which he had referred to an expert, as they were difficult to recognize. The eczema was one only of a large number of skin conditions noted in a similar association; some might have psoriasis. He had taken the chronic cases consecutively, and found a large proportion suffering from some serious circulatory defect.

Case II.—The second patient was a woman of middle age, who was attending a heart hospital, where many skin lesions would be found if sought for. She was under the care of Dr. Halls Dally for depression, weakness, insomnia, and palpitation. She had had four attacks of influenza, and the cardiac condition was considered to be post-influenzal. She was obviously the subject of myxœdema, and her hair had been falling off a great deal. The skin was dry, rough, and the trunk and upper limbs were covered with close-set spiny papules suggesting lichen rubra acuminatus.

DISCUSSION.

Dr. HALLS DALLY said he took the blood-pressure in each of these cases, and he did it as a routine procedure. In the first case the blood-pressure was normal. In the second, when she came for treatment on July 6, 1911, the systolic pressure (Pachon) was 210, while the diastolic was 110. On September 19, 1912, the systolic pressure had been reduced to 180 and the diastolic to 100. Here cardiac debility was manifest consequent upon cardiac degeneration. The first case was that of an early compensated aortic stenosis, with some mitral regurgitation superadded, which would probably account for the circulatory disturbance. Dr. Walsh had been to his (the speaker's) out-patient department at the National Hospital for Diseases of the Heart, and there had examined many consecutive cases of heart disease. In some of these patients there appeared to be a direct and close connexion between general circulatory disturbance and skin affections.

The PRESIDENT said the myxœdema in the second patient would account for the skin condition.

Dr. WHITFIELD said the case was one of myxœdema, which had the skin lesions usually associated with that condition and which had always been recognized. The fact that this patient had myocardial degeneration was most likely a coincidence. He imagined it was not claimed that most cases of myocardial lesion showed this particular dermatosis. The balance of evidence was that the dermatosis was connected with the myxœdema, and not with the cardiac lesions. He did not deny that cardiac lesions might have a marked influence on the production of skin lesions, but if these cases now shown were the best which Dr. Walsh could show to establish his contention, they were unfortunate instances, because they could be explained more easily in another way.

Dr. F. PARKES WEBER agreed with Dr. Whitfield that the two cases could scarcely be accepted as supporting the theory advanced by Dr. Walsh. The second patient had obvious myxœdema, and myxœdema might favour myocardial degeneration and likewise give rise to skin changes. In the first case the cardiac valvular defect was compensated and the skin lesion was unilateral. He asked Dr. Walsh whether the skin changes which he had noticed occurred in association with compensated or with uncompensated cardiac disease. Failure of compensation might lead to general œdema and sometimes to extreme passive venous congestion of the skin, a kind of "livedo annularis" or "erythema figuratum" in the extremities.

Dr. GRAY asked how Dr. Walsh correlated the heart condition with the skin lesion, and whether any blood-pressure observations had been carried out.

Dr. WALSH replied that he could only say in the most general terms that the associated skin lesions appeared to him to be due to some disturbance of the balance of circulation on the surface of the body, in which event the nutrition and general defensive mechanism of the cutaneous tissues must necessarily be upset; probably in most instances there was some kind of traumatism concerned. He had a long list of cases illustrating the point, but unfortunately the patients chosen had not been able to come. He wished to bring the point to the attention of the members, though nothing could, of course, be established by two cases. He had been to Dr. Meachen's clinic and seen his chronic cases; the results were astonishing, and he asked members of the Section to examine the heart in fifty consecutive chronic cases. He believed the skin condition, in some instances, to be a sign of failing compensation, manifested much earlier than those set out in text-books; indeed, he regarded the skin condition as a delicate symptomatic clinical test.

Note.—Excluding diseases of such definite and obvious origin as scabies, ringworm, syphilis, tuberculosis, and so on, any kind of cutaneous lesion could be investigated, provided it were chronic or recurrent.

Dermatological Section.

November 21, 1912.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case of Acne urticata (Neurotic Excoriations—One Type).

By H. G. ADAMSON, M.D.

THE patient was a married lady, aged 44. The skin trouble had begun eight years ago during a confinement. There was now, over the lower jaw and adjacent parts of the neck upon each side of the face, a group of closely set, dead white, oval scars of about the size of a split-pea. There were about thirty or forty scars on each side. Each scar had its long axis in the direction of the long axis of the jaw. The scars were not atrophic—that is to say, there was no feeling of loss of tissue as in an atrophic scar. Each scar was surrounded by a zone of pigmentation, so that the affected areas appeared as a network of pigmentation with dead white meshes. There were a few scattered scars below and behind the ears. In addition to the scars there were two or three crusted excoriations, and the patient stated that there appeared every few days one or more small (pinhead-sized), red (not raised), itchy spots; that the itching was so intense that she could not refrain from scratching the spots or rubbing them with a towel, and that then there appeared a bleeding patch, upon which there formed a crust, and that the fall of the crust was followed by a scar. The patient was apparently in good health. She said she was nervous and easily upset by little worries. The palate was less sensitive than normal, but not anaesthetic. There had been no confinement since eight years ago.

The exhibitor regarded the case as one of acne urticata or neurotic excoriations of the "dug-out" type of Colcott Fox.

Neurotic excoriations were seen in two forms: The neurotic excoriations of hysterical girls, in which there occurred long oval excoriations produced by rubbing with the tip of the finger; in this class of case the patient always denied that she produced the lesions. The second class were patients of nervous temperament, but not hysterical, who admitted that they rubbed or scratched the skin on account of the intense itching of a small spot or papule. The patient now shown belonged to this second group.

DISCUSSION.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) said it would be interesting to see whether there would be any scars in a patient who did not scratch or irritate the area concerned. In one or two strong-minded people who had abstained from scratching the lesions were very indolent.

Dr. MACLEOD said that he had recently seen a somewhat similar case in a neurotic girl, aged 16, in which the lesions were present on the legs, arms, and face. They consisted of dug-out excoriations which she confessed to have done with her finger-nails for the relief of intolerable itching. There were no definite papules present, nor could any history of such be obtained either from the girl herself or from her mother; but the itching seemed to be localized to individual spots, possibly in some cases about hair-follicles, and was only relieved when the skin was excoriated. He also referred to another case in a young woman, aged 20, in which the face was affected with similar excoriations. In this case there were some inflammatory papules present and the itching seemed to be localized in them.

Dr. PRINGLE agreed with Dr. Adamson in regarding the condition as what is generally called acne urticans, and he believed Kaposi would have done so too. But the name was an unfortunate one, as the lesions were not really acneiform. He believed he *had* repeatedly verified the existence of deep-seated nodules preceding the scratch-marks seen later. It was a class of case which was very obstinate, and was often misunderstood. His cases had almost, if not all, been in youngish women, but many of them were highly intelligent and presented no signs of hysteria.

Further Researches on Trichomycosis Flava Rubra et Nigra of the Axillary Regions.

By ALDO CASTELLANI, M.D.[1]

IN a note published last year in the *British Journal of Dermatology*[2] I called attention to a nodular affection of the hair of the axillary regions, observed in natives and Europeans living in Ceylon, resembling *Trichomycosis palmellina* of Pick. Since then I have seen numerous other cases, and am now in a position to give a short general account of the condition, its ætiology, and treatment.

Ætiology.—My further researches have confirmed the conclusion I came to in my previous paper. The yellow variety is due to a very thin, bacillary-like fungus, for which I have proposed the name "*Nocardia tenuis.*" The black and red varieties are due to a symbiosis of this fungus with chromogenic cocci, a coccus producing black pigment (*Micrococcus nigrescens*) in the black variety, a coccus producing red pigment in the red type.

Discomyces tenuis, Cast., 1912.—The microscopic examination of the nodules reveals the presence of enormous numbers of bacillary-like bodies, which are Gram-positive,[3] but not acid-fast. They vary in length, 4 to 10 microns and more; they are rather thin, 1 to 1½ microns; they may be straight or variously bent, occasionally branching; they are fairly closely packed together, and are imbedded in an amorphous cementing substance. I have not succeeded in cultivating the fungus.

Characters of the Coccus-like Organism found in the Black Variety.—It is a Gram-positive, rather large, non-motile coccus, which in certain media may take the appearance of a cocco-bacillus. Sugar media are more suitable for the growth of the organism than the ordinary agar.

Sabouraud Agar.—Colonies appear twenty-four to forty-eight hours after inoculation. They are roundish, at first white, but after a couple of days the centre of each colony turns black, and this pigmentation slowly spreads eccentrically. After a time the colonies coalesce into a jet-black mass.

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[1] From the Clinic for Tropical Diseases, Colombo (Ceylon).

[2] *Brit. Journ. Derm.* 1911, xxiii, p. 341.

[3] If the nodules are kept in alcohol or formalin for several months the fungus apparently loses partially or totally its property of being stainable by Gram's method.

Glucose.—Growth similar to Sabouraud but slightly less abundant; the black pigmentation develops from the centre of the colonies and slowly spreads towards the periphery.

Ordinary Laboratory Agar.—Growth much less abundant than on most sugar agars, and black pigmentation less marked.

Lævulose Agar.—Identical to glucose.

Saccharine Agar.—The pigmentation is less pronounced, and does not spread to the whole of the growth.

Raffinose Agar.—Same as saccharine.

Lactose Agar.—Scanty pigmentation.

Alkaline Maltose Agar.—Black pigmentation well marked, though in many cases it does not extend to the whole of the growth.

Acid Maltose Agar.—Growth less abundant than on acid maltose; black pigmentation well marked.

Mannite Agar.—As alkaline maltose.

Inulin Agar.—As alkaline maltose, but pigmentation less pronounced.

Saccharose.—As inulin.

Glycerine Agar.—Abundant growth, the whole of which after a time becomes of jet-black colour.

Galactose.—As inulin.

Adonite.—Like acid maltose.

Serum.—Growth fairly abundant, but there is only a trace of pigmentation. The medium is not liquefied.

Gelatine.—No liquefaction; the growth on the surface shows after a time some dark pigmentation, but the colonies along the stab are white.

Milk.—No change.

Broth.—General turbidity; a thin pellicle is often present. The microscopical examination shows cocci arranged in pairs or irregularly; they are not capsulated.

Peptone Water.—Some growth at the bottom, while the rest of the tube is clear.

Sugar Broths.—No formation of acid or gas.

Indol.—Most strains produce a trace of indol.

Characters of the Coccus-like Organism found in the Red Variety of the Affection.—The coccus found in the red variety is more difficult to isolate and grow than the coccus observed in the black type of the affection: as a rule it grows better and shows more pigment on ordinary agar than on sugar media. It is non-motile and Gram-positive.

Agar.—The growth is at first white, then a red or red-yellowish spot appears in the centre. The pigmentation very slowly progresses towards the periphery, but, in my experience, never spreads to the whole of the growth ; hence I was inclined for a long time to believe that I was growing two different cocci ; repeated plating, however, has convinced me that this is not the case. On maltose and glucose agar the same pigmentation is present, but on most of the other sugar media no pigment is produced. Gelatine and serum are not liquefied. This coccus, as already stated, is Gram-positive and non-motile.

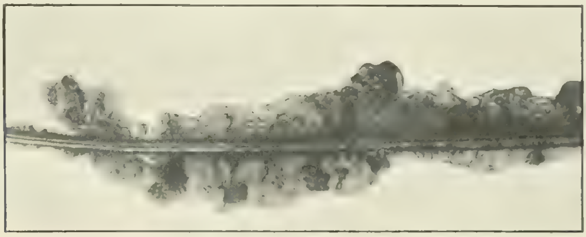

FIG. 1.
Trichomycosis flava.

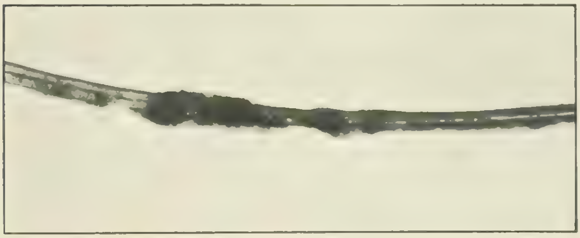

FIG. 2.
Trichomycosis nigra.

The coccus is closely allied culturally to *Micrococcus ruber* of Trommsdorff, found in cases of chromidrosis, and also to *Micrococcus rubicus* of Hefferan. In none of my cases, however, was the condition associated with chromidrosis.

Symptomatology.—The affected hairs of the axilla present nodular formations, plainly visible to the naked eye, of rather soft consistency ; they are easily removed by scraping with a triangular needle or any similar instrument. The formations are either yellow or black, or less frequently red ; they may be very abundant, and form a yellow, or black or red, sheath round the hair (figs. 1 and 2). The same patient may have two varieties; the hairs of one arm may show the yellow variety, while the hair of the other armpit may present the black type; sometimes the same individual hair may present some of the nodules yellow and others black, or, rarely, red. I have not yet observed all the three varieties present at the same time on the same patient.

The microscopical examination with a low power shows that the affected hair is covered at several places by roundish formations, partially or totally encircling the shaft. Using a higher power, these formations will be seen to consist, in the yellow variety, of enormous numbers of bacillary-like bodies embedded into an amorphous cementing substance; in the red and black varieties, in addition to these masses of bacillary bodies and mycelial segments of the discomyces, large groups of cocci-like bodies are observed. The affection was never associated with chromidrosis.

Diagnosis.—The condition must be differentiated from other nodular parasitic conditions of the hair. As is well known, the principal nodular affections of the hair of parasitic origin are:—

(1) *Trichosporosis tropica*, or *piedra*.

(2) *Unna's trichosporosis*, or *piedra nostras*.

(3) *Behrend's trichosporosis*, or "*nodular trichomycosis*."

(4) *Beigel's trichosporosis*, or *tinea nodosa*.

(5) *Du Bois's trichosporosis*.

(6) *Lepothrix*, or *Pick's trichomycosis palmellina*, or *trichomycosis nodosa* of temperate zones.

Trichosporosis tropica, or *piedra*, investigated by Morris Osario, Magalhaes, and recently by MacLeod, generally affects the hair of the head; the nodules are extremely hard, hence the name "piedra." The fungus found belongs to the genus *Trichosporon* (*Trichosporon giganteum*, Behrend, 1890). There are probably several varieties of piedra, due to different varieties of trichosporon; one such has been recently described by Horta in Brazil.

Unna's trichosporosis, or *piedra nostras*, has been described by Unna in the hair of the moustache and beard. It is due to *Trichosporon ovale* (Unna, 1896).

(3) *Behrend's trichosporosis*, or "*nodular trichomycosis*," described by Behrend, affecting the hair of the beard. It is due to *Trichosporon ovoides* (Behrend, 1890).

(4) "*Beigel's trichosporosis*," or "*tinea nodosa*," discovered in London by Cheadle and Morris, and later in Breslau, Nancy, &c. It attacks the hairs of the head and is due to *Trichosporon Beigeli* (Rabenhorst, 1867).

(5) *Du Bois's trichosporosis* of the hairs of the pubic region due to *Trichosporon glycophiles* (Du Bois, 1910).

(6) *Lepothrix* (E. Wilson), or *trichomycosis nodosa* (Patterson), or *trichomycosis palmellina* (Pick). In this condition, as described by Paxton, Wilson, Pick, and later by Payne, Patterson, Crocker, Pusey, &c., the hairs present irregular, lobed masses of hard consistency, in which are often imbedded some of the fibres of the cortex; according to Crocker the fibres of the whole shaft may be split up and the hair may break off with a brush-like termination. The researches on the ætiology of this condition seem to have given widely different results: Payne and Patterson described a short bacillus which penetrates under the cortical scales. Eisner describes a diplococcus, his results being confirmed by Sonnenberg; Colombini has found cocci; Babes, Pick, Balzer and Barthemly, having found the condition often associated with red sweat, consider the bacillus found to be the *Bacillus prodigiosus*.

With which of the above conditions can the nodular affection I have observed in Ceylon be identified? It certainly has nothing to do with piedra or with any other form of trichosporosis, as in my cases no trichosporon is found. The fungus being a *Nocardia* with very thin, bacillary-like mycelium, the condition most closely resembles, as I stated in my first paper, the lepothrix. It differs from the typical lepothrix of temperate zones by the nodules being soft, easily removed, and by the hairs not becoming brittle. Moreover, it is easily cured.

Course, Prognosis, and Treatment.—The course is chronic, but the condition may subside or disappear on the patient going to a cold climate. The affection, if of very little pathological importance, has a certain practical interest, being much objected to by patients, especially by ladies. The treatment is not difficult; a very efficacious one consists in dabbing the hair two or three times daily with a solution of formalin in spirit (1 dr. to 6 oz.) and applying at night a sulphur ointment (2 to 5 per cent.).

Note on Copra Itch.

By Aldo Castellani, M.D.

For several years past I have noticed, in Ceylon, in people working in copra mills a peculiar eruption, which, for convenience sake, I called "copra itch." Copra, as is well known, is derived from coconuts. The first impression on seeing a patient suffering from the condition is that he is suffering from scabies, except that no burrows are present. The hands, arms, legs, and sometimes the whole body except the face present fairly numerous, extremely pruriginous papules, often covered by small bloody crusts due to scratching; papulo-pustules and pustules are generally also present. The eruption begins as a rule on the hands, and from there spreads to the arms, legs, and trunk; it never affects the face. The eruption has very little tendency to heal spontaneously, at least while the patient continues working in infected mills and handling copra.

Ætiology.—In a patient suffering from the eruption who came to see me one day immediately on leaving the mills I noticed on one of his arms two tiny white specks moving about. I picked them out and examined them microscopically, when I saw they were acari-like parasites. On questioning the patient he told me he had often observed small whitish bodies moving about in copra dust. I asked him to bring me some copra dust, and he very kindly did so. The copra dust was swarming with minute white bodies, which on microscopic examination appeared to be identical with the two I had found on his body. Since then I have examined many other samples of copra containing the same mite. According to the zoologist to whom I have given specimens it is not a sarcoptes. Mr. Stanley Hirst describes it as a new variety of *Tyroglyphus longior (Tyroglyphus longior*, Gerv., var. *Castellanii*, Hirst). The mite of copra itch does not appear to bury itself in the skin; it apparently induces the dermatitis in the same manner as *Pediculoides ventricosus* (Newport), which lives in diseased cereals, and produces an eruption in persons handling such cereals; but further investigation is necessary to settle this point.

Experimental Reproduction of the Disease.—I have made repeated experiments in persons who have volunteered. By rubbing in copra dust containing the mite itching frequently begins shortly after, and twenty-four to forty-eight hours later an extremely pruriginous papuloid eruption often develops. The same result is obtained by picking out of copra dust the mites and placing them (alone without any dust) on the skin under a covering such as a piece of lint kept in place by a bandage. The pustular stage did not occur, but this being due to scratching and secondary pyogenic infections, there was not time for it to develop, all the persons refusing to continue the experiment after the second day. A few individuals seem to be unaffected by the presence of the sarcoptes or the copra dust containing it.

Diagnosis.—As already stated, the eruption on superficial examination may be mistaken for scabies ; true burrows, however, are always absent, and the two parasites are totally different.

Course and Progress.—The eruption has very little or no tendency to heal spontaneously while the patient goes on working with the infected copra. If the patient abstains from his work for some time the condition disappears spontaneously.

Treatment.—Beta-naphthol ointment (5 to 10 per cent.) is, in my experience, very useful. The mode of action of the naphthol in these cases is not quite clear : its beneficial effects cannot be solely due to its parasiticide properties, because the copra acari remain for only a short time on the body, and in most cases, when the ointment is applied at night, the parasites are no longer on the skin of the patient. It may act as an antipruritic antiseptic agent in this way, diminishing scratching and secondary pyogenic infections; it is probable, also, that a little ointment may remain on the skin after the morning bath, and be repellent to the copra mite, and in this way prevent reinfection.

Report on the Mite causing the Copra Itch.[1]

By STANLEY HIRST.

DR. ALDO CASTELLANI has kindly allowed me to examine his specimens of the mite which is the cause of the copra itch in Ceylon. This acarus belongs to the genus *Tyroglyphus*, and resembles *Tyroglyphus longior*, Gerv., exceedingly closely in structure. I think it is a new variety of *Tyroglyphus longior*, and I propose the name *Castellanii* for this variety. A comparison between this new variety and the typical form of the species is given below.

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[1] Published by permission of the Trustees of the British Museum.

The mites of the family Tyroglyphidæ chiefly feed on dried vegetable and animal substances, and are often present in great numbers in cheese, cereals, and other products. A number of instances of these tyrogyphid mites attacking human beings are known, and several species have been recorded as causing eruptions or other affections of the skin of the persons attacked (a list of these species is given in Professor Gedoelst's "Synopsis de Parasitologie," 1911, pp. 172-75). Sometimes the resulting effect is not very serious, and is only of a transient nature. The water itch of Indian coolies on the tea plantations, which is also caused by an acarus of this family (*Rhizoglyphus parasiticus*, Dalgetty), seems, however, to be a serious complaint.

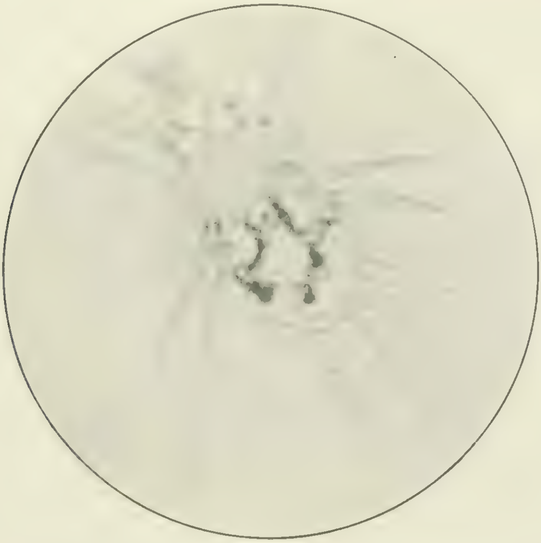
Tyroglyphus longior, Gerv., var. *Castellanii*, var. nov.

This new variety differs from the typical form in the following respect: The male of the typical form of the species has a pair of rather short hairs on the ventral surface of its body, some distance behind the anal suckers, and these two hairs are very much shorter than any of the other hairs at the hinder end of the body. In the male of the variety *Castellanii* there is no pair of short hairs in this position, even the shortest of the hairs of the seven pairs at the posterior end of the body being comparatively long.

The following details of structure may also be useful: The hairs of the body appear smooth, unless carefully examined under a rather high power of the microscope, and then they are seen to be very slightly feathered. There is the same number of hairs on the cephalo-thoracic part of the body as in the typical form of *Tyroglyphus longior* and they are similar in appearance, the hairs of the inner pair (of the two hinder pairs) being considerably longer than those of the outer pair. With the exception of the difference described above, the hairs on the abdominal part of the body are also similar to those of the typical form. (The hairs on the dorsal surface of the male are not shown correctly in Michael's figure of this species.) Tarsi of legs apparently of much the same length as in the typical *Tyroglyphus longior*; the usual two little projections are present on the upper surface of the fourth tarsus of the male.

Length of male, 0·3 mm.; of female, 0·4 mm.

Note.—I have carefully compared Dr. Castellani's specimens with freshly procured examples of *Tyroglyphus longior* found living in Gorgonzola cheese purchased in London. The feathering of the hairs was easily seen in specimens examined alive under the microscope. According to Michael, *Tyroglyphus longior* has a very wide distribution in Europe and is found on most kinds of dried or preserved animal and vegetable matter. He says that it is found in almost all houses upon dried provisions, often swarming in enormous numbers. Many other details of its habits are given in Michael's account of this species (*see* "British Tyroglyphidæ," vol. ii, pp. 123-31).



Tyroglyphus longior, Gerv., var. *Castellanii* (Hirst).

Note on the Ætiology of some Tropical Dermatomycoses (Tinea cruris, Tinea flava et nigra, Tinea imbricata).[1]

By Aldo Castellani, M.D.

Skin diseases due to fungi are extremely common in the Tropics; the damp, hot climate of most tropical countries being very favourable to the growth of vegetal parasites. The dermatomycoses observed in the Tropics may be classified in the following groups:—

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[1] Illustrations, drawings, and photographs of the various dermatomycoses and their fungi will be found in the second edition of the "Manual of Tropical Medicine" (Castellani and Chalmers).

TROPICAL DERMATOMYCOSES.

Genera	Species	Condition caused
(I) Due to fungi of the genera *Saccharomyces*, *Cryptococcus* (Kitzing), *Zymonema* (Beurmann)	*Saccharomyces Gilchristi* (Viellemin) and other species	Blastomycosis
(II) Due to fungi of the genus *Sporotrichum* (Link, 1809)	*Sporotrichum Beurmanni* (Matruchot and Ramond, 1908); *Sporotrichum asteroides* (Splendore, 1910); *Sporotrichum indicum* (Castellani, 1908)	Sporotrichosis
(III) Due to fungi of the genus *Pityrosporum* (Sabouraud, 1895)	*Pityrosporum Cantlici* (Castellani, 1898)	Tropical seborrhœa of children
(IV) Due to fungi of the genus *Hemispora* (Viellemin, 1906)	*Hemispora stellata* (Viellemin, 1906)	Hemisporosis
(V) Due to fungi of the genera *Aspergillus* (Micheli, 1727) and *Penicillium* (Link, 1809)	*Aspergillus barbæ* (Castellani)	Aspergillosis of hairy parts
	Penicillium barbæ (Castellani)	Penicilliosis of hairy parts
(VI) Due to fungi of the genera *Aspergillus* (Micheli, 1725); *Penicillium* (Link, 1809); *Monilia* (Persoon, 1801); *Montoyella* (Castellani, 1907)	—	Pinta
(VII) Due to fungi of the genus *Trichosporum* (Behrend, 1890)	*Trichosporum giganteum* (Behrend)	Piedra
(VIII) Due to fungi of the genera *Nocardia (Toni and Trevisan)*; *Madurella* (Brumpt, 1905); *Indiella* (Brumpt, 1905); *Aspergillus* (Micheli, 1725); *Oospora*; *Sporotrichum*	—	Madura foot
(IX) Due to fungi of the genus *Malassezia* (Baillon, 1889)	*Malassezia tropica* (Castellani)	Tinea flava
(X) Due to fungi of the genus *Microsporoides* (Neveu Lemaire, 1906)	*Microsporoides minutissimus* (Burchardt)	Erythrasma
(XI) Due to fungi of the genus *Foxia* (Castellani, 1908)	*Foxia Mansoni* (Castellani)	Tinea nigra
(XII) Due to fungi of the genera *Epidermophyton* (Sabouraud, 1907) and *Trichophyton* (Malmsten, 1845)	*Epidermophyton cruris* (Castellani); *Epidermophyton Perneti* (Castellani); *Epidermophyton rubrum* (Castellani); *Trichophyton nodoformans* (Castellani)	Dhobie itch
(XIII) Due to fungi of the genus *Trichophyton* (Malmsten, 1845)	*Trichophyton albiscicans* (Nieuwenhuis)	Tinea albigena
	Trichophyton Blanchardi (Castellani)	Tinea Sabouraudi
(XIV) Due to fungi of the genus *Endodermophyton* (Castellani, 1908)	*Endodermophyton concentricum* (Blanchard); *Endodermophyton indicum* (Castellani)	Tinea imbricata
	Endodermophyton Castellanii (Berry)	Tinea intersecta

From the above table it will be seen that tropical dermatomycoses *sensu strictu*—viz., occurring only in the Tropics—are comparatively few; most of them are endemic also in temperate zones, though occurring there rarely, or, at any rate, less frequently than in the Tropics. I may mention tinea cruris, madura foot, &c. The same remark, however, applies to every other branch of tropical medicine.

Moreover, owing to the enormous increase in traffic and intercourse between tropical countries and Europe, diseases have been imported into Europe from the Tropics and vice versa.

The subject of dermatomycoses in the Tropics being a very large one, I propose in this paper to limit myself to some brief remarks on the ætiology of only a few of them—those in which I have been most interested during my stay in Ceylon:—

(1) Tinea cruris.
(2) Tinea flava et nigra.
(3) Intertrigo blastomycetica.
(4) Tinea imbricata.

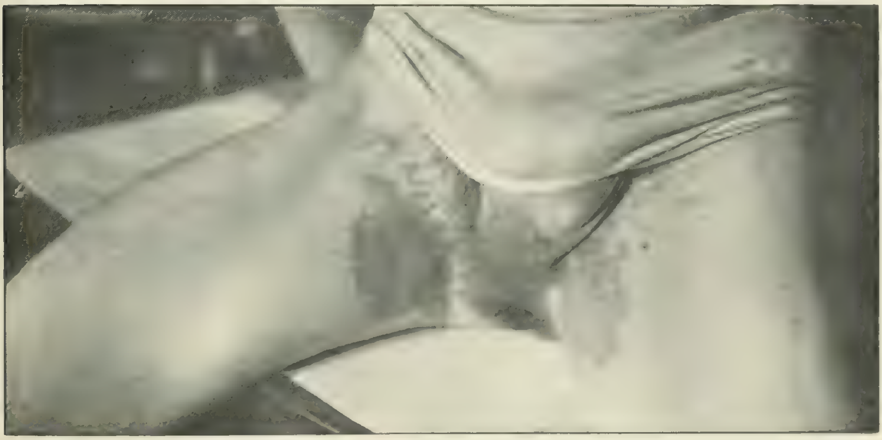

FIG. 1.

Tinea cruris.

Tinea Cruris (*Dhobie Itch*).—Tinea cruris, under the name of dhobie itch, Burmah itch, &c., has been known to tropical practitioners for a great many years (fig. 1). In 1905 I suggested that it should be separated from the ordinary forms of tinea corporis, and MacLeod suggested the name of tinea cruris. For the fungus most frequently found in such cases characterized by the yellowish cultures, I suggested shortly after the term "*Trichophyton cruris*." Pernet found and described a trichophyton, which I later named *Trichophyton Perneti*.

In 1907 Sabouraud, in a masterly manner, investigated the condition in France, which he called *tinea inguinalis*. There is no doubt that Sabouraud's "tinea inguinalis" is the dhobie itch of tropical authors.

or what MacLeod and I called *tinea cruris*, as Dr. Sabouraud and Dr. Pinoy, having kindly examined my cultures, have come to the conclusion that *Epidermophyton inguinalis* and *Epidermophyton cruris* are the same fungus.

Though *Epidermophyton cruris* is the fungus most frequently observed in tinea cruris, it is not the only one which can give rise to the condition; tinea cruris may be caused by several species of fungi, each of which gives rise to a slightly different clinical variety of the disease. Up to the present time I have observed the following organisms: *Epidermophyton cruris*, *Epidermophyton Perneti*, *Epidermophyton rubrum*, *Trichophyton nodoformans*.

Epidermophyton cruris, Cast., 1905, syn. *Trichophyton cruris*, 1905, *Epidermophyton inguinale*, Sabouraud, 1907, *Trichophyton Castellanii*, Brooke, 1908.—This is, as already stated, the commonest species. The fungus is very abundant in recent cases, but is extremely scarce in cases of old standing. The mycelial tubes in fresh cases are generally straight, have often a double contour, and the segments are rectangular, their breadth being 3½ to 4½ microns; branching is not rare. The spores are rather large (4 to 7 microns), roundish, and present a double contour; they never collect in clusters. In chronic cases degeneration forms of the fungus are met with; the mycelium may be banana-shaped, presenting several constrictions, or long strings of ovoid elements may be seen. The fungus grows well, but rather slowly, on Sabouraud's agar; the growth begins to be visible after four to eight days, the colonies being at first of peculiar yellowish colour; later they may become white with pulverulent surface. Pleomorphism develops very quickly.

Epidermophyton Perneti, Cast., 1907.—This fungus was first described by Pernet. It grows more rapidly than *Trichophyton cruris*, and the cultures have a delicate pinkish colour, which is generally lost in subcultures. It is very rare in Ceylon.

Epidermophyton rubrum, Cast., 1909 (syn. *Trichophyton purpureum*, Bang, 1910).—This fungus was described by me in Ceylon in 1909, and later, independently, by Bang in Europe, under the name of *Trichophyton purpureum*. In microscopical preparations in liq. potass. from the affected parts mycelial tubes are seen, and free spores identical to what one sees in *Epidermophyton cruris*, and very similar to any trichophyton of the *megalosporon* type.

Cultures: Sabouraud's Agar.—The growth begins to appear four to six days after inoculation as a raised red spot, which gradually enlarges. The full-grown colonies are of a deep red colour, either with a central knob or crateriform, and are partly covered by a delicate white duvet. In old colonies the white duvet is more abundant and thicker. It may hide the red pigmentation almost completely.

Glucose Agar.—This is the best medium for the growth of the fungus. The cultures are of a very deep blood-red colour, and the pigmentation may spread to portions of the medium itself. In old cultures abundant white duvet is present, and this may hide the pigmentation, but on scraping out this duvet the red pigmentation will be found to be still well marked.

Ordinary Agar and Glycerine Agar.—The fungus grows fairly well, but there is no red pigmentation.

Trichophyton nodoformans, Cast., 1910.—On Sabouraud agar the colonies have a white powdery surface, with small central knob. The growth slowly deepens in the medium, and the submerged portion has a characteristic brick-red colour, which generally disappears after repeated transplantations.

Glucose Agar.—Growth somewhat more abundant than on Sabouraud agar, but the peculiar brick-red pigmentation of the submerged growth is usually absent.

Glycerine Agar.—Fairly abundant growth; no pigmentation.

Ordinary Agar.—Scanty growth of whitish colour.

Although in this paper I do not propose to enter into any clinical details, I may say that *Epidermophyton cruris* causes the commonest and best known type of tinea cruris, as described by all tropical authors, and in Europe by Sabouraud; characterized by large festooned patches, with elevated margins on the scrotum, perineum and inner surface of the thighs.

In one of my papers published in the *British Journal of Dermatology* in 1910 (p. 147), I stated that it is an error to consider *Epidermophyton cruris* as always localized to the groin and the armpits. In many cases it spreads to other parts of the body, excepting only the scalp; indeed, the affection may first start on the chest or arms, &c., and then spread to the groins and armpits, or may not even affect these regions. Hence, perhaps, the terms "tinea cruris" and "tinea inguinalis" are not altogether appropriate; the term "tinea tropicalis" might be suggested. Sabouraud and Whitfield have made the important observation that *Epidermophyton cruris* often produces a peculiar type of pruriginous dermatitis of the toes.

In the cases of tinea cruris due to *Epidermophyton rubrum* the patches present, often from the very beginning, an eczematoid appearance; the edge is perhaps not so elevated, but very abrupt, dotted by numerous papules often covered by minute bloody crusts due to scratchings. The lesions are arranged in complete or incomplete rings, sometimes very large, but solid patches may also be present. This type of eruption has the greatest tendency to spread from the scroto-crural regions to other parts of the body.

Trichophyton nodoformans gives rise to a peculiar type of tinea cruris with very thick, elevated margins and along the edge deep-seated nodules resembling, to use a popular expression, "blind boils." It has pyogenic properties and may attack the hair-follicles. Dr. Chalmers and myself once saw a case presenting a nodular eruption due to this fungus in the groins, and at the same time presenting a patch of sycosis due to the same organism on the right cheek.

Tinea Capitis.—In Ceylon, while dermatomycoses of every kind are extremely common, tinea capitis is comparatively rare. All the cases I have seen were of the same type and due to the same *Endo-ectothrix trichophyton*; the scalp presented in all cases numerous white patches covered by an enormous number of whitish squamæ. An interesting fact is that the patches remain bald permanently. The fungus is practically identical with *Trichophyton violaceum*, and therefore there is no need to give a special description of it.

Intertrigo saccharomycetica, syn. *Intertrigo blastomycetica.*

Remarks.—Cases of this affection have been observed by me in Ceylon several years ago, and one similar has been reported by Whitfield in England. The affection is apparently rare. It generally attacks the scroto-crural and axillary regions. The affected skin is red, and there may be slight exudation. The borders of the eruption are fairly well marked, but never elevated. In most cases there is not much itching, and the affection may recover spontaneously.

Etiology.—In scrapings a saccharomyces (*Saccharomyces Samboni*, Cast., 1907), which is easily cultivated on sugar media, is found. According to recent investigations the fungus should be placed in the genus *Monilia*.

Treatment.—The treatment consists in washing the affected parts with potassium permanganate lotion 1 in 4,000, or resorcin 1 in 100, followed by the application of powders of boric acid and talc.

Tinea flava.—This dermatomycosis is confused by several authors with the pityriasis versicolor of temperate zones, but the researches of Jeanselme and myself tend to prove, I think, that it is a separate entity. The disease, which is extremely common in Ceylon, is characterized by the presence of bright yellow patches found, in order of frequency, on the face, neck, chest, abdomen, arms, and legs; it is of very difficult cure. The fungus is a malassezia (*Malassezia tropica*, Cast., 1905) which, microscopically, can hardly be distinguished from *Malassezia versicolor*; it is not cultivable. In fresh preparations in liq. potass. one sees mycelial threads 3 to 5 microns wide, with numerous swellings, constrictions and other irregularities; spores roundish, 4 to 5 microns, with double contour, often collected in clusters.

Tinea Nigra.—This dermatomycosis was first described by Manson in China in 1870, but his observations were forgotten as they were not quoted by him in his subsequent publications. It was re-described by me in Ceylon in 1905. It is characterized by the presence of black patches due to a fungus belonging to a new genus which I called "*Foxia*," in honour of the distinguished scientific dermatologist Dr. Colcott Fox. The fungus, *Foxia Mansoni*, in fresh preparations from the affected parts, shows mycelial articles, straight or variously bent and shaped, 2½ to 3½ microns wide, non-ramified; spores large, globular, 5 to 10 microns in diameter, collected in clusters. The fungus is easily grown on maltose and glucose agar, also on ordinary agar, giving rise to greenish-black colonies, which later fuse into a jet-black mass. It slowly liquefies gelatine.

Tinea Imbricata.—As I propose to give shortly a full account of this interesting malady in the *British Journal of Dermatology*, I will limit myself here to only a few remarks. As is well known, the ætiology of this dermatomycosis has been, and still is, the subject of numerous controversies. Manson first, in 1872, described a trichophyton-like organism in the squamæ; with the laboratory technique of that time attempts at cultivation did not succeed. Blanchard considered it non-cultivable and called it "*Trichophyton concentricum*"; on the other hand, Nieuwenhuis stated that it was quite easily cultivated and was characterized by the colonies being crateriform. In recent years the general opinion has been that aspergillus-like fungi were the real cause of the disease. Tribondeau described fructifications somewhat similar to those of an aspergillus and created for the fungus the genus *Lepidophyton*. Wehmer has described it as a true aspergillus *Aspergillus Tokelau*. From the investigations I have carried out in Ceylon, I think

I am justified in stating that the aspergillus and aspergillus-like fungi have nothing to do with the disease. When they are present in the squamæ they are merely saprophytes or contaminations. By using a special technique I have succeeded in growing what I consider to be the true fungi causing the disease: they are not trichophytons, they resemble more—according to Sabouraud, who has kindly examined my cultures—the achorions. According to all probabilities they will have to be placed in a separate genus, for which I have suggested the term "*Endodermophyton.*" I have isolated so far two species, *Endodermophyton concentricum* and *Endodermophyton indicum.*

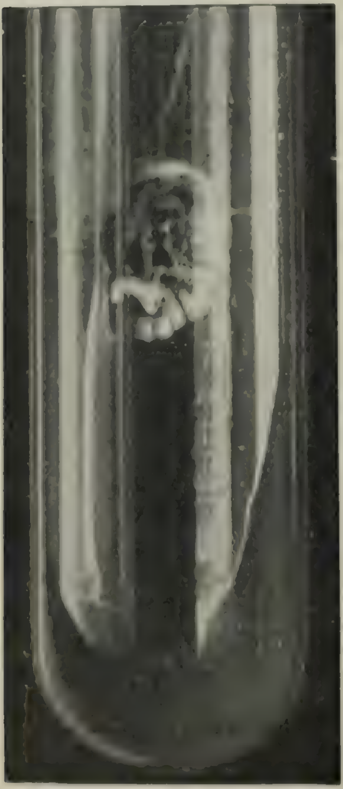

Fig. 2.

Endodermophyton indicum (glucose agar).

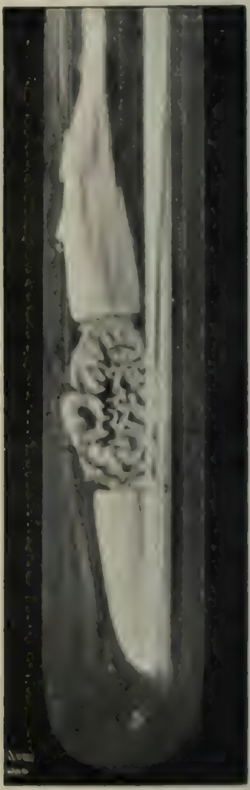

Fig. 3.

Endodermophyton concentricum (glucose agar).

Endodermophyton concentricum on glucose agar (4 per cent.) shows a growth with cerebriform or crinkled surface (fig. 2). The growth and medium show a slight amber colour, which later may become of a much deeper hue. In recent cultures duvet is absent; after repeated transplantations, however, a little duvet appears.

Endodermophyton indicum on the same medium (glucose agar 4 per cent.) shows a growth with surface somewhat convoluted or furrowed (fig. 3); in most cultures a portion of the growth, generally the central portion, is of a deep orange colour, or red-orange, or pink-orange; the rest of the growth appears white and powdery, being covered by a very short, delicate duvet; some cultures are of a beautiful red colour with very little or no duvet. By inoculating either of the above fungi I have been able to reproduce the disease typically in human beings. I venture, therefore, to state that the two fungi I have described are the true ætiological agents of the disease. *Endodermophyton indicum* seems to give rise to a less severe type of the malady than *Endodermophyton concentricum*, but further investigation is necessary on this point.

Two Cases for Diagnosis.

By S. E. Dore, M.D.

Case I.—The patient was a woman, aged 41, who presented accurately symmetrical, oval or circular, eczema-like patches on the lateral surfaces of the neck, on the front of the chest, in the axillæ, flexures of the arms, groins, popliteal spaces, and on the soles of the feet. There was also a linear patch on the right shin. The lesions were of a bright pink or vermilion colour when seen by daylight, and had clearly defined margins. There was slight thickening of the skin in some of the patches, and no exudation or scaling. The duration of the complaint was three months, and had resisted all kinds of local treatment. It was accompanied by intense itching, which was said by the patient to precede the eruption. The distribution of the lesions was similar to that of flexural eczema, but some of the features of the case suggested the possibilty of an "artefact."

Case II.—The patient was a woman, aged 49, who had suffered for about two months from an eruption of small oval or circular patches on the back and extensor surfaces of the limbs. The lesions consisted of minute rounded, shiny, closely aggregated follicular papules of a pale red to crimson colour. There was also slight general prominence of the follicles on the extensor surfaces of the body. Some of the patches, as on the shins, were circular or irregular in shape, and the papules were less closely aggregated and of a darker colour. The duration of the eruption was from six to eight weeks. It was attended by intense itching of a paroxysmal character. The case was thought by the exhibitor to be an example of lichen simplex (Vidal) or diffuse névrodermite (Brocq).

The PRESIDENT said it was very difficult to give the condition the correct name, and many names had been applied to it. There should be some uniformity of nomenclature for these conditions, which were quite common.

Case of Generalized Macular Pigmentation of Trunk and Extremities.

By J. M. H. MacLeod, M.D.

(For W. T. Freeman, M.D.)

THE following are Dr. Freeman's notes of the case: The patient was a well-developed girl, aged 12. She was brought to the Skin Department of the Royal Berkshire Hospital on Tuesday, October 22, and was sent for diagnosis. The chest, abdomen, and back, and both limbs were covered with a darkly pigmented macular rash (figs. 1 and 2), the macules accounting for about 50 per cent. of the total skin surface, and the remaining area showing a shade of lightish fawn colour. The face was affected to a much less degree. At the examination this mere child was found to be four months pregnant.

In December, 1909, and January, 1910, this child and her brother were under treatment at the hospital for psoriasis. The psoriasis cleared up then very quickly, and she was discharged cured on January 15, 1910. The treatment included moderate doses of arsenic.

In August of this year, when probably she was about one month pregnant, she was treated for anæmia by her doctor at home, and the treatment included for about four weeks small doses of liq. arsenicalis.

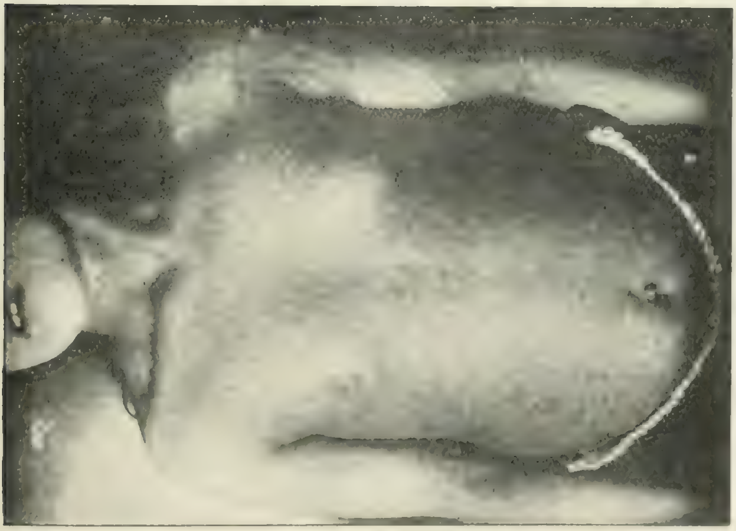

Fig. 1.

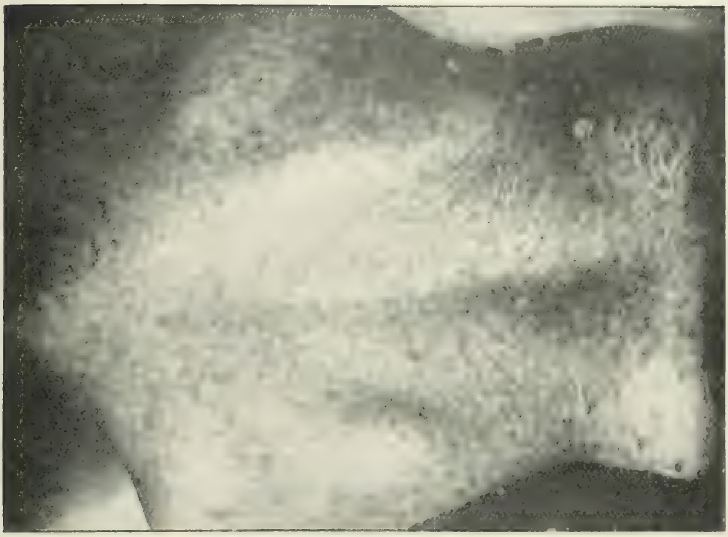

Fig. 2.

Generalized macular pigmentation of trunk and extremities.

It has to be noted that the arsenic given in 1909-10, up to 5 minims liq. arsenicalis, helped in the cure of the psoriasis, but without producing any after-effects. For this reason Dr. Freeman formed the opinion that the present rash, although probably accentuated by the administration of the arsenic in August, would not have appeared had it not been for the uterine condition. He therefore regarded it as a case of chloasma uterinum, an anomalous case, not unlikely modified by the giving of small doses of arsenic in the early stages of pregnancy in such a very youthful patient.

Case of (?) Dermatitis Herpetiformis.

By A. M. H. Gray, M.D.

The patient was a woman, aged 29, who eighteen months ago developed symmetrical painful patches on the inner aspect of each cheek. These were subsequently followed by more or less symmetrical patches on the abdomen and lower part of the chest. The appearance of the patches was preceded by intense burning, followed later by itching. The lesions themselves were blisters, varying in size from a lentil to a bean, and tended to be grouped and surrounded in most cases by an erythematous area.

The exhibitor considered the case to be one of "dermatitis herpetiformis," but it seemed to him to resemble cases described by Kaposi as "zoster atypicus et hystericus," though no evidence of self-production was forthcoming.

Dr. Pringle said he believed that almost all cases described as bilateral or otherwise atypical cases of herpes were nothing more than dermatitis herpetiformis.

Case of Erythema Multiforme of Unusual Type.

By E. G. Graham Little, M.D.

The patient was a lady, aged 34, now a few months pregnant. She had had the disease, with short intervals only of freedom, for seven to eight years. She had been sent to the exhibitor by Dr. Harold Clapham, of Peterborough, who kindly gave the following history: He had seen the case at its inception; the lesion then consisted of large blisters, which appeared especially in the spring and autumn, and occupied the joints of the arms and legs and the mucous membranes. The diagnosis of dermatitis herpetiformis was made, and the eruption invariably cleared up rapidly with arsenic. Two years ago the same kind of lesions appeared on the chest as well; these also cleared up with arsenic.

The patient had had several miscarriages in earlier married life and had not had living children during the past eight years.

The lesions when shown were large, limpet-shaped, erythematous and, in some places, vesicating patches, chiefly about the elbow, forearm, and neck. They were itchy, and latterly the patches had been more persistent.

In view of the earlier diagnosis, it is interesting to note that the lesions as now seen strongly resembled the plate of a rare skin eruption figured in the "St. Louis Atlas" (Plate X) as "dermatite herpetiforme en cocarde," and so described by Hallopeau. This was possibly in reality an erythema multiforme.

Case of Epitheliomatous Growth on the Foot.

By E. G. Graham Little, M.D.

The patient, a woman, aged 70, kindly sent by Dr. Clarke, of Horley, had a growth on the outer margin of the right foot near the sole. The tumour had commenced about eighteen months ago with a small "pimple," which had steadily grown larger. When shown she had a circular erosion of the bony layer about the size of half-a-crown, the whole centre of which was filled up by a red, non-ulcerative excrescence, moderately hard, not bleeding very readily on contact, and raised ½ in. above the level of the skin. No glands could be felt in the groin or popliteal space. The duration of the swelling and the absence of glandular enlargement suggested the diagnosis of epithelioma of the rodent type. The diagnosis of epithelioma was questioned by several members at the meeting, but since then a portion of the central mass of the tumour had been examined by Dr. E. H. Kettle, late Pathologist at the Cancer Hospital, and now at St. Mary's Hospital, who reports that the tumour is definitely epitheliomatous.

DISCUSSION.

Dr. GALLOWAY said that, judging by the appearance of the sore on the heel, he thought the diagnosis rested between a granulomatous tumour due to some chronic septic infection or a malignant growth. He wished to mention the case of a patient in whom a very remarkable development of carcinoma of the skin of the right leg and thigh had commenced with a sore closely resembling the one now demonstrated by Dr. Little. The patient was a woman, now aged about 55. Seven or eight years ago she had consulted her doctor on account of a button-like mass, resembling granulation tissue, on the heel. The doctor, thinking that the ulcer was of pyogenic origin, cleansed it and removed the excess of tissue. The ulcer, however, did not heal properly and a fungating mass once more grew. Dr. Galloway had seen the patient within the last few weeks, nearly eight years after these events had happened. The right foot, leg and thigh were now deformed by large nodular masses of new growth, so that scarcely any normal skin remained. These nodular, rounded masses, variously discoloured, of a pink or purple tint, in some cases projected from the original surface to the extent of two or three inches. The whole lower extremity was therefore greatly increased in size, and could be moved only with much difficulty. Very little ulceration, however, had occurred, even where the tumours formed the largest projecting masses, with the exception of the original sore, already mentioned, on the heel. The patient was ill, but not to an extent at all corresponding to the exceedingly dangerous aspect of the local growth. One of the glandular masses had been removed by his recommendation and he had made histological examinations. The tumours consisted of large masses of epithelial growth very regular in the distribution of its epithelial elements, resembling an adeno-carcinoma, such as was more familiar in the case of an organ like the liver. Though the gland-like masses of epithelium were large and in enormous quantity, very little degeneration of the growth was taking place. The tumour could be compared to a gigantic exaggeration of the type of growth seen in true rodent ulcer. The glands in the right groin were now becoming enlarged, but there was no distinct evidence up to the time when Dr. Galloway had seen this patient of visceral infection in the abdomen or thorax. He would like to mention to Dr. Little that the patient now before the Section suggested to him the early stages of the malady which he had seen in an advanced state in the patient described. The most remarkable features were the extensive and massive characters of the growths, their long duration, and the comparatively small interference with the general health of the patient.

Dr. WHITFIELD considered that in the absence of a histological diagnosis one was not justified in saying it was rodent ulcer, nor indeed in thinking it was malignant, because, as far as the finger could determine, there was no spread of the tumour beneath the horny margin outside; and the red material inside was soft. With regard to the case mentioned by Dr. Galloway, there was one with a similar history at the London Hospital years ago, sections of which were shown to him by Dr. Bulloch. It came from the dorsum of the foot, and was a typical sebaceous gland carcinoma. The secondary nodule was carcinoma, but of such appearance that it might have come from any part of the body.

Dr. A. EDDOWES said he considered this case to be one of erythema exudativum multiforme. He had seen some such cases last a short time, some come and go, and others in which the condition never thoroughly disappeared.

Case of so-called "Acne Agminata of Crocker."

By H. MACCORMAC, M.B.

THE patient was a man, aged 42, and there was nothing in the history except that during the last few months a brother had developed tuberculosis. A section was shown exhibiting typical giant cells, epithelioid cells, and other structures of tuberculosis. Inoculations into an animal had not been done. The papules were confined to the face, and had only recently shown necrosis with scarring.

DISCUSSION.

Dr. PRINGLE agreed with Dr. MacCormac's diagnosis and thought his microscopical sections quite convincing as to the tuberculous nature of the disease. Indeed, he could not but think that the demonstration of tubercle bacilli must be near at hand despite the negative findings of the exhibitor, of Dr. Galloway, and of a considerable number of previous investigators. He hoped that inoculations in animals as well as the usual clinical tests for tubercle would be carried out. Dr. Pringle was glad that the exhibitor had inserted the qualifying word "so-called" in his notice of the case announced as one of "acne agminata." This name, first used by Radcliffe-Crocker, was that by which the disease was most commonly known to British dermatologists, and as such had served its purpose of establishing the disease as a clinical entity. He had always considered it an extremely unfortunate name, as the lesions presented only the faintest superficial resemblance to acne, and only implicated sebaceous and sweat-glands incidentally. Nor was their arrangement necessarily or even prominently always "agminate." The French appellation of "acnitis" was barbarous, and, he believed, had generally been discarded in favour of tuberculide hypodermique à type nodulaire (Darier); but Tilbury Fox's original name of lupus disseminatus was equally close to the mark and less cumbersome. As to differential diagnosis, mistakes might be made with colloid disease—an example of which the speaker happened to have under his present observation—in which no spontaneous evolution of the nodules with pitting resulted. A condition fairly frequently mistaken, in the speaker's experience, for the tuberculide under discussion was that which he described some years ago as "a peculiar seborrhoide," severe cases of which were remarkably like it: and the resemblance was increased by the shallow pitting which resulted from the absorption of the seborrhoide. This was, however, only temporary, and the result of treatment soon and invariably cleared up all difficulty of diagnosis. The question of treatment was of great importance, and had hitherto, he believed, been unsatisfactorily solved. Piece-meal methods by *raclage*, &c., were slow and unsatisfactory; the little granulomatous nodules were obviously of low vitality, with so marked a tendency in this, as in many cases, to spontaneous disappearance that the suitability of X-rays had suggested itself to his mind as likely to effect good results with the minimum possible of scarring.

Dr. PERNET considered that this was an instance of Radcliffe-Crocker's acne agminata. In a case which he (the speaker) examined histologically he found the sweat-glands disorganized by leucocytic infiltration. Some of the hair-follicles were partially involved and there was peri-vascular infiltration. No tubercle bacilli were found. There was no so-called tubercular architecture. As to the nodule examined by Dr. Galloway and referred to by Dr. Pringle, that was another case altogether, as a reference to Radcliffe-Crocker's third edition would show.[1] In two cases of acne agminata under Dr. Pernet's observation there was a history of excessive sweating. In Dr. MacCormac's case the man's work led to sweating, too.

Dr. DOUGLAS HEATH said he had a case in a man of about the same age, in whom the grouping of nodules on the face was, as Crocker described it originally, particularly marked in the eyelids, and the nodules had a typical tubercular architecture, but he had been unable to recover tubercle bacilli from them. He had portions of the material injected into a guinea-pig, but without result. Therefore he could not say, from his own experience, whether the lesions were tuberculous or not.

Dr. WHITFIELD urged that one should be careful about making a diagnosis of tuberculosis, even where there was the most typical tubercular architecture. He called attention to a recent exhaustive treatment of the subject in an article by Schamberg, of Philadelphia, who examined the cases bacteriologically, and injected them with tuberculin. In most of the cases the reaction to tuberculin was negative. If one cut a number of sections of the skin of the acneiform type, it was extraordinary how one got epithelioid cells and giant cells surrounding a central area of necrosis, which latter, however, was not typical. And Gilchrist, a man with great experience, had shown many photographs of acne packed with what looked like tubercular giant cells. Therefore he did not think that, in the absence of experimentation, one was justified in saying a case was a tuberculide. He quite agreed that the name acne agminata must be given up now; he was assuming it was the same thing as acnitis of the face.

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[1] Radcliffe-Crocker, "Diseases of the Skin," 3rd ed., 1903, ii, p. 1097.

Extensive Case of Infective Angioma (Hutchinson).

By Sir MALCOLM MORRIS, K.C.V.O., F.R.C.S.Ed., and S. E. DORE, M.D.

THE patient was a healthy-looking girl, aged 24. Her parents and brother were healthy and there was no history of any skin disease. An uncle on the mother's side, however, had been troubled with a "bad circulation." Recently the patient had complained of aching of the right arm and of general fatigue and morning faintness. She occasionally experienced a feeling of numbness of the head in cold weather and never felt so well in winter. There was no morbus cordis. The disease began on the right arm at the age of 2 and had continued to spread slowly since that time. During the past three years it had spread more rapidly, especially on the front of the thighs. The lesions were distributed over the right hand and arm, terminating at the axilla, on the

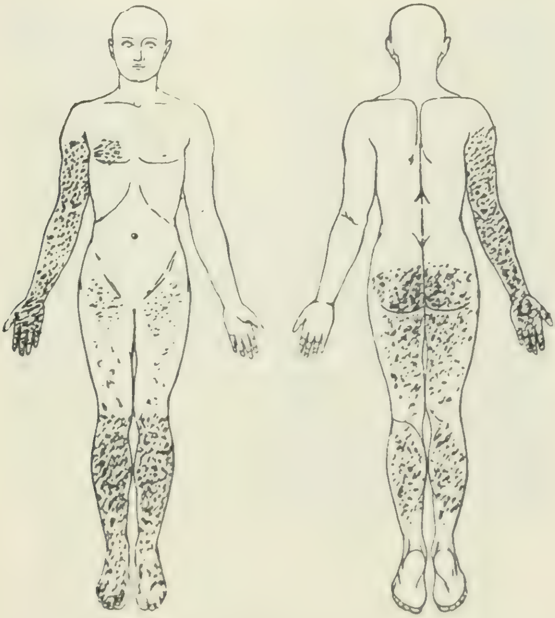

FIG. 1.

Extensive infective angioma.

right breast around the nipple, on both buttocks and anterior and posterior surfaces of the upper parts of the thighs, and on both legs and feet (figs. 1 and 2). The skin showed a red or purple mottling with minute telangiectatic "cayenne pepper" points. The right hand was œdematous and deeply cyanosed, and the feet were blue. She also suffered from chilblains of the feet. The patient stated that the patches became paler or disappeared when she was in a hot bath, but became blue afterwards. They were also made paler by pressure but did not completely disappear, except for the greater extent of the lesions. The case appeared to be in every way similar to the one shown by Dr. Sequeira, and published in the *British Journal of Dermatology* for October, 1912.[1]

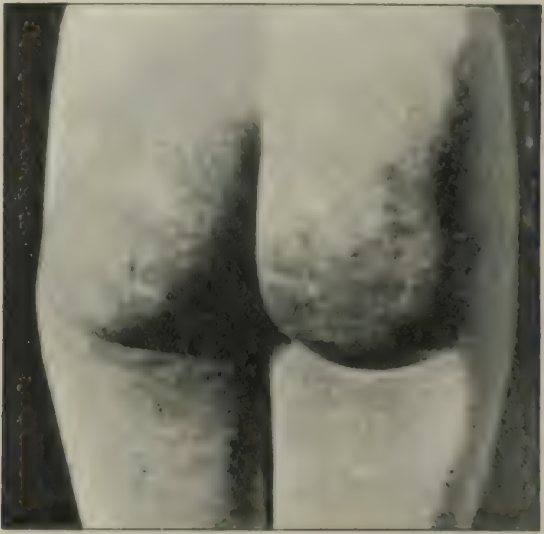

FIG. 2.

DISCUSSION.

Dr. SEQUEIRA said there was a later note in connexion with Sir Jonathan Hutchinson's case. The patient was now a married woman, and she still had the eruption, in which there had been very little alteration since.

The PRESIDENT replied that he believed there were no particular subjective symptoms in Sir Jonathan Hutchinson's case, but the present patient complained of pain in the arm, and there were symmetrical vasomotor disturbances, so that after taking a hot bath the area became blue. On pressure it almost disappeared, leaving only bright points. This was the most extensive case he had seen, it was on both legs and buttocks and the posterior parts of the thighs. In the early morning she had feelings of lassitude and depression. The heart and sensation were normal, but she felt worse in cold weather.

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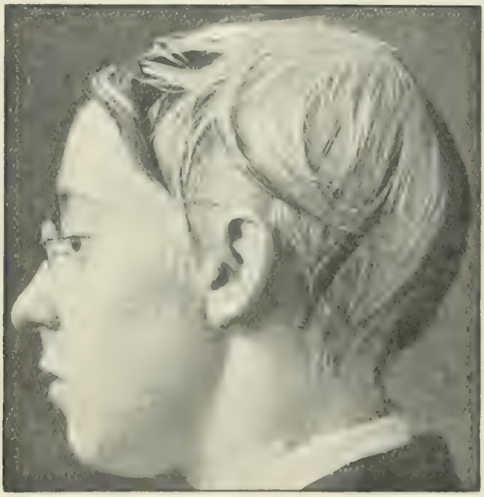
[1] *Brit. Journ. Derm.*, 1912, xxiv, p. 355.

Case illustrating a Sequel to Alopecia Areata.

By J. H. SEQUEIRA, M.D.

THE patient, an anæmic girl, aged 18, first attended the London Hospital in January, 1911. She was then suffering from alopecia of the whole scalp and eyebrows. The affection had started as an alopecia areata in several round areas which rapidly coalesced. Beyond the anæmia there was no evidence of general ill-health. The teeth were good and there was no dyspepsia. The patient was hypermetropic and had suffered from headaches, but the vision had been corrected by appropriate glasses and the headaches had ceased. Under gentle stimulation with a lotion containing oleum myristicæ in olive oil (1 in 4) and a course of iron and arsenic, the hair returned.



Sequel to alopecia areata.

On January 11, 1912, the condition was as represented in the accompanying photograph. The appearance was remarkable, the hair had grown to a fair length, but there were extensive areas of leucotrichia. This condition persisted until July last, when a great improvement in the colour of the hair was noticed. When shown at the meeting the hair was of good length and of normal colour, except for a few white hairs here and there. The patient was positive that very little of the white hair fell out, and the growth was so abundant that this seemed certain, and, moreover, she had been under the exhibitor's observation once a month throughout. It appeared that, in this case, the white hairs had become pigmented after growth. During the past year the only treatment has been by tonics internally.

DISCUSSION.

Dr. DORE said that until recently he had been under the impression that the white hair which grew after alopecia areata fell before the dark hair appeared. But he had a case not long ago in which the gradual extension of the pigmentation from the root to the distal end of the hair could be clearly observed.

Dr. WHITFIELD said that after the use of X-rays it was common for the hair to be fair and fine. It then darkened again. If such a case were seen eight or nine months afterwards, silky ends could be seen to coarse hairs. As the hair grew in strength it gained in pigment; but this case was interesting, as one must conclude from the dates that pigment had run down the hair.

Case for Diagnosis.

By A. WINKELRIED WILLIAMS, M.B.

THE patient, a woman, aged 32, an artist, sometimes had Raynaud's signs in her fingers, but had never had chilblains on hands. There was no history of rheumatism, of "growing pains," or anything of the kind; neither could anything be found in her occupation to account for it. Both hands were now affected with the lesions. There is a family history of gout on her mother's side. The affection began two years ago on the knuckles of the right little finger; six months later the other fingers of the right hand were similarly attacked. Left index-finger developed same condition twelve months ago, and successively the other fingers became affected; the little finger developed the condition four weeks ago. The condition is progressing, being more marked in the first finger affected. The affection consists of a soft growth in the corium and subcutaneous tissue, which stands out as prominent pads over the knuckles, but which, when pressed between the fingers, gives very little resistance and feels more like a bursa than anything else. There is no evident enlargement of the bones, no creaking or tenderness in the joints. The toes are not affected.

DISCUSSION.

Dr. F. PARKES WEBER remarked that in the *Quarterly Journal of Medicine* Dr. W. Hale White called these thickenings over the knuckles simply "pads on the finger-joints": he omitted the qualification "gouty." He gave the results of microscopic examination.[1]

Dr. WHITFIELD pointed out that Dr. Garrod had named the condition "gouty pads."

A Discussion on Erythema Multiforme.

Opened by H. G. ADAMSON, M.D.

THE subject selected for discussion this evening is "Erythema Multiforme."

The term "erythema multiforme" has probably for some of us a wider meaning than it has for others. The point of view I shall take is that it indicates a very definite and distinct affection possibly due to some one specific cause.

Erythema exudativum multiforme was first described by Hebra in 1876 by the name it now bears. Under the term "polymorphous erythemata" Hebra grouped together several diseases, all of which were characterized by erythematous eruptions with exudation of serum into the skin. This group of "polymorphous erythemas" included (*a*) erythema exudativum multiforme, (*b*) erythema nodosum, (*c*) roseola exudativum, and (*d*) urticaria. It should be noted that although he grouped these affections under the one name polymorphous erythema. Hebra regarded them as ætiologically distinct diseases and brought them together, in accordance with his general plan of classification, because they were eruptions having a similar pathological basis—namely, erythema with exudation. I mention this point because some confusion has arisen owing to the fact that many writers have failed to maintain the distinction between these eruptions, and have discussed their ætiology as though they were one disease—polymorphous erythema.

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[1] *See* Hale White, *Quart. Journ. Med.*, Oxf., 1908, vol. i, p. 479.

It is to the merit of Hebra to have recognized the affection he named erythema multiforme as a distinct malady, and to have rescued it from the confused mass of erythemas then described. Hebra's description is classical, and has served as the model for all subsequent descriptions.

As one of the most striking characters of the eruption, Hebra mentions its peculiar *distribution* upon the extremities. "In every instance," he says, "it is present on the dorsal surfaces of the hands or feet. In the more severe cases, but only in these, it may be observed on the forearms and legs, on the arms and thighs, and even on the trunk and face. It is, however, only in very exceptional instances that it affects the regions last mentioned, and when it is found on them it invariably exists also on the backs of the patient's hands, where, indeed, this cutaneous disease generally first appears."

"*The efflorescence*," he states, "consists of flattened papules or tubercles, of a dark blue or a brownish-red colour, between lentils and beans in size. Their number varies in different cases." He then describes how in slight cases the lesions rapidly fade, leaving a little pigmentation, while in other cases they spread into rings (erythema annulare), or into concentric rings (erythema iris), or form gyrate and marginate figures by expansion and blending of the rings (erythema gyratum seu marginatum). He insists that all these forms, papular, annular, gyrate, marginate, iris, previously described as distinct eruptions, were really phases of the same affection. Hebra also describes the vesicular or bullous form of this eruption, which he identifies with herpes iris of previous writers. As regards *subjective symptoms*, Hebra says that these are trifling: "Some patients complain of slight burning sensation, others of slight itching" or a feeling of tenseness when the papules are closely approximated.

"Concomitant and febrile symptoms are to be observed only in exceptional cases, in those cases, namely, in which the affection spreads over large tracts of the surface, or even over the whole skin. No important complications, or sequelæ, occur in the train of this eruption. Its whole duration varies between one and four weeks." Once only Hebra observed the erythema papulatum accompany a pneumonia of which the patient died.

The liability of the eruption to relapse and its tendency to occur during the months of April and May, and October and November, are mentioned, as also that there are persons in whom such an erythema breaks out during many successive years in the course of the same month.

This account of the disease as given by Hebra accurately describes, as far as it goes, the very great majority of cases of erythema multiforme. But, curiously enough, Hebra did not mention the now well-known fact that these eruptions are sometimes associated with joint pains and even, though exceptionally, with effusion in and around the joints. And to complete the description a few other features must be recalled. It has been noticed that in a small proportion of cases the mucous membranes of the mouth are affected, and, still less often, the conjunctivæ. Bronchial catarrh and diarrhœa have also been observed, as also abdominal colic, symptoms which are thought to indicate that the intestinal canal may be the seat of lesions similar to those seen upon the skin. That this eruption is of somewhat frequent occurrence may be gathered from some figures which I have taken from the out-patient department at St. Bartholomew's Hospital. In 1910 there were twenty-one cases among 3,700 patients with skin disease; in 1911, seventeen cases among 4,000—i.e., roughly 0·5 per cent.

The features of these cases are usually so striking and so characteristic that a diagnosis is easily made.

Looked at from this point of view the affection would not appear to be a very important one, and although, like many minor skin diseases, it presents features of interest in regard to its nature and causation, it might not be considered a sufficiently important subject for an evening's discussion. But the question has arisen whether this disease is not sometimes accompanied by more serious symptoms and consequences, and involved in this question are several others of interest. Some of these I now propose to consider, and I shall take them in the following order:—

(1) The question of the relationship of erythema multiforme with erythema nodosum.

(2) The question of the relationship of erythema multiforme with rheumatic fever.

(3) The question of the relationship of erythema multiforme with lupus erythematosus.

(4) The question of the relationship of erythema multiforme with (*a*) some other skin eruptions, including pemphigus, dermatitis herpetiformis, exudative erythema associated with administration of drugs, or serums, or with microbic infections; (*b*) purpura rheumatica and Henoch's purpura.

(5) The question of the occurrence of visceral complications in erythema multiforme.

(6) The ætiology and pathology of erythema multiforme.

THE RELATIONSHIP OF ERYTHEMA MULTIFORME AND ERYTHEMA NODOSUM.

I have already stated that many writers have incorrectly assumed that Hebra made of erythema multiforme and erythema nodosum one disease because he included them under the one title—polymorphous erythema. When Hebra published his account of erythema multiforme, erythema nodosum had been already recognized as a definite disease for more than half a century, and Hebra distinctly says that erythema nodosum differs from erythema multiforme in its form, seat and course, and also in the symptoms by which it is accompanied, and that erythema nodosum, or dermatitis contusiformis, must be described as an independent malady. Sel and Talaman and Düring, of Constantinople, have also insisted upon the differentiation of these two affections as distinct diseases. On the other hand, among those writers who have regarded erythema multiforme and erythema nodosum as varieties of the same disease are Lewin, a pupil of Hebra, who published a monograph in 1878, Besnier and Doyen, who edited Hebra's "Diseases of the Skin" in French, Molènes-Mahon, who wrote a thesis in 1884, and Stephen Mackenzie at the Seventh International Congress of Dermatology in 1896.

Examples of erythema nodosum and erythema multiforme occurring simultaneously in the same patient have been cited as indicating the identity of these affections. But if one reads carefully the records of these cases, one finds that they are really examples of widespread erythema nodosum. I have myself notes of three cases of erythema nodosum, in which the lesions occurred not only upon the shins, but also upon the face and upon the backs of the hands and forearms. But the lesions in these cases, although they occupied the common position of those of erythema multiforme, were more nodular and more deeply seated and they were tender on pressure. Apart from the nodular character of the lesions of erythema nodosum, their oval shape, their extreme tenderness and their situation, particularly on the fronts of the shins, we have also the fact that erythema nodosum is usually not recurrent as is erythema multiforme. Erythema nodosum is much more often accompanied by marked febrile symptoms than is erythema multiforme.

It seems, at any rate at present, better to regard these two affections as distinct diseases, since their confusion tends only to make the elucidation of their cause more difficult. The evidence which is accumulating in regard to the not infrequent association of erythema nodosum with measles, with tuberculosis, or with meningitis—an association which does not belong to erythema multiforme—is an instance of the need for the separate study of the ætiology of these two eruptions.

ON THE QUESTION OF THE RHEUMATIC NATURE OF ERYTHEMA MULTIFORME.

The possible relationship between exudative erythema and rheumatism has been discussed for more than a century. Lorry, in 1777, described several eruptions which occurred in association with articular pains, and affirmed that they were rheumatic symptoms just as were the joint affections. Schönlein, in 1829, gave the name "peliosis rheumatica" to certain erythemas with purpura. From 1835 onwards many authorities, including Rayer, Begbie and Todd, Coulaud, Garrod and Stephen Mackenzie expressed the opinion that erythema multiforme is a cutaneous manifestation of rheumatism. Many of these writers, including Mackenzie, who was one of the most ardent exponents of this theory, have included not only erythema nodosum and erythema multiforme, but also all kinds of erythemas in this category when drawing their conclusions, and the real proportion of cases of erythema multiforme of Hebra associated with supposed rheumatic symptoms has not always been apparent. At the present day the tendency is to regard the joint pains and swellings which are present in cases of erythema multiforme as further evidence of some general toxæmia or infection quite distinct from rheumatic fever. A quotation from a short paper by Dr. Samuel West well expresses the present view from the physician's side. "If," he says, "the diagnosis of rheumatic fever be more definite and more carefully and correctly made, as it is now, . . . the frequency of skin eruptions is reduced very low, almost, except sudamina, to a vanishing point." And most dermatologists will agree, I think, that cardiac lesions are rarely, if ever, present in cases of erythema multiforme. The practical outcome of this view is, that in cases of erythema multiforme one does not give a bad prognosis in respect to the possibility of the subsequent development of rheumatic fever.

QUESTION OF THE RELATIONSHIP BETWEEN ERYTHEMA MULTIFORME AND LUPUS ERYTHEMATOSUS.

The suggestion has been made by several observers that there is a relationship between lupus erythematosus and erythema multiforme. Liveing, in his book on diseases of the skin, says that "it is not uncommon to find a history of polymorphic erythema associated with erythematous lupus, and very common to meet with patches of chilblain-like erythema on the hands of those who are suffering from erythematous lupus of the face." He also says that "patches of erythema are sometimes mixed with lupus erythematosus, and show their nature by disappearing rapidly, leaving only the lupus patches."

Galloway and MacLeod, in the *British Journal of Dermatology* (1908, vol. xx, p. 65) uphold the view that "certain cases of lupus erythematosus and certain types of erythema multiforme are so closely related that they may be regarded as the ends of a chain, in which all transitional stages may be encountered." Perhaps in the present state of our knowledge, and while we are ignorant of the true cause of either of these affections, this must be a matter of opinion. My own view is, that they are distinct affections, probably due to distinct causes, and that although cases of acute lupus erythematosus may simulate erythema multiforme, yet there are always certain features which distinguish them. For example, lupus erythematosus is very rare in young subjects, erythema multiforme is common. Although there may be fugitive erythematous patches in cases of lupus erythematosus, these patches are never sharply rounded like those of erythema multiforme, nor do they present the dull purple centre with red margin of the latter eruption, and there are always present some lesions with the characteristic stippled surface of lupus erythematosus, and others which leave scars. The lesions of erythema multiforme, even the bullous lesions, never leave scars. Again, in erythema multiforme, the eruption avoids the scalp and the central parts of the face, while in lupus erythematosus these are favourite situations. In fact, although both diseases are alike in that they may be regarded as toxic erythemas they are sufficiently distinct to point to their being due to distinct toxins. A case which appears to support this view was shown at a meeting of this Section by Dr. Sequeira on April 21, 1910. In this case a patient with lupus erythematosus developed erythema multiforme of the iris type, but there was no transition from one type of lesion to the other, and the two affections appeared as distinct as are psoriasis and a syphilitic eruption when seen simultaneously in one patient.

I hope that some members present will have more to say upon this interesting point regarding the possible relationship between these two affections.

ON THE RELATIONSHIP OF ERYTHEMA MULTIFORME TO SOME OTHER SKIN DISEASES.

In the years following Hebra's description of erythema multiforme there was a tendency among writers to extend the scope of this affection, more especially to include cases with eruptions of a more severe type, and even fatal cases. This was particularly the case with the French school. Molènes-Mahon, in a monograph inspired by Besnier, enlarged Hebra's conception to include many vesico-bullous eruptions of long duration and wide distribution which we now call dermatitis herpetiformis. Brocq, who for many years was a strong advocate of this view, has lately written an article in the *Annales de Dermatologie*[1] in which he says that he was in error, and he now admits that Hebra's disease has nothing in common with the group of dermatitis herpetiformis. Others have included in one common group of polymorphic erythemata, erythemata associated with serum injections, with cow-pox vaccination, with typhoid fever, pneumonia, gonorrhœa, syphilis, diphtheria, and with septic infections, regarding as erythema multiforme all these eruptions which are characterized by exudation as well as erythema. But many, indeed most, of these erythemas which have been described as erythema multiforme have not the peculiar distribution of Hebra's eruption nor the round disk-like patches with purplish centre and with tendency to spread at their periphery, and it is a question whether we ought to regard them as imitations of the true or "idiopathic" form of erythema multiforme rather than as the real thing. My own view is that we have no more reason for calling these rashes erythema multiforme than we have for giving the names scarlet fever or measles to the scarlatiniform or morbilliform rashes which may occur under similar conditions.

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[1] *Ann. de Derm. et de Syph.*, Par., 1912, 5 sér., iii, pp. 1-31.

On the other hand, I do think we are justified in extending our conception of Hebra's disease in another direction, and that we ought to include with it those eruptions known as Schönlein's disease (purpura rheumatica or peliosis rheumatica) and Henoch's purpura. In each of these affections we have an eruption of erythema exudativum in the form of disks or patches on the sides of the face, on the forearms and hands, and on the legs and thighs. The eruptions may be accompanied by joint pains, and even by serous effusions in and around the joints. Each may have abdominal pains as a symptom, and, except in the milder forms of erythema multiforme, a transitory albuminuria is of common occurrence. The eruptions have a tendency to come out in crops, each outburst being accompanied by a slight rise of temperature. They are prone to recur at intervals of months or years. In fact, the difference between these three affections is only one of degree. The symptoms vary according as there is more or less escape of blood into the tissue involved. As I have already stated, there is some evidence that in erythema multiforme lesions may occur in the alimentary canal similar to those we see on the skin. In purpura rheumatica and in Henoch's purpura these lesions are made more evident by the fact that hæmorrhage takes place into them. In the autumn of 1910 there was what might be regarded as an epidemic of erythema multiforme, and during the same period there occurred also an unusual number of cases of purpura rheumatica and of Henoch's purpura, and this fact seemed to me to lend support to the view that these three complaints are merely phases of one disease.

THE QUESTION OF THE VISCERAL MANIFESTATIONS OF ERYTHEMA MULTIFORME.

In recent years attention has been called by Sir William Osler and by Dr. Galloway to the occurrence of visceral manifestations in patients who present erythematous eruptions, and to the importance of this association. The observation is not altogether new, for so long ago as 1878 Lewin insisted upon the frequency and importance of visceral complications in erythema exudativum multiforme, and Molènes-Mahon, in his thesis already referred to, said that although the cutaneous manifestations had been well described, the occurrence of general disturbances of health and of serious visceral complications had not been sufficiently recognized. The visceral complications included angina, broncho-pneumonia, endocarditis, pericarditis, and nephritis. But as we have already seen, authors had begun to include under the term "erythema multiforme" all kinds of exudative erythemas and vesicular and bullous eruptions whose characters were not strictly those of Hebra's disease, and when one examines the records of the cases reported in which there were fatal complications, it is clear that not many, if any, of them ought to be classed as erythema multiforme of Hebra. This, too, applies to the observations of Osler and of Galloway. Osler includes exudative erythemata without the typical characters of Hebra's erythema, purpuras, urticarias, angio-neurotic œdema, and one case of, possibly, acute lupus erythematosus. In one of Galloway's cases of erythema exudativum associated with cirrhosis of the liver there were erythematous disks and segments of circles occurring most profusely on the trunk, and in another case, also of cirrhosis of the liver, the eruption was regarded as probably acute lupus erythematosus. I do not by any means wish to deny the accuracy nor the great interest and importance of these observations relating to the association of erythemas and visceral troubles, but merely to point out that they are by no means all of them examples of erythema multiforme. They may, I think, be divided into several distinct groups, according to the nature of the affection upon which the rash and the associated visceral troubles depend. For example, with angio-neurotic œdema there occur gastric crises, asthmatic attacks, and œdema of the glottis; with acute lupus erythematosus there may be pneumonia and nephritis; acute generalized erythemas and purpuras of septic origin may be associated with high fever, broncho-pneumonia, nephritis, and ulcerative endocarditis; with cirrhosis of the liver go urticarias and purpuric rashes; and a form of mild purpuric eruption of the legs is sometimes associated with œdema of those parts and with passing albuminuria.

To return to the erythema multiforme group, I do not suggest that these are not associated with visceral troubles, but merely that they, too, have their own particular type of visceral manifestation. Some of these I have already mentioned. They are mainly abdominal symptoms and albuminuria. Endocarditis or lung troubles do not occur, or are, at any rate, very rare.

In mild forms of erythema multiforme abdominal pains are not uncommon, and there may be enlargement of the spleen. Albuminuria may occur, but it is rare, except in cases of a severe type. In purpura rheumatica abdominal pains are of common occurrence, and there may be vomiting and passage of blood by the bowel, and albumin, or even blood, in the urine. Henoch's purpura differs only in that the abdominal pains are a more marked feature and that swellings occur, which may be mistaken for intussusception, or actual intussusception may be produced.

Sachs, in a paper in the *Archiv für Dermatologie und Syphilis*, 1909 (vol. xcviii, p. 35), records five cases of erythema multiforme with albuminuria. In three cases the albumin disappeared with the patient's recovery, and two cases, in which acute nephritis was followed by erythema multiforme, were fatal. Sachs raises the question whether, in these fatal cases, the nephritis may have been due to the same toxin which later produced the eruption of erythema multiforme. Welander has also published a fatal case of erythema multiforme in which there was nephritis. It therefore appears that, although symptoms pointing to exudation or hæmorrhage into the wall of the bowel are the more common, in rare instances erythema multiforme may be accompanied by fatal nephritis.

ÆTIOLOGY AND PATHOLOGY.

One of the earliest theories of the causation of erythema multiforme was that of Lewin, who believed it to be the result of a reflex vasomotor disturbance from some irritation arising in disease of the urethra or genital organs. Others have suggested that the vasomotor apparatus is acted upon by some toxin circulating in the blood. But the theory of vasomotor disturbance would seem to be entirely upset by the knowledge of the fact that there is evidence of marked inflammatory reaction in the lesions. A microscopical section of the lesion of erythema multiforme shows the blood-vessels in the upper part of the corium distended with red blood cells and surrounded by a cell exudation made up of mononuclear and polynuclear leucocytes, together with epithelioid cells, the result of proliferation of the endothelial or connective tissue cells. There is also some œdema and proliferation of the epidermal cells. These features seem to point to an inflammatory reaction to some irritant brought by the blood-stream. We can conceive that it might be a toxin absorbed from the alimentary canal, or produced at some distant focus of microbic infection, or that such a toxin might even be produced by the local presence of micro-organisms in the capillaries of the skin. But whether we are to regard this eruption as the result of some one particular toxin, or of some specific microbic infection, or whether it is to be looked upon as a symptom of various toxins or infections, is still a matter of opinion.

Streptococci, or other cocci, have been found in the lesions or in the blood or urine in cases of erythema exudativum, notably by Finger, Ziegler, Carruccio, Petrini, and Haushalter. But a careful study of the description of these cases makes one hesitate to regard them as examples of erythema multiforme of Hebra. Indeed, Finger (whose cases are sometimes quoted as examples of erythema multiforme) himself draws a distinction between erythema multiforme associated with coccic infections and "idiopathic erythema multiforme," and says that he has been unable to find micro-organisms in the lesions of the latter. Geber, in a recent communication of the *Dermatologische Zeitschrift*, records a number of cases of "so-called idiopathic erythema," in some of which he found cocci in the lesions or in the blood, and in others demonstrated the presence of a focus of coccic infection. One only of these cases can be diagnosed as a typical erythema multiforme, and in that one the coccus found was not identified.

On the other hand, many observers have had invariably negative results from examination of the blood or lesions for micro-organisms in erythema multiforme. In three cases of unusually severe erythema multiforme under my own care the results of blood cultures were negative, and on searching the hospital records of severe cases of erythema multiforme, purpura rheumatica and Henoch's purpura in the wards of St. Bartholomew's Hospital during the past two years, I cannot find any positive blood culture in a large number of cases examined.

These negative results do not, of course, prove that erythema multiforme is not due to a bacterial infection. It is well known that negative results are often obtained in cases of known microbic infection, and this is much more likely to be the case with a mild type of infection. But it is possible that with improved methods a micro-organism will be found as the cause of erythema multiforme. Geber suggests that only quite recent lesions should be examined; that serial sections should be cut, attention being directed especially to the demonstration of thrombophlebitis, and that micro-organisms are to be sought for in the thrombus; that blood cultures should be made while there is fever; and, finally, that complement-fixation methods should be employed, using staphylococcus, streptococcus and gonococcus as antigens.

One point which seems to favour the microbic infection theory is the occurrence of this disease in epidemics, as has been noted by several observers.

I ought, perhaps, to mention a recent view which seeks to draw an analogy between "serum disease" and erythema multiforme, and which regards the recurrence of the rash and joint pains as phenomena of anaphylaxis or hypersensitiveness to some foreign proteid or toxin absorbed from the alimentary canal. As to the nature and origin of such toxin, if it exists, we have as yet no clue.

In conclusion, erythema exudativum multiforme of Hebra is an affection with very characteristic and constant features. It is, in the majority of cases, not a serious disease, although serious and even fatal cases may occur associated with nephritis. It has no relation to rheumatic fever, and is probably distinct from erythema nodosum. It is distinct also from lupus erythematosus. It may be imitated by erythematous eruptions associated with various known toxic and infectious conditions. Erythema multiforme, so-called purpura rheumatica and Henoch's purpura, are probably modifications of one and the same malady. The nature of the disease, whether a toxæmia or due to a microbic infection, is not yet known.

DISCUSSION.

The PRESIDENT said he was sure all present would wish to thank Dr. Adamson for having opened the debate in so masterly and scholarly a manner.

Dr. PRINGLE desired to join in the President's congratulations to Dr. Adamson on his paper. He agreed with the whole of Dr. Adamson's conclusions, but, with regard to his view that exudative erythemas occurring in association with drugs, poisonings, &c., were not true erythema multiforme, he asked how Dr. Adamson would classify such a case as that of which he exhibited a couple of coloured drawings. That case, according to all accepted nomenclature, was a characteristic severe example of bullous erythema multiforme. The patient was a student at Middlesex Hospital, aged 24, and when seen in 1899 he had an enormously severe attack of bullous erythema, typical in distribution—i.e. on hands, feet, &c.—and with great abundance of lesions in the mucous membrane of the mouth and throat. The attack lasted two months. The patient recovered and had not had an attack since. The immediate cause was undoubtedly drain-poisoning and was an incident in his midwifery work outside the hospital. He believed that in Dr. Adamson's own mind there was a little doubt about the differentiation of what he described as microbic diseases from those which he classified as toxæmic. The reasons for and evidences of such a distinction had not been made so clear as he (Dr. Pringle) would have liked.

Dr. WHITFIELD said he felt himself in almost entire agreement with Dr. Adamson, except possibly—and on that he had an open mind—as to whether the purpura should be included with erythema exudativum. One point which he believed Dr. Adamson did not mention was in the separation between erythema nodosum and erythema multiforme. Dr. Adamson said that erythema nodosum did not, as a rule, relapse; he (Dr. Whitfield) would go further, and say it was very rare to find a second attack occurred at any time in the patient's life. Another point was that some years ago the incidence of the two affections in different years was carefully worked out by the Vienna school, and it was found that the seasons in which erythema nodosum was prevalent—and that occurred also in epidemics to some extent—were not the seasons or years in which erythema multiforme was marked. To him that seemed a very important point. He felt fairly confident that erythema nodosum had some dim relationship to rheumatism, though he did not regard it as a definitely rheumatic affection. Still, it occurred markedly in families where there were other rheumatic manifestations, such as acute rheumatism and chorea. The constitutional symptoms of erythema multiforme were chiefly depression, and the patients generally had eye or mouth symptoms; but they did not show the marked and intense anæmia which seemed so characteristic of erythema nodosum, the subjects of which always had an intense pallor, and their hæmoglobin index fell as low as 50 or 60 per cent., even with a slight attack of erythema nodosum. He believed erythema multiforme would turn out to be a toxin eruption of a single general bacterial infection—i.e., that the lesions would not be found to contain the organism. They were familiar with all kinds of rashes which were now associated with streptococcal and staphylococcal infections, also tubercular and other, but they did not see erythema multiforme with these. He did not say one never saw erythema multiforme with known bacterial infections, but it was extraordinarily rare to do so. One saw all kinds of fleeting erythema, but it was rare to see a typical case of erythema multiforme with any known bacterial infection.

Dr. GALLOWAY said that dermatologists would probably look back on the history of this disease as developing in four stages: First the period marked by the work of Willan. Robert Willan's remarkable writings were those of a pioneer in the subject, and marked the path through the wilderness of the crude conceptions then existing of the appearance and causation of this malady. Then came the classical work of Hebra, who, following Willan's pathway, indicated and defined the boundaries of the subject by landmarks still easily recognizable. The third stage many of us associated with the painstaking and accurate observations of Dr. Colcott Fox. We were now in the fourth stage, when we hoped that the recent additions to our knowledge of pathology might result in a still more accurate and fruitful explanation of the subject which Dr. Adamson had brought before the Section. He willingly agreed with Dr. Adamson in most of his observations, but in reference to the ætiology of this group of diseases he thought that we should widen rather than narrow our conception as to the possible underlying causes, even while adhering to the strict definition given to erythema multiforme by Dr. Adamson. To attribute erythema multiforme to a specific microbial infection did not seem to him to be an adequate explanation. He thought that we must all recognize the disease as being due to a circulating poison in the blood and tissues which might have different origins, more probably from some distant source than by the actual development or growth of the poison, bacterial or other, at the point of lesion. He could not help thinking that the actual lesions of the disease, either cutaneous or visceral, were probably to be explained by local failures or diminution in resistance to the noxious influences of a generalized toxæmia. Our knowledge of the phenomena occurring during the condition of anaphylaxis resulting from certain forms of poisoning might well be applied to explain the actual local phenomena occurring in erythema multiforme. On this hypothesis, however, a general toxæmia would be the most likely foundation for the anaphylactic phenomena of the local lesion. From the observations of others and also of what had occurred in his own clinical experience, he felt that such widely different types of toxæmia as those producing cirrhosis of the liver on the one hand and "catarrhal" inflammation of the bowel on the other might all be followed by the local inflammatory and even destructive lesions of erythema multiforme. There were many other points in Dr. Adamson's paper which opened the gates for theoretical discussion, but in the meantime he thought that closer clinical observation and chemical and bacteriological investigation in the wards were urgently stimulated by this evening's discussion. He would like to mention one point in connexion with Dr. Adamson's reference to his old friend, Sir Stephen Mackenzie. Sir Stephen was well known to be a vigorous supporter of the rheumatic origin of certain lesions of the skin, but he would have been unwilling to state that ordinary erythema multiforme was always a rheumatic manifestation; but he did hold very strongly that true erythema nodosum was a manifestation of rheumatism, for he used to say that he regarded erythema nodosum to be as distinctly a mark of rheumatic disease—meaning by that simple acute rheumatism—as was pericarditis or endocarditis. The present tendency of opinion was to regard acute rheumatism as a definite or even specific bacterial infection; therefore he thought it might be a legitimate inference to go to the length of including even erythema nodosum itself in a class with other anaphylactic skin lesions, resulting from the reaction of the organism to a specific rheumatic infection. He would like, with others, to offer his thanks to Dr. Adamson for the care and trouble he had taken in preparing his opening paper.

Dr. MACLEOD said that he agreed with Dr. Adamson in the majority of his conclusions. He believed that the ordinary toxic rashes, such as drug rashes due to sera, &c., were different from the classic erythema multiforme exudativum of Hebra, though he had met with borderline cases in which a certain diagnosis between the two was very difficult. Just as in the case of the ordinary toxic erythemata, similar eruptions resulted from diverse causes, so it seemed to him this was also the case in erythema multiforme. He considered that the peculiar types of reaction associated with it were capable of being produced by a variety of causes, probably toxic in origin. He referred to one case of erythema multiforme in which recurrences seemed to take place owing to the drinking of beer. The patient was a male attendant at a bar and an out-patient at the Charing Cross Hospital. His first attack, which was extremely severe, involving not only the skin of the arms, face and neck, but also the mucous membrane of the mouth, came on as the direct result of a bout of beer drinking, and the subsequent attacks seemed to take place whenever he indulged too freely. The actual toxin was probably an autogenous toxin eliminated as the result of beer-poisoning. A case of this kind did not suggest a microbic origin for the disease. With regard to the relationship of lupus erythematosus to the disease under discussion, he considered that there was a closer relationship between the two diseases. He referred to one case in a young woman who died of nephritis in which acute lupus erythematosus was present on the face, neck and arms, and when it appeared it was diagnosed as erythema multiforme; and to another case, also in a woman with nephritis, in which similar erythematous lesions, which did not scar, persisted until she died.

Dr. WILFRID FOX said the only point in the paper about which he disagreed was concerning lupus erythematosus. He had a case of a hospital nurse who for two years had typical erythema multiforme and the third year had lupus erythematosus, though no scarring was left by the lesions of the first two years. Yet there was scarring after the third year. He agreed with Dr. Galloway that there were multiple causes quite apart from the different soil on which the seed might be sown in various persons. It was well known that various persons reacted differently to the same poison. There seemed no reason to seek one particular cause for lupus erythematosus.

Dr. GEORGE PERNET considered that the paper just read brought all the facts together and stated them in a clear and lucid manner. On the main points he agreed with the author—viz., that true erythema multiforme exudativum was distinct from erythema nodosum and from lupus erythematosus. The point which Dr. Adamson had brought out in regard to the possible relationship of erythema exudativum multiforme of Hebra with Henoch's purpura was very important. In erythema exudativum multiforme one met with hæmorrhage into the lesions. In his own mind he had always kept erythema multiforme limited to the erythema exudativum multiforme of Hebra. But unfortunately the name erythema multiforme had been loosely applied. In the thesis which he published in 1908[1] Dr. Pernet had given the full details of a case which Franz Koch first considered was acute lupus erythematosus, but, influenced by the views of some Berlin dermatologists, veered round to the diagnosis of erythema exudativum multiforme. Later, however, Koch went back to his original view, and anyone who read the account would agree that the case was one of acute lupus erythematosus. In that same thesis Dr. Pernet mentioned a colour drawing of Hebra's, in an atlas of that authority's, of a patient with erythema exudativum multiforme, which had quite a different facial aspect from that of the case which Franz Koch eventually diagnosed as a lupus erythematosus. But Hebra[1] did not give a very clear and detailed account of erythema exudativum multiforme. A curious feature pointing to the endemic or epidemic condition was given in a footnote to Hebra's works, in which he referred to that condition being endemic in some parts of European Turkey, citing a book by Rigler.[2] But von During, to whom Dr. Adamson referred, whose exhaustive monograph on erythema exudativum multiforme went into great detail, did not in the diagnosis say anything about lupus erythematosus.[3] Further, von Düring referred to a case of erythema exudativum multiforme by Lewin, of Berlin, but did not accept it as an instance of that disease. This showed the conception of erythema exudativum multiforme of Hebra had been modified in Berlin.[4] The work of Lendon,[5] of Adelaide, on erythema nodosum should be mentioned in connexion with the above discussion.

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[1] Pernet, "Le Lupus erythémateux aigu d'emblée," Paris, 1908, pp. 55 and 101.

Dr. BOLAM (Newcastle) said that one point to which Dr. Adamson referred was to him of great interest—namely, with regard to albuminuria in these cases and the terminal association with nephritis. From what one had seen of these cases, it would suggest that the severity of the eruption bore a relationship to the degree of renal insufficiency, just as the outbreak of drug eruptions was related in degree with the extent of kidney mischief. In nearly every case of severe drug eruption there would be found some renal trouble. With regard to erythema nodosum, he went further than most of the evening's speakers, regarding it as a subdivision of erythema multiforme determined by the rheumatic poison, just as he expected that erythema multiforme would be further split up when the causal organism was found in particular instances. He did not gather from the paper, as Dr. Galloway seemed to, that Dr. Adamson attributed the eruption to a local infection, but rather to one of a general nature.

Dr. HEATH said the frequent recurrence of erythema exudativum was, in his mind, against a microbial origin. He saw no reason against supposing it to be due to a chemical poison, as opposed to a bacterial one. With regard to erythema nodosum he had always seen the typical eruption on the front of the legs, and occasionally on the forearms. Recently he saw a case with

[1] Hebra, *Hautkrankheiten*, B. iii, Th. i, pp. 198 *et seq.*

[2] Rigler, *Die Türkei und deren Bewohner*, 1842, B. ii, p. 44.

[3] Von Düring, *Arch. f. Dermat. u. Syph.*, 1896, xxxv, p. 219 *et seq.*

[4] Pernet, *op. cit.*, p. 103.

[5] Lendon, "Nodal Fever," 1905.

pain and swelling in the joints, associated with an acute erythematous eruption on the face, looking like acute lupus erythematosus. That was new to him, and it suggested that erythema nodosum was not quite the entity it had usually been considered, and that even that affection might be due to more than one agency. Though he had seen dozens of cases of erythema nodosum he had never before seen it associated with acute erythema of the face.

Dr. GRAY said that a year ago he saw a child which had been vaccinated ten days before, and it had the most marked typical erythema multiforme that he had ever seen. It was partly bullous, but mainly of the ordinary discoid type. That seemed to support the toxic rather than the bacterial view, though, of course, it did not definitely prove anything.

The PRESIDENT said the debate had been short but useful, and it had brought out certain points. He had done a good many years' work in out-patient departments, while having his share of private work, and his impression was that erythema multiforme was distinctly rare in private as compared with hospital practice. The explanation he would give of that was, that most of the patients he had seen came after they had been exposed to extreme cold ; in some there was a definite lack of warmth and a comfortable existence. When the disease was seen in private patients it was generally evident that they had been driving in a cold wind, or had otherwise subjected themselves to unusual exposure, but, of course, the generality of people of that class were usually comfortable, hence the difference in the incidence. He could recall a considerable number of cases in policemen who developed the eruption after night-duty in severe weather. He believed it was due to toxæmia generated in the tissues the vitality of which was lowered by the extreme cold. The next point was, that there was a vast difference between the acute cases caused in the way he had mentioned and the chronic cases which relapsed time after time. The latter he considered to be due to a chronic intestinal disturbance of a complex kind which had not yet been explained. He disagreed with one of the speakers, in that he did not think the Hebra's erythema multiforme which he saw in Vienna was bacterial in origin. He believed it to be due to some form of chronic toxæmia, which was not yet understood, and that it was parallel to the forms of erythema due to drug eruptions and other forms of poison which certainly were not bacterial. He agreed with what Dr. Bolam said as to the prognosis in the chronic cases depending very much on the degree of renal sufficiency. One of the worst cases of erythema multiforme which he had ever seen—a quite typical one, which was shown at one of the societies—went on to a condition of desquamative dermatitis, and the patient ultimately died insane. There was no means of ascertaining whether there was any kidney disease during life. There was no albuminuria, and the urine appeared in other respects normal. At the post-mortem examination it was found that there was only one kidney; the other was a large white kidney. The fatality was due to the kidney disease.

Dr. ADAMSON, in reply, said that he did not know what name to give to Dr. Pringle's case, but he thought the distribution and character of the eruption were not typical of erythema multiforme. He agreed with Dr. Galloway that erythemata might result from a large number and variety of toxic and infective conditions, and that such erythemata might be of morbilliform or scarlatiniform type, or roseolous, or erythemata with exudation. But he thought the characters of erythema multiforme and the peculiar distribution were so definite and distinct that *one* could only suppose some one specific toxin or infection. The more we learned about skin diseases the more we found that eruptions having very distinct features were produced by single causes. The eruptions of the specific fevers—measles, scarlet fever, chicken-pox, enteric fever, &c.—were instances. Lately we had learnt how even different varieties of one organism—the ringworm fungus—might each produce their own distinct eruptions. He thought that all diseases such as lichen planus, psoriasis, scleroderma, lupus erythematosus with sharply marked and constant features, must certainly be due to single causes. The case mentioned by Dr. MacLeod, he thought, was unique. It was interesting, but as the man had long been an excessive beer-drinker it seemed possible that the attacks of erythema multiforme were merely coincident. He could offer no explanation why the lesions of erythema multiforme were localized mainly upon the extremities and sides of the face, or, rather, it seemed to him the most likely position, since stagnation of toxins or micro-organisms would occur here, and it was really more difficult to explain why all toxic erythemas did not favour these parts. In answer to Dr. Bolam, the speaker said that when he suggested a microbic infection as the cause of erythema multiforme he meant a general microbic infection—that the lesions were the result of micro-organisms carried to the skin by the blood-stream. He did not think, however, that a specific toxæmia could be excluded. Sir Malcolm Morris had suggested that the frequent history of exposure to cold as an exciting cause seemed to point to a toxæmic origin; but was not this also a common predisposing factor in microbic infections? He did not wish to maintain that all erythemas of which the cause had been demonstrated had proved to be the result of the local presence of micro-organisms. The eruptions of serum disease and erythemata due to drug poisons were instances of true "toxic" erythemata. He was interested to hear that the President regarded this affection as far less frequent in private practice than among hospital patients, for he had been under the impression that its incidence was not influenced by the conditions of living.

Dermatological Section.

December 19, 1912.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Two Cases of Dermatitis Papillaris Capillitii (Kaposi), or Acne Keloid.

By H. G. ADAMSON, M.D.

THIS affection was apparently uncommon in this country, for since Morrant Baker first showed a case in London thirty years ago not more than seven or eight examples had been recorded here or exhibited at the Section or at the old Dermatological Society. The two cases now shown were, as usual, in men, and the eruption was seated transversely across the back of the neck. One patient, aged 30, had had the complaint for five years; the other, aged 55, for twenty-five years. The latter case showed the classical keloid-like growth with tufts of hair at the margin. The former was interesting as an example of the earlier stage before the keloid appearance had developed. In this case there were raised rounded red nodules of the size of a pin's head up to that of a large pea. Many had a central hair, but there was nowhere any appearance of comedo as in ordinary acne.

Modern investigations tended to show that this complaint was probably the result of some local infection through the pilo-sebaceous follicle, but that it was neither acne nor keloid, and the exhibitor thought that the original name given by Kaposi, although cumbersome, was preferable to that of acne keloid. Local friction from collars or collar bands probably formed a factor in ætiology. Histologically the lesions showed dense infiltrations of plasma cells between newly formed connective tissue bundles. Sections were shown.

(The exhibitor hoped to report these two cases more fully, together with an abstract of the literature of this disease, in the *British Journal of Dermatology*.)

DISCUSSION.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) expressed his surprise at Dr. Adamson's statement that so few instances of the condition had been shown in London. Certainly several had been seen at meetings of the British Medical Association. He had seen very good results follow the application of radium.

Dr. WHITFIELD said that in some of these cases comedones were found when the condition was advanced, but probably this was purely accidental. The epithelium collected, and it became a scar comedo, similar to that following a burn. By this time it might even contain the acne bacillus, but it would be quite secondary.

Case of Sporotrichosis of the Disseminated, Ulcerating, Gumma Type, in which there occurred Acute Synovitis.

By H. G. ADAMSON, M.D.

THE case was of interest as being the first example of *disseminated sporotrichosis* which had been reported in this country, and in that the patient was a woman who had never been out of London. The history of the discovery of sporotrichosis and a summary of the recent knowledge of the disease had been recently given by de Beurmann, at the meeting of the British Medical Association this year,[1] and the exhibitor would therefore merely relate the main facts of his case, which was a typical one.[2]

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[1] *Brit. Med. Journ.*, 1912, ii, p. 289.

[2] The literature of sporotrichosis is now very extensive. The most important work on the subject is "Les Sporotrichoses" by de Beurmann and Gougerot—a volume of 850 pages—Librairie Felix Alcan, Paris, 1912, with a complete bibliography (23 pages). For a short summary see article "Sporotrichosis," Allbutt and Rolleston's "System of Medicine," 1911, ix, pp. 525-31. The only cases hitherto reported in this country are: (1) the case of Norman Walker and Ritchie (*Brit. Med. Journ.*, 1911, ii, p. 1), localized type, infection in Cumberland; (2) a case of localized type reported by myself—infection in South America (*Brit. Journ. Derm.*, 1911, xxiii, p. 239); (3) a doubtful case by Ofenheim (*Lancet*, 1911, i, p. 659).

E. W., needlewoman, aged 60, was admitted into St. Bartholomew's Hospital, on August 16, 1912, suffering from ulcers on the arms and legs. The history given by the patient was as follows: In August, 1911, there had appeared upon the outer side of the right thigh a swelling of the size of a pigeon's egg. Two or three weeks later there was another similar swelling on the right arm. The swellings were not red or painful. A similar lump appeared upon the left shoulder. She was then admitted to a general hospital, and whilst there other lumps appeared on the back, these ulcerated and were fomented. She had "vaccines" twice a week for three months. As no improvement took place she left the hospital, and later attended at St. Bartholomew's Hospital, and was there admitted.

On admission to St. Bartholomew's Hospital there were numerous ulcerations upon both arms and both legs, and several upon the buttocks, shoulders and back. There were also about half-a-dozen deeply seated, soft, nodular swellings (gummata) varying in size from that of a small marble to that of a hen's egg (fig. 1). It was obvious that the ulcerations had resulted from the breaking down of the gummatous swellings. These ulcerations were peculiar in that they were not "open," but covered by skin, which was thinned and perforated with numerous holes through which there discharged serum and pus. The legs especially were thickly studded with these cribriform patches, which were deeply pigmented, almost black (fig. 2). The lesions resembled in some degree multiple syphilitic gummata. They also somewhat resembled tuberculous abscesses. But they were not typical of either of these affections and both Wassermann and tuberculin tests gave negative results. The case, indeed, at once suggested the multiple gummatous sporotrichosis described by de Beurmann and by other French observers, and a cultural examination proved this diagnosis to be correct.

The cultures (figs. 3 and 4) were obtained by the method recommended by de Beurmann. About 1 c.c. of pus was withdrawn from an unbroken gumma by means of a small exploring syringe, and squirted on to the surface of a sloped glucose-peptone-agar tube. The tube was kept at room temperature and uncapped. In ten days' time white points or tufts (soon becoming dark brown) appeared here and there in the spread-out pus. From these subcultures were made, and the characteristic dark brown convoluted growths of sporothrix were obtained. A nodule was removed for *microscopical examination*. This showed the mixed tuberculoid, syphiloid and ecthymatoid types of infiltration, as described by de Beurmann. At one part there were epithelioid cells and giant cells, at another collections of plasma cells, and at another groups of polynuclear leucocytes. As was usual, no sporotrichial elements were discovered in smears or in the sections.

The *course of the case* while in hospital had presented some incidents of special interest. During the first few weeks fresh lesions continued to appear, and the temperature rose occasionally to 100° F. When the diagnosis of sporotrichosis was made potassium iodide (the drug which is known to cure sporotrichosis) was given in increasing doses until the patient was taking 3 dr. daily. Rapid improvement took place in the skin condition. The ulcers healed, and the gummata for the most part disappeared. But the patient began to have an irregular rise

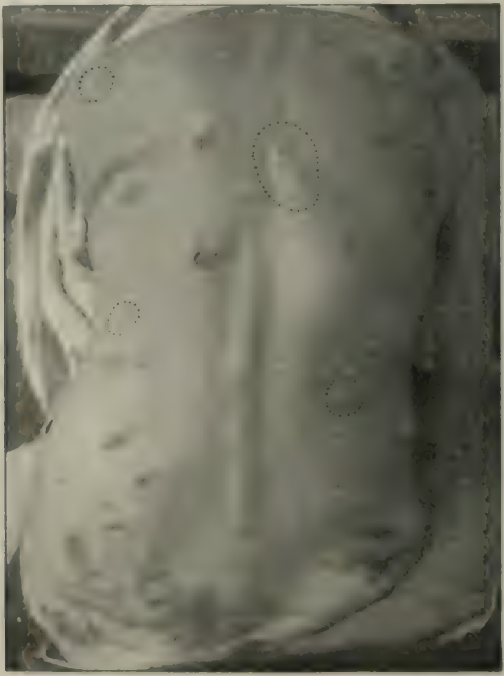

Fig. 1.

Multiple sporotrichosis. Shows the back of the patient with several lesions involving the skin and similar to these seen and described in fig. 2. There are also a few deeply seated gummata (the earlier stage of the lesions). The areas included in the dotted lines were the sites of gummata.

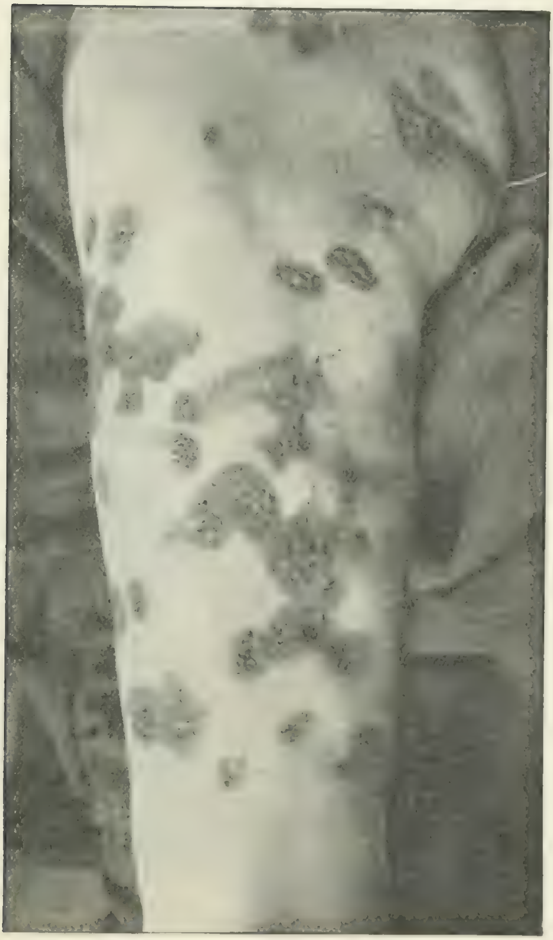

FIG. 2.

Multiple sporotrichal ulcerating gummata. There are deeply pigmented areas, the skin over which is cribriform and covers a pocket containing pus. These lesions evidently result from gummata which have involved the skin and discharged their contents through the numerous small openings. The lesions were only slightly raised above the level of the skin and their margins had little or no appearance or feeling of infiltration.

of temperature to 101° to 103° F. This first took place after opening a very large gummatous lesion on the back, but also soon after the potassium iodide was first administered. Then there developed an attack like "acute rheumatism." The left hand and fingers, the elbows, the right foot and ankle, became very much swollen and very painful on movement. The temperature was from 101° to 103° F. The swellings round the joints were œdematous, but there was obviously also fluid in the joints and tendon-sheaths. It was now decided to discontinue the potassium iodide and to give a sporotrichial vaccine But on discontinuing the medicine the temperature fell to normal and the joint swelling rapidly disappeared, and the vaccine was not given. But several fresh gummata appeared. The potassium iodide was again given, and the fever and the joint swellings returned, to subside at once when the drug was again omitted. This occurred several times—

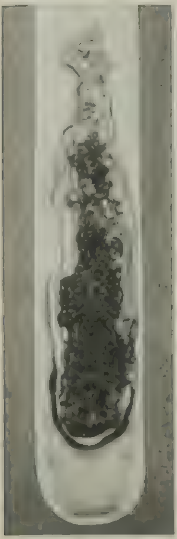

Fig. 3.

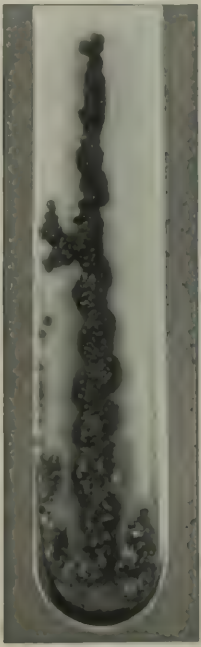

Fig. 4.

Figs. 3 and 4.—Cultures of sporotrichium from the case exhibited, on glucose-peptone-agar, grown at room temperature. The culture has at first a pale grey, downy appearance, but soon becomes darker in colour, pasty and convoluted, as in the photographs.

immediately the potassium iodide was given the fever and synovitis returned. As one member of this Section had suggested, this seemed to be analogous to Herxheimer's reaction in syphilis.

The occurrence of acute synovitis in sporotrichosis was not uncommon, and de Beurmann had written as follows: "Sporotrichial synovitis may simulate acute coccal synovitis, subacute gonococcal synovitis and synovitis of chronic tuberculosis. In the presence of acute, subacute or chronic synovitis one must now systematically discuss a mycosic infection."[1] At the present time the patient was free from synovitis; the ulcers were all healed, but they had left deeply pigmented scars; the nodules had all disappeared. It could not be said that the patient was cured, for the fact that the joint trouble was lighted up on administration of potassium iodide suggested that there were still foci of disease in their neighbourhood.

Dr. GRAHAM LITTLE congratulated Dr. Adamson very heartily on his elucidation and demonstration of the ætiology of this difficult case. The patient was shown to the Section by Dr. Little on October 19, 1911,[2] as a case for diagnosis, with the suggestion that it was either sporotrichosis or actinomycosis, but evidence of either condition was not forthcoming. In the subsequent discussion Dr. Adamson and Sir Malcolm Morris both inclined to the diagnosis of tuberculosis. The patient was admitted to the Inoculation Department under Captain Douglas on November 8, and Dr. Little wished to express his thanks to Captain Douglas for permission to quote the following notes taken in that Department: The diagnosis of sporotrichosis, which Dr. Little had favoured from the clinical appearances, was kept in mind; pus from fresh lesions, obtained by aseptic puncture of the deeper nodules and by scraping the wall of abscesses, was planted on agar, blood agar, acid glycerine agar, &c.; and attempts to grow were made in hot and cold incubators, aerobically and anaerobically. The tubes were kept for weeks in the cold incubator. Blood cultures were made on November 27, when temperature was 101° F.; on December 5, when temperature was 103° F.; in both instances with negative results. In all the attempts at culture no organism was grown except staphylococcus and a Gram positive bacillus. From both of these organisms a vaccine was made and tried, with apparently good result on the temperature—e.g.: On December 6, the temperature being 103° F., vaccine was given and temperature fell by a degree each day until it was normal on December 9. On December 16, temperature again reached 103° F.; autogenous vaccine given on December 16, temperature fell progressively, reaching normal on December 19. Autogenous vaccine given on December 21, December 27, and January 1; throughout this period temperature was below 100° F. These vaccines were continued throughout January, and the temperature only twice reached 101° F., for the whole of the rest of the time being about 99° F. During February the vaccine treatment was continued and the temperature kept below 99° F. On February 20 trypsin was injected, and vaccines were continued throughout March, the temperature several times reaching 100° F., but usually being about 99° F. She was then transferred to the Infirmary.

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[1] "Les Sporotrichoses," p. 367.

[2] *Proceedings*, 1912, v, p. 11.

An Angiokeratomatous Family.

By J. J. PRINGLE, M.B.

THE affected members were: the father aged 46, a boy aged 17, a boy aged 13, and a boy aged 8. There were no other male members of the family. All four showed classical angiokeratomata on the hands, and to a less degree on the feet. They all suffered from severe perniones, leaving deep scars, and had extremely pronounced "chilblain circulation." The father and eldest boy—both of whom described themselves as suffering from phenomena almost tantamount to Raynaud's disease—presented marked "sclerodactylia" with necrosis of the finger-tips.

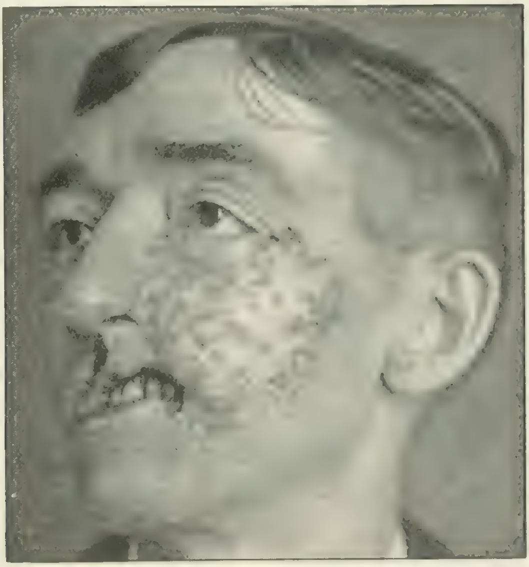
The father and two elder boys showed unmistakeable evidences of tuberculous disease of the lungs or joints. The youngest boy as well as the mother and the one existent sister presented no signs of tuberculosis.

The exhibitor had not had the opportunity of thoroughly studying the cases, but he called attention to the *familial occurrence* of angiokeratoma in them, which was a new point to him. He also referred to the association of angiokeratoma with tuberculosis, which had often been noted, although wide divergences of opinion existed as to the relationship between the two conditions and its interpretation.

An Unusual Form of Rodent Ulcer.

By J. H. SEQUEIRA, M.D.

J. F., AGED 44, was first seen at the London Hospital on November 7, 1912. He was suffering from an extensive crusted eruption on the left cheek. The appearances strongly suggested a tertiary syphilide. The patient stated that seven years ago a small "pimple" appeared on the left side of his nose. Its exact site was the sulcus between the ala nasi and the cheek. The growth gradually increased until it formed a large "red patch." After some time this healed up, but at the upper margin a fresh outbreak occurred under the left lower eyelid, and this gradually spread during five years until the whole of the left cheek was affected. About twelve months ago the lesions began to discharge some "thin yellowish matter" and this came from numerous "small sores" which had never healed properly. Six months ago a deep ulcer developed at the left angle of the mouth, and a little earlier the outer canthus of the left eye was attacked. The sole application had been a widely advertised ointment. With the numerous small rounded crusts the appearance strongly suggested multiple gummata, and this was supported by the history that the patient had had a penile sore fifteen years ago and that he had had treatment by pills for fifteen months at a general hospital. He, however, denied ever having a rash or sore throat and his hair had not fallen out.



Unusual form of rodent ulcer.

Mercury and iodide of potassium were first prescribed, and the crusts were removed by fomentations and the white precipitate ointment. As soon as the crusts were removed it was obvious that the case was one of rodent ulcer, for the margin of the lesion near the outer canthus of the left eye and of the one at the angle of the mouth had the characteristic beaded edge. The appearance is well shown in the accompanying photograph. The major portion of the left cheek was covered by superficial scar tissue, with a few telangiectases. Scattered over the scar were numerous raised, rounded, pinkish growths, varying from a millet seed to a split pea in size. Some of them showed a tendency to central ulceration. At the outer canthus there was more extensive growth in the form of a raised, irregular, somewhat festooned beaded edge surrounding an ulcer which exuded a thin, sanious discharge. At the angle of the mouth there had been more destruction, and there was considerable deformity. The ulceration extended to the muco-cutaneous junction, but did not involve the buccal mucosa. Here there was a wide, raised margin, and considerable gummy discharge.

The Wassermann reaction was negative. A portion of the growth was excised and examined microscopically. It showed that the neoplasm was a basal-celled carcinoma. The rodent growth was evidently of the superficial cicatrizing type, and the rounded nodules left in the scar tissue were portions which had not cicatrized, or, possibly, recurrences.

In the exhibitor's experience, rodent ulcer of the cicatrizing type usually occurs on the forehead and temples, above the zygoma, and he had attributed this superficial localization to the toughness of the fascia which has its lower attachment along the zygoma, the basal-celled carcinomata often being brought to a halt in their development by the presence of a tough fibrous membrane. The resemblance to a gummatous ulceration was striking, and, with the history, the diagnosis of syphilis was natural. The fact that the Wassermann reaction was negative after only eighteen months' treatment by pills is strongly in favour of the patient never having had syphilis. There was no evidence of past syphilitic lesions on the skin or mucous membranes.

DISCUSSION.

Dr. PERNET said that when he was a dresser to the late Marcus Beck, a case was in the wards which was looked upon as syphilitic, but which proved to be a rodent ulcer. It was deeper than in this case, and occupied somewhat the same region; it encroached on the orbit, and eventually became very destructive, going down to the bone. The patient was put upon iodide of potassium, and there was a good deal of improvement, but only up to a certain point. It was then referred to the late Dr. Radcliffe-Crocker, who diagnosed rodent ulcer.

Dr. WILFRID FOX said there was under the care of Sir Malcolm Morris, at the Seamen's Hospital, Greenwich, a sailor suffering from rodent ulcer with a similar condition to that described by Dr. Pernet. It was on the nose, was cured by zinc cataphoresis—this was before the days of radium—and the patient went away to sea. He remained well for eighteen months, then came back with a recurrence, which was cured by X-rays. He again remained well for a year, then returned with the condition breaking down. X-rays were thought to irritate him, and cataphoresis was resumed, but it ended in disaster, because the growth began to penetrate his nasal cavity; the bone was exposed, and it spread to the orbit. As it had then become a surgical case, the surgeon was asked to clear out the orbit. The surgeon diagnosed tertiary syphilis, and said he would cure it by iodide of potassium. Microscopical specimens showed its nature, but the iodide of potassium was given, and it improved very much. Then the improvement ceased, and progress was in the undesirable direction.

Case of Onychogryphosis.

By A. M. H. GRAY, M.D.

THE patient was a lady, aged 62, and was at present under the care of Dr. Batty Shaw in University College Hospital on account of cardiovascular trouble. The nail condition was probably due to the fact that owing to her stoutness she had had difficulty in cutting her toe-nails for five or six years. The case was shown owing to the marked degree of deformity.

Case of Seventh and Eighth Nerve Paralysis after Neo-salvarsan Injection.

By A. M. H. GRAY, M.D.

THE patient was a young man, aged 22, seen on October 17, and he then had a typical secondary syphilitic eruption all over him. He was taken into hospital and given 0·9 grm. of neo-salvarsan. As the symptoms cleared up he did not come for further treatment, although warned to do so. Six weeks after the injection he became deaf on the left side, and noticed that the left side of his face did not move, and he complained of considerable pain behind the left ear. A point of distinct tenderness was detected over the left mastoid. He attended the Medical Out-patient Department and was seen by Dr. T. R. Elliott, who found that he had in addition complete facial paralysis on the left side and complete left deafness. There was also slight left-sided weakness, associated with ataxia and marked coarse nystagmus when looking to the right, and a fine nystagmus when he looked to the left. The sense of taste appeared to be somewhat diminished on the left side. This patient, who had had syphilis, had obviously had insufficient treatment, and had developed, apparently, a localized lesion in his internal auditory meatus, which was most probably syphilitic in nature. It might be a localized syphilitic meningitis in the internal auditory meatus, or thrombosis of the auditory artery. It did not appear to be a neuritis, nor an affection of the nuclei of the seventh and eighth nerves.

DISCUSSION.

Dr. Pringle said his experience of salvarsan in syphilis was small as compared with that of some other Fellows. The only accident he had observed after its use was unilateral tinnitus and deafness: this he had noticed in two cases, both in the early secondary stage, and occurring soon after injection. He would hazard no opinion as to whether the symptoms were the result of the disease or of the remedy; but before the introduction of the latter he had never personally encountered a similar complication.

Dr. Pernet said he had a case some time ago, sent to him from the Continent, in which the patient had had four intravenous injections of salvarsan (doses unknown). The last injection of the series had been given six weeks before Dr. Pernet saw him (Case Book I, fol. 443), when the patient complained of deafness. A few days later facial paralysis had developed. He then presented much the same symptoms as the patient before them.

Dr. Wilfrid Fox said he had had two cases in which herpes developed after the use of salvarsan; in one after intravenous injection, and in the other after intramuscular. Joha was the preparation used for the latter.

Mr. McDonagh said he had seen two or three syphilitic cases which had never had treatment develop into the condition which the present patient had, consequently he did not attribute this man's lesion to the salvarsan, but to the disease. A guarded prognosis should always be given, because though some of these cases cleared up, others remained *in statu quo* however vigorous treatment might be, but nevertheless active treatment should invariably be prescribed. He had shown a case of eighth nerve paralysis in a patient who had not had "606," but only mercury, and she was still as bad as when he first saw her. He had at present under care a syphilitic male patient with paralysis of seventh and eighth nerves on one side, which in spite of six injections of "606" and mercury left him no better than when he first sought treatment. In this case the paralysis occurred three months after the primary sore was first noticed. He had seen cases of syphilitic neuritis of the sciatic nerve and of the brachial plexus which salvarsan did not improve immediately, not until three to six months had elapsed since beginning treatment. Therefore in all cases of syphilitic inflammation of nerve tissue, all hope of improvement should not be given up until the patient had been well treated and kept under observation over an extended period.

Dr. WHITFIELD said he agreed with Mr. McDonagh as to the influence of salvarsan. He had only had one case of severe early symptoms in syphilis. It was the case of a woman who was under mercury, and had had no salvarsan at all. In a fortnight she went stone-deaf. Mr. Cheatle saw her and said it was the acute auditory paralysis of secondary syphilis. At the time of the onset of the deafness all the other symptoms of syphilis (rash, sore throat, &c.) had disappeared. Mr. Cheatle advised treatment with salvarsan, and this was administered, but no good effects were observable six weeks later. Since then Dr. Whitfield had not seen the patient, but he had heard from another patient that the deafness had ultimately cleared up. He regarded the recovery as being due to natural cure and not to the efficacy of the treatment.

Dr. MACCORMAC said he had seen one case of seventh nerve paralysis, in a patient who had had salvarsan, and it cleared up after a second injection of that substance. He considered the paralysis was due to the syphilis, not to the salvarsan.

Dr. F. PARKES WEBER referred to a man, aged 34, whom he had at present under care in the hospital, who had tertiary syphilitic trouble and gave a positive Wassermann's reaction. He was given salvarsan—namely, an intravenous injection of 0·6 grm. on October 26, and another one of the same amount on November 5. On November 7 he complained of weakness and giddiness and disliked the noises in the street. In the night he had headache and could not sleep, but there was no vomiting. Next day he remained in bed (November 8), and in the afternoon did not answer questions when spoken to. On November 9 he was admitted under Dr. Weber's care, at the German Hospital, in a state of stupor. In the hospital there was left-sided facial paresis, but of the cerebral (lower face) type, not (as in Dr. Gray's case) of the facial nerve (whole face) type. The patient improved very much, but there were now peculiar emotional symptoms (exaggerated tendency to smile, &c.), suggesting a "pseudo-bulbar" (bilateral cerebral) origin. For a short time treatment by mercurial inunction and iodide of potassium had been employed.

Dr. WHITFIELD asked what grounds Mr. McDonagh had for saying the clearing up of the case he mentioned after three months was due to salvarsan. The usual experience was that if symptoms did not show signs of clearing up after salvarsan in a fortnight, they did not do so at all.

Dr. GRAY replied that the Wassermann reaction in this patient was positive. Since a second injection four days ago he said he could hear a little. In the pre-salvarsan days it was found to be difficult to get such cases to clear up with mercury. He thought that there might be different types of eighth nerve lesion, for many of the recorded cases had been bilateral, and one could not understand that on a localized inflammatory hypothesis. Some recorded cases of the kind had cleared up without treatment. Other cases got worse when the arsenic was given, and improved when it was left off.

Case of Raynaud's Disease.

By H. C. SEMON, M.D.

THE patient, a woman, aged 56, came with a seven years' history of recurrent local asphyxia of fingers, toes, and nose. The tips of the fingers of the right hand had become gangrenous since September. Wassermann's reaction was positive, but she had had no miscarriages, and no other symptoms pointing to syphilis.

DISCUSSION.

Dr. GALLOWAY said that he always found difficulty in making the diagnosis of Raynaud's disease in the type of case now shown. It was not sufficient foundation for the diagnosis that the patient suffered from a chronic variety of gangrene of the tips of the fingers and toes. In this patient the disease had commenced a few years ago, when she was already aged 49. When the possibility of disease of the blood-vessels produced by various causes, specially resulting from earlier syphilitic infection, had to be taken into account, the likelihood of the existence of uncomplicated Raynaud's disease diminished. In the case of persons such as the patient before the Section, whose position in life had involved overwork and strain of various kinds, the likelihood of disease of the blood vessels was greater than in those in a more favourable condition of life. Factors in the causation of the disease such as those mentioned must be borne in mind, so that the later in life that pain in the extremities with terminal gangrene occurred, the less was the likelihood of the case being simple Raynaud's disease, and the more likely was it due to disease of the vascular system, and the consequence of an imperfect terminal circulation. It must also be recollected, in cases of terminal gangrene clearly associated with endarteritis and arterial degeneration, that attacks of pain, pallor, and congestion of the extremities succeeded by gangrene usually showed paroxysmal features. It might possibly be argued that the Raynaud phenomena might more easily develop in the case of a patient already affected by disease of the peripheral blood-vessels. Speaking generally, however, it might be said that the later in life the symptoms resembling Raynaud's disease developed, the less likely was the attack to be an uncomplicated case of this condition.

Dr. WHITFIELD pointed out that when Raynaud described his cases he had not the advantage of the Wassermann reaction. In this woman there was no history of syphilis. Most of the cases of Raynaud's disease which he had seen showed a positive Wassermann, and even young patients with Raynaud's disease might have congenital syphilis. Apart from hæmoglobinuria, the classical symptoms of Raynaud's disease were spasmodic syncope, anæmia followed by cyanosis, with gangrene as a late development. This patient had all these and the disease was symmetrical. He did not see how one could have that repeated spasmodic symmetrical condition from endarteritis unless every artery in the body was involved, and that was not so in this patient. Possibly it might be a system disease, akin to general paralysis of the insane.

Dr. F. PARKES WEBER said it was difficult in such a case to explain how syphilis could be the direct cause of the gangrene. For a long time the two chief predisposing causes of Raynaud's symptoms had been supposed to be syphilis and malaria, of which the first had the better claim of the two. Dr. Weber himself believed that syphilis was the chief *predisposing* cause of Raynaud's phenomena (at least, of the gangrenous form of Raynaud's phenomena), but syphilis was not sufficient to account for the gangrene in a case like that of Dr. Semon. In other words, syphilitic arteritis could not be so distributed as suddenly to give rise to an obstructive (not merely angiospastic) gangrene, involving all, or nearly all, the finger-tips of both hands. There was, however, some room for doubt in cases when only one or two finger-tips or toes were affected, especially if, in addition to a history of syphilis, active malaria was present, as in the case of a sailor, aged 41, formerly under Dr. Weber's care with toe gangrene. Malarial parasites were found in that patient's blood, and he gave a history of having had syphilis seven years previously.[1]

Dr. SEQUEIRA said that at a recent meeting he showed a man, aged 60, with necrosis of the terminal phalanges on one side of the hand, and it proceeded to one or two toes. The Wassermann reaction was negative, and there was no history of syphilis. The man believed himself to be gouty, and described the picking out of chalk-stones, but he had no tophi. Though there was a history, seven years before, of a similar attack of necrosis, he hesitated to class it as Raynaud's disease. He had neither malaria nor hæmoglobinuria.

Dr. PERNET said he did not see how this case, with the paroxysmal bilateral symptoms described, could be due to syphilitic endarteritis. Where syphilis was responsible for gangrene, the lesion in his experience was unilateral and terminal (the big toe, for instance).

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[1] F. P. Weber, "Raynaud's Disease in a Malarial Subject," *Trans. Med. Soc. Lond.*, 1909, xxxii, p. 370. Cf. Sir W. Osler, "A Case of Multiple Gangrene in Malarial Fever," *Johns Hopkins Hosp. Bull.*, Balt., 1900, xi, p. 41.

The PRESIDENT remarked that in the pre-Wassermann days a large number of cases had been shown as cases of spasmodic Raynaud's disease in which there was no ascertainable history of syphilis.

Dr. PRINGLE was, of course, aware that a positive Wassermann reaction was commonly present in cases of Raynaud's disease. He was, however, unaware of what its connexion with syphilis was, or was supposed to be; and he could not conceive it to be an essential one. He could not see any incompatibility in the divergent views as to the nature of the vasomotor conditions in Raynaud's disease expressed by various Fellows. Might not spasmodic vascular phenomena be superimposed upon organic syphilitic arterial disease? Might not the latter actually predispose to the former?

Dermatological Section.

January 16, 1913.

Sir Malcolm Morris, K.C.V.O., President of the Section, in the Chair.

Erythema Induratum with Tuberculosis.

By F. Parkes Weber, M.D.

The patient, a young woman, aged 30, single, has a mass of enlarged lymphatic glands on the left side of the neck and typical "erythema induratum" (Bazin's disease) on both legs. In regard to her general condition there is nothing special to note beyond slight anæmia and a tendency to chilblains on the fingers. The heart, lungs, and abdominal organs seem healthy, and the urine is free from albumin and sugar. The erythema induratum, which is most advanced on the left leg, consists of patches of brawny swelling, with a more or less livid appearance, chiefly at the back, over the lower portion of the calf muscles. There has been ulceration, but there is none at present. The history of glandular enlargement on the left side of the neck dates from the age of 10, but the swelling seems soon to have subsided and not to have returned till about the age of 19 or 20. At the age of 22 the glands were removed by operation; but at the age of 26 enlarged glands were again noticed, and an abscess in connexion with them was opened. There was further enlargement one and a quarter years ago, and the patient underwent a long course of tuberculin treatment, under Dr. A. White Robertson, in 1912, from January to the end of September. In November, 1912, however, there was again much swelling. At present the glands on the left side of the neck might suggest the diagnosis of Hodgkin's disease (lymphadenoma), but against this is the long history without enlargement of other superficial lymphatic glands or of the spleen or liver. Moreover, the presence of the erythema induratum is probably a point in favour of the tuberculous origin of the glandular enlargement. In regard to the history of the erythema induratum, it may be stated that it was present, to a slight extent, in both legs at the age of 13. At the age of 16 both legs were worse, and there were broken-down places—deep ulcers, like "holes in her legs." Since then the state of her legs has varied, but on the whole they have improved. The tuberculin treatment in 1912 seemed to make no obvious difference to them.

DISCUSSION.

Dr. PARKES WEBER asked members whether they knew if any of the cases formerly diagnosed as erythema induratum or Bazin's disease had really been local sporotrichosis infections.

Dr. WHITE ROBERTSON said he used for this patient, first tuberculin P.T.O. followed by P.T., then worked to Koch's tuberculin to the full cubic centimetre dose. The swelling of the original enlarged gland had been 3½ in. long, but there had been no suppuration. Under tuberculin it steadily went down to 1¼ in. Most of the present glandular swellings had come up since he last saw her on October 6, and he could not accept them as tuberculous in view of the recent high dosage without reaction.

Dr. WHITFIELD said that in his experience erythema induratum reacted with old tuberculin—i.e., it tumified and, if not already red, became red and swollen, and after a single dose frequently got much better. Like all tubercular lesions, this condition was very variable in its reaction to tuberculin. He had a case in which a nodule was already liquefied, and yet it disappeared when the patient was treated with tuberculin. Previously to using the tuberculin the nodules had always ulcerated.

Dr. PARKES WEBER remarked that the result of the tuberculin treatment in the present case seemed unsatisfactory. Though the glands had subsided for a time they had soon enlarged again with a rush. Possibly some slight septic infection in the fauces had started the flaring-up again of the lymphatic glands in the neck.

Case of Granuloma Inguinale Tropicum.

By C. C. Choyce, F.R.C.S., and H. MacCormac, M.B.

The patient was a native of China, and, as far as can be gathered, the disease began in October, 1911, somewhere in the neighbourhood of the penis. From that position it has gradually spread backwards until the present state of affairs has been attained. In the left groin, extending along Poupart's line, almost to the anterior superior spine, there is a broad line of mixed granulomatous and scar tissue. There is evidence that the disease has been more active here, but has undergone a certain amount of healing. In the peri-anal region there is both granulomatous formation and ulceration, and from these ulcers there is a foul discharge. Around the coccyx the disease assumes a horseshoe shape, being composed of large nodular masses with central ulceration. The peculiar pink colour of these lesions is in strong contrast to the normal pigmented skin. Besides the ulceration and granulomatous changes there is also a considerable degree of hardening or sclerosis. Wassermann's reaction has been tried, with negative results, on three occasions; no micro-organisms, except such as would be found as contaminations, have been isolated by cultural methods. A blood count demonstrated the existence of some anaemia, together with an eosinophilia (5 per cent.). An injection of salvarsan had been tried without benefit. The general health appears to be little affected.

Microscopic sections demonstrate that the condition is neither tuberculosis, syphilis, nor malignant.

DISCUSSION.

Dr. PRINGLE briefly referred to the case seen by him in 1889, which he believed to be the first observed in this country. His notes were incorporated in an article by Dr. Galloway.[1] Up to that time all recorded cases occurred in the West Indies, or in West Indians; but the disease was now known to be of very wide tropical distribution.

Dr. SEQUEIRA reminded the members of a case shown by him in which there were not only lesions of this type in the inguinal region but also a horseshoe-shaped tumour at the angle of the mouth. The case was described, with a coloured illustration, in the *Proceedings*.[2] The infiltration cleared up entirely under the X-rays.

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[1] *Brit. Journ. Derm.*, 1897, ix, p. 133 *et seq.*

[2] *Proceedings*, 1908, i, pp. 57 and 92.

Dr. MACLEOD said that he had exhibited a case of granuloma tropicum at the Dermatological Society of London, in which the disease was chiefly located in the gluteal fold and extended for a short distance into the rectum on the mucous membrane. After trying various forms of treatment a complete cure was obtained by exposure to the X-rays followed by scraping. An attempt to scrape the warty granulations, before employing X-rays, was unsuccessful, owing to their toughness. He considered that in situations where the disease could be satisfactorily exposed the X-rays alone could effect a cure. In this case the only organism which was found was a staphylococcus, and it was conceived possible that it might be the pathogenetic agent. No Leishman-Donovan bodies were found such as had been reported in one or two cases.

Mr. HAYWARD PINCH said that the last case of the kind which he saw in India was in the person of an Englishman, an occurrence which was very uncommon. It took a long time for the patient to get well, though the case was seen early. It was ultimately cured by scraping followed by zinc ionization.

Mr. McDONAGH said that Wise had described spirochætæ as being found in this condition, but was not certain as to whether they were the cause thereof. The absence of response to salvarsan was certainly against a protozoal origin. This supported Flu's observation, who considered that the disease was due to a capsulated intracellular diplobacillus not unlike the bacillus of rhinoscleroma. Flu's work had been confirmed by one or two other observers.

Case of Erythema Iris (? due to Potassium Iodide).

By J. L. BUNCH, M.D.

THE patient was a man, aged 31, the conductor of a tram, who some weeks ago came to hospital with some raised, firm, slightly indurated lesions on both knees and elbows. These lesions were of about the size of a pea, grouped in patches of five or six, pinkish or yellowish in colour, and in a few cases they contained fluid of a purulent character. There was no affection of the buccal mucous membrane, and no spots elsewhere. There was a history of the patient having taken some medicine, but the nature of this is unknown. The character of the lesions and their distribution gave rise to the suspicion that they might have been produced by potassium iodide. Under the administration of arsenic internally the lesions all cleared up. This was then discontinued and 5 gr. of potassium iodide given three times a day. After a few days almost precisely similar pea-sized, partly vesicular or pustular spots again appeared on the knees and elbows, but now accompanied by small white patches on the mucous membrane of the inner sides of the cheeks. Little variation showed itself in the character or distribution of the patches until about a week before the meeting, when raised, circular lesions appeared on the anterior surfaces of the wrists, with a pinkish periphery, separated by a paler zone from a purplish centre, which was in some cases surmounted by a vesicle or pustule. These lesions were very numerous and spread some distance up the forearms. They are present now, and in my opinion they present the typical appearances of an erythema iris eruption. As to how far they have been caused by the potassium iodide is perhaps uncertain. The mere stoppage of the arsenic internally may possibly account for the reappearance of the eruption on the knees and elbows (even if the administration of potassium iodide is a more probable explanation), but this eruption had neither the characters nor the distribution of erythema iris, and it must not be forgotten that the co-existence of an iodide eruption with lesions of typical erythema iris or any other variety of erythema multiforme is not impossible. Cultivations of some of the pustules showed only the presence of *Staphylococcus albus*.

Dr. PRINGLE entirely agreed with the diagnosis of erythema multiforme iris and with the exhibitor's observation that the original distribution of the lesions on the elbows and knees was puzzling. In his experience iodide of potassium did much harm in such cases. He had given it a large trial about twenty years ago, when it was greatly advocated in France for erythema multiforme, but had soon abandoned it.

Case for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

The patient was a fireman and had been sent to St. Mary's Hospital for diagnosis by Sir John Collie. The history was that exactly six weeks previously the eruption had begun at first on his back, and then quite quickly spread until, as at present, it covered the trunk, back and front, and was less thickly grouped but definitely existent on the groin, thighs, legs and upper arms. The lesions now consisted of pigmented macules, very numerous, and shaped much as the lesions are in pityriasis rosea, but the colour was very much darker than the exhibitor had seen in pityriasis rosea, being a walnut or light mahogany colour. There was no scaling or crinkling of the surface as was so characteristic in pityriasis rosea. There was some general enlargement of glands—in axillæ, groin, and posterior cervical triangles. The patient had not had any chancre or other symptom of syphilis. He had been treated by Sir John Collie's assistant, on the assumption that the disease was psoriasis, with iron and arsenic. The pigmentation had apparently increased lately, so that some of the effect might be attributed to arsenic. Three Wassermann tests had been made, the report in the first two cases being "doubtful"; in the third it was stated to be negative. A portion of the skin from one of the more deeply pigmented areas was examined histologically. There was no inflammatory infiltration of the corium or in the neighbourhood of the vessels; there was probably a slightly increased number of mast cells in the superficial zones of the corium, but not enough to warrant a diagnosis of urticaria pigmentosa. The man had complained in the earlier stage of the eruption of considerable itching, and the colour of the lesions had suggested the possibility of urticaria pigmentosa. On January 27, some nine weeks after the appearance of the eruption, the rash had not faded and the pigmentation remained practically unaltered.

Dr. PRINGLE, Dr. WHITFIELD, Dr. SEQUEIRA, Dr. MACLEOD, Dr. PERNET and Dr. ADAMSON all considered the case as one of fading pityriasis rosea with a somewhat unusual degree of pigmentation.

Case of Dermatitis Artefacta.

By E. G. GRAHAM LITTLE, M.D.

THE patient was a spinster, aged 33, a schoolmistress at Bletchley, and the remarkable feature in the case was the strictly unilateral distribution. On the left leg and the left cheek there was a vesicular and excoriated surface with sharply defined borders; the greater part of the front of the leg and the whole of the left cheek were thus affected. There was no anæsthesia of the palate, as is so often present in these cases. The agent of production had not been identified, the patient having been seen only once. She had had previous lesions in the same position. No motive could be ascertained for the self-mutilation. An American observer had noted the curious frequency of the condition in spinster school teachers. In the exhibitor's experience the face was seldom chosen for the production of the artificial dermatitis, however widely this might be present.

DISCUSSION.

Dr. WHITFIELD said he detected a smell of acetic acid on the leg lesion. It was probable, therefore, that either strong acetic acid or more likely acetum cantharidis had been applied.

Dr. PRINGLE said he had an interesting case of similar nature in the Middlesex Hospital at the present time. The patient was a girl, aged about 26, employed as a masseuse in a "beauty specialist's" place of business in Bond Street. She had been for several weeks in a private nursing home when first seen, where she had been successful in producing crops of lesions apeing—by no means unsatisfactorily—dermatitis herpetiformis. The affected areas were almost "universal" as far as the skin could be reached by either hand, but the face and neck had been left alone. The parts had become septic, the temperature was high, and the patient had become really very ill. The nature of the case was immediately recognized on admission to the hospital, and the sepsis relieved by prolonged boric and starch baths. She produced a few vesicating lesions during the night on parts purposely left exposed, the agent employed being carbolic acid sent in from outside the hospital. The girl was silly and neurotic; the palate was absolutely insensitive; her general cutaneous sensibility was intact, but there was universal loss of appreciation of pain. Unfortunately, her thermic sensibility had not been tested. After her imposture was exposed, and the skin sepsis cured, she got quite well.

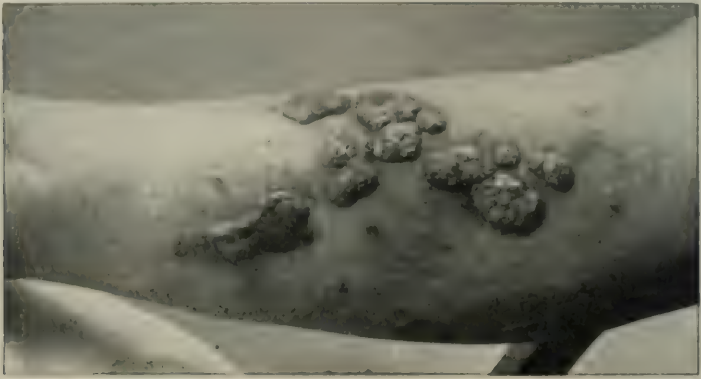
Dr. F. PARKES WEBER suggested that the production of skin lesions by this class of persons, by providing a kind of safety-valve to their feelings, sometimes made them temporarily more mentally normal in other ways, and possibly saved them from troublesome psychical disorders. Self-produced cutaneous lesions might in a kind of way take the place of attacks of hysterical vomiting, so-called "hysterical pseudo-appendicitis," and functional conditions simulating acute intestinal obstruction, for which laparotomy had occasionally been performed owing to mistaken diagnosis.

Case of Hypertrophic Lichen Planus.

By E. G. GRAHAM LITTLE, M.D.

THE patient was a woman, aged about 45, in whom the tumours had been present for at least two years. She gave no history of generalized lichen planus, and showed at the present time only very doubtful lesions of the flat type of lichen planus, on the front of the wrists and at the upper and inner part of the right knee. On the right leg, however, over the middle third, there was a remarkable efflorescence of tumours, raised

½ in. above the skin, rounded and oblong in shape, and about the size of the distal phalanx of the thumb in an average man. The surface was granular and lobulated, the mass overhanging its base so as to give the appearance almost of a pedunculated tumour, but in reality constituting strictly sessile excrescences. They were bluish in colour and very itchy; the tumours numbered about twelve. There was only one elsewhere than on the right leg, and that was in the left popliteal space. There was no lesion in the mouth. The patient had not been taking drugs, and was otherwise in good health.



Lichen planus hypertrophicus.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) said shaving off the lesions had been effectual in similar cases. He had known cases cured by X-rays. He had one case in which an enormous lesion, as large as an orange, was on the margin of the vagina, and that did very well with X-rays—four pastille doses.

Urticaria Bullosa in an Infant.

By J. H. SEQUEIRA, M.D.

URTICARIA of the common type, characterized by the development of extensive wheals, is rare in infancy, and there exists a difference of opinion as to whether strophulus or papular urticaria is to be considered a true urticaria. In the case shown, wheals have been the predominant feature throughout, and, in addition, the formation of bullæ have been a constant phenomenon during the past two months.

The patient, a female infant, aged 8 months, the daughter of parents of Polish origin, was first seen at the London Hospital in September, 1912, when she had a red papular eruption all over the body. Under a simple soothing ointment this eruption cleared up. In November, 1912, the infant was again seen. The skin of the chest, abdomen, and back was covered with wheals, and groups of flaccid bullæ, varying in size from that of a pea to that of a small nut, and containing a clear fluid, were scattered over the trunk. Many of the blebs ruptured and left raw surfaces which healed readily. From that time until January, 1913, the child has been seen several times a week and the condition has remained unchanged. Several times while under observation wheals have been observed to develop into blebs. Although there was no surface of healthy skin upon which factitious urticaria could be produced, the act of stroking or rubbing the affected area produced extensive whealing. The blood and also the serum from blebs were examined, but no excess of eosinophile cells was found.

There were three other children in the family quite healthy. The mother also appeared to be quite healthy and had been taking no medicine which could have accounted for the condition. The infant had been brought up at the breast throughout. It appeared quite well in other respects. The motions were of normal appearance and there was no sickness. Small doses of hydrarg. cum cret. had been given and an emollient applied locally. The baby had at first been washed with curd soap, but more recently oatmeal had been used in the water.

DISCUSSION.

The PRESIDENT said his experience of calcium lactate was that it was of no benefit in this condition. A very important point in bad cases of dermatitis herpetiformis and old-standing pemphigus was that starvation seemed to cure it. He was in Edinburgh a few weeks ago and he saw, in Dr. Norman Walker's wards, a case of dermatitis herpetiformis which had been of many years' standing. Every ordinary means of stopping it failed, and starvation was the only thing which influenced it—i.e., taking nothing but water. When he returned, he told this experience to a patient who had had this disease for many years, whom he had shown once before the Section, and she went without food for five days, with the result that the condition quieted down enormously. It was only in very bad cases that the patient could be expected to agree to this.

Dr. AGNES SAVILL said she sometimes saw cases cured by calcium lactate alone, but her custom was to give hydrarg. cum cret. first in order to disinfect the intestinal canal, for she had found the calcium did not act, in the majority of cases, until this prior clearance had been effected.

Dr. PRINGLE said the patient's skin was urticating under one's eyes. Some discussion having been raised by the President as to the value of calcium salts in similar conditions, Dr. Pringle said that he had never been able to convince himself of their possessing any therapeutic value for the relief of itching, or for the prevention of urticarial conditions, although he had administered them in all the various ways advocated since their introduction into practice several years ago. The value of lactate of calcium as a haemostatic previous to operations was, however, easily demonstrated. He agreed with Dr. Savill that eruptions in children, probably referable to intestinal toxaemias, were best treated by keeping the bowel thoroughly cleared out with mercury and chalk or other mercurials.

Dr. DORE remarked on the rarity of ordinary urticaria in infants; there must be some difference in the causation or in the texture of the skin of infants compared with that of adults, for in the former one found lichen urticatus, and in adults ordinary urticaria.

Dr. WHITFIELD considered that the condition at the present time was urticaria. Factitious urticaria was present in a marked degree.

Dermatological Section.

February 20, 1913.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case for Diagnosis.

By ALFRED EDDOWES, M.D.

THE patient, a married man, aged 56, showed a remarkably raised, circinate, gyrate, erythematous, psoriasiform eruption, distributed chiefly on the limbs, less upon the trunk, and absent from the head, face, and hands. The eruption had come and gone for several years; but a year ago it became much worse, and had increased up to the present, and now there are several tumours formed. The patient is liable to "indigestion," wind, and pain. Wife and five children living and well; two children died in infancy, the latter of whom had a rash soon after birth, and died when 4 months old, thirteen years ago. There have been no children since, and no miscarriages. At that time, and probably previously, he (the patient) had pimples under the soles of his feet. "Never had syphilis." No internal treatment has been given for twelve months, and no mercurial local treatment for seven months or more. Microscopically the chief change is seen in the epidermis, and presents some of the features of psoriasis, but with more disturbance in the interpapillary processes. Wassermann's sero-diagnostic test gave a negative reaction in all dilutions.

DISCUSSION.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) said that the Section was much indebted to Dr. Eddowes for bringing this case forward, and invited suggestions as to diagnosis and treatment. Personally, having seen five or six cases exactly similar, he had no doubt that it was a psoriasiform type of mycosis fungoides. He believed that considerable benefit would be got from X-ray treatment. One of his patients, whom he never could persuade to present himself before the Section, was treated by means of X-rays, and his life was unquestionably prolonged thereby. The tumours in his own cases came and went, just as they did in Dr. Eddowes's case, but with X-rays they tended to disappear. It would be well if, to this external application of the X-rays, large doses of arsenic were added internally.

Dr. ARTHUR WHITFIELD agreed with the President that this was a case of mycosis fungoides of the psoriasiform type, and that it would be amenable to X-ray treatment, with temporary, if not permanent, benefit to the patient.

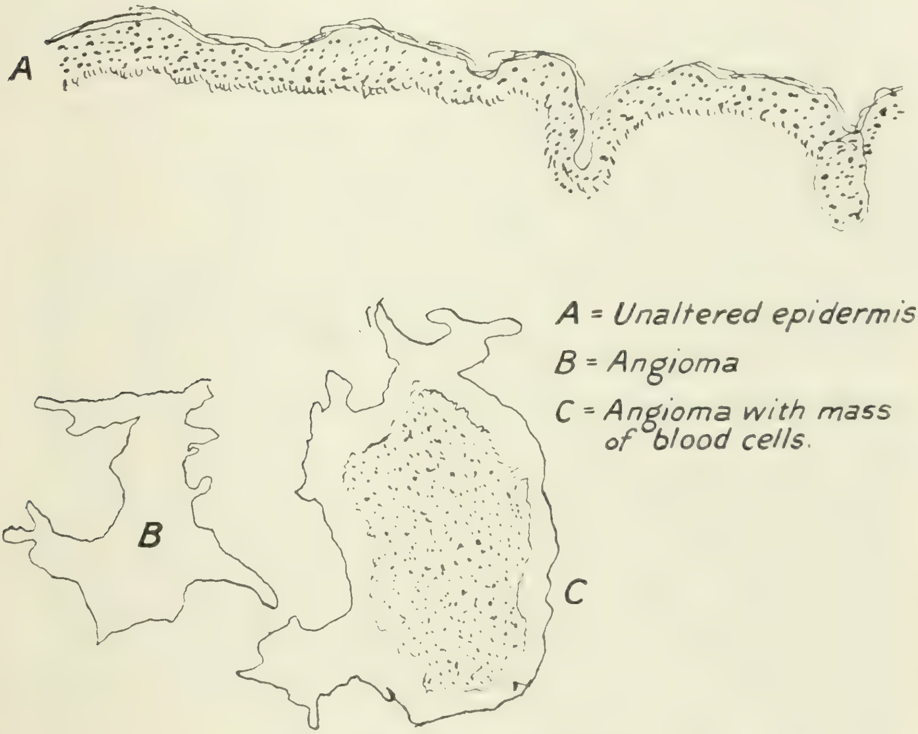
Dr. EDDOWES, in reply, said that he was very much obliged for the suggestion, and would certainly use X-rays and arsenic. The history was curious, in that this condition should have come and gone apparently for thirteen years, and should have been diagnosed as psoriasis. He would be very glad to adopt the treatment suggested, and to report progress, if possible, at a future meeting. He added that a microscopic section under low power was on exhibition, and it was striking to see the great change which had taken place in the epidermis, and how little was wrong with the cutis.

Case showing Multiple Subcutaneous Nodules.

By E. G. GRAHAM LITTLE, M.D.

THE nature of the case was doubtful before microscopic examination of sections which had since been effected, and this demonstrated the tumours to be a form of angioma. The case had been sent for diagnosis by Dr. W. W. Walker, of Cricklewood, who stated that the patient, E. W., aged 33, a cellarman at a club, had developed these lesions all within the last eighteen months. These were blue subcutaneous swellings, varying in size from ⅛ in. to ½ in. in diameter, semi-attached to the skin, and movable with it; giving somewhat an elastic feel on pressure, and not apparently changed in colour by deep compression, and in no way tender or sensitive; they were not in the course of veins or nerves, and were distributed chiefly on the backs of both hands, the forearms, forehead, trunk, thighs and legs, in all about thirty or more in number. There was a single brown pigmented mole on the face, but with this exception there was no other pigmented lesion. No visceral disease could be detected, the liver and spleen were of normal size. The man was anæmic but not ill-nourished, and felt well.

[The exhibitor had suggested the diagnosis of melanotic sarcomata of primary development in the skin, but the microscopic evidence conclusively demonstrated the nature of the growth, which may be thus described: The epidermis is unaltered. In the middle and deeper parts of the corium are numerous vascular dilatations (cavernous angiomata), the cavities in some cases being partially filled with aggregated masses of blood cells, and lined with several layers of cells (endothelium showing proliferation). Nuclear mitoses are present in small numbers, but as yet there is no evidence of invasion of the surrounding tissue, and therefore no evidence of malignancy.



Microscopical section of the growth.

The exhibitor has to thank Dr. B. H. Spilsbury, Pathologist to St. Mary's Hospital, for cutting the section and for furnishing part of the above report. The drawing, made with a camera lucida, roughly shows the appearances under the microscope with a 1-in. magnification.

DISCUSSION.

Dr. ARTHUR WHITFIELD said that he did not think the case was one of sarcoma. He believed it would prove to be a case of multiple angio-lipoma.

The PRESIDENT said that some years ago Sir Rickman J. Godlee showed him a case exactly similar, and which had begun in the same way as this.

It was unusual for the condition to begin cutaneously and then go on in the way this had developed. As to whether it was melanotic sarcoma, he did not see how anyone could make a diagnosis except microscopically.

Dr. STOWERS said that in 1893[1] he brought before the Society a case of melanotic sarcoma of the ear in a girl, aged 11. The upper two-fifths of the cartilage were removed, and the case had gone on most satisfactorily since then, with no relapse. It was not, however, of the same type as the one under discussion. He thought there was no question as to the diagnosis in Dr. Graham Little's case.

Case for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

THE patient was a lad, aged 14, who had been sent to the Inoculation Department at St. Mary's Hospital, about three years ago, with a chronic ulceration of both legs below the knees. He had been under observation and treatment for several months at a time. The ulcerations had healed and broken down again and again. For two years X-rays had been applied, but without much success. Various vaccines, prepared from organisms isolated from his own lesions, had been injected, including staphylococcus, streptococcus and coli bacillus; he had apparently made most progress with staphylococcus vaccines. At the present time the right leg was quite healed over, showing much scarring; but the left leg from knee to ankle was occupied by an infiltrated, sharply circumscribed inflammation of the skin, with œdema and several rather superficial ulcerations. Opsonic examination repeated frequently showed normal index to tubercle, and the Wassermann reaction had been consistently negative. The boy had been kept from school by his condition during the past three years. There was at present marked anæsthesia of the palate, and the suggestion of artificial dermatitis had been made. The patient would be admitted to hospital and watched with that possibility kept in view.

DISCUSSION.

Dr. J. H. SEQUEIRA said that for a considerable time he had a similar case under his observation at the London Hospital. The patient was a lad about the same age as Dr. Little's patient. He was employed as a telegraph messenger. He developed, apparently as a result of a slight injury, a chronic ulceration on the leg and wrist which lasted for months. As he did not improve as an out-patient he was admitted into the ward and the lesions were covered with an occlusive dressing. Under this they rapidly healed, but on the boy leaving the ward they soon reappeared. This occurred twice at least. It appeared that the boy, who was earning a small wage, was by some peculiar arrangement under the Workmen's Compensation Act getting exactly the same weekly payment whether he was at work or away on account of some trouble alleged to be due to his employment.

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[1] *Brit. Journ. Derm.*, 1893, v, p. 305.

The PRESIDENT could not help thinking that this boy under proper observation would get all right. He could hardly imagine an ordinary patient going on for so long a time. [Dr. GRAHAM LITTLE interjected that the boy was an epileptic and had taken bromides.] The President said that, of course, this complicated the situation. Bromide sometimes induced an extraordinary sensitiveness. He had a case at the present time in which the patient had developed an eruption on the leg. The patient was compelled to take bromide by order of the physicians, and the skin lesions were most painful. He asked whether members of the Section had noticed any difference in this respect between the effects of bromide of sodium and bromide of strontium. Physicians who were now giving bromide of strontium for preference said that it did not produce such eruption as the sodium compound. It seemed that there were differences between the various kinds of bromides in this respect, and some were supposed to be non-depressant and not to affect the skin.

Dr. ARTHUR WHITFIELD suggested the use of X-rays. In one case which was sent to him by a colleague the patient, who was taking 60 gr. of bromide per day, had a severe chin eruption. There was no question but that the lumps were diminished by means of the X-rays, and the treatment cleared up the condition for several weeks. He was sorry to say that the lumps had begun to come back, but the X-rays gave at any rate temporary relief. He followed the plan, when the condition was very bad, of giving him a pastille dose.

Case of Circumscribed Symmetrical Dermatitis, "Parapsoriasis en Plaques disseminées" (Brocq).

By E. G. GRAHAM LITTLE, M.D.

THE patient was a motor engineer, aged about 30, in whom the disease had been present for six years. He had been kindly sent up by Dr. Findlater, of Edgware, to whom the exhibitor expressed his thanks. Patches, roughly symmetrical, of scaly, dry, red, slightly infiltrated dermatitis, ranging in size from 3 in. by 2 in. to disks of about 1½ in. in diameter, were present on the upper and middle of the front of the thighs, on the hips, on the neck, on the legs, the back of the lower third of the forearm and the wrist. The scalp, trunk and face remained free. There was no itching, but slight sensation of burning in the parts affected. Scrapings from the scaly patches had been examined for fungus, with a negative result. The man appeared otherwise well. The extreme persistence of the lesions, which seemed unaffected by ordinary local treatment, their symmetry, and the relative absence of subjective symptoms, bring the cases into line with Brocq's group, cited above. The redness of the patches differentiated the case from Crocker's "Xantho-erythrodermia perstans."

DISCUSSION.

Dr. ARTHUR WHITFIELD said that in a private case with an appearance of this kind he had found a very large amount of oxaluria periodically (there was no stone), and he wondered whether that was associated at all with the condition. There was an entire absence of pain in this instance. He thought it probable that the case was not a local parasitic affection, but something of the nature of an intoxication. He had frequently examined the scales from such cases and had been unable to demonstrate any parasites.

Dr. J. M. H. MACLEOD said that he considered that the case belonged to the parapsoriasis group and was similar to that described by Brocq as "Erythrodermie pityriasique en plaques disseminées," and that it was not the same affection as described by the late Dr. Radcliffe-Crocker under the heading of "Xantho-erythrodermia perstans."

Dr. S. ERNEST DORE said that he had seen one such case, which had cleared up under X-ray treatment.

Dr. ALFRED EDDOWES said that he saw no striking difference between this condition and that of parakeratosis variegata. He had seen cases of the latter in which some parts of the rash looked extremely like this.

Chronic Raynaud's Symptoms, probably on a Syphilitic Basis, associated with Livedo reticulata; Remarks on Livedo reticulata (Livedo annularis, Livedo figurata, or Cutis marmorata).

By F. PARKES WEBER, M.D.

THE patient, E. P., is a married woman, aged 54, of medium height and weight, who has been subject to Raynaud's symptoms (of the local asphyxia type) during cold weather, chiefly in the left foot and left hand, for the last fourteen years. On one or two occasions very slight gangrene occurred at the tips of some of the toes of left foot. The last bad attack was about eight years ago. The hands have always been less affected than the feet. Cold always increases the symptoms, but she cannot stand exposure to artificial heat either. In explaining the way in which artificial heat affects her, she describes occasional spontaneous attacks of heat and flushing, rising up towards the head, and accompanied by sweating, the face and ears becoming red. She has been subject to these flushings since the age of 40, but they became more troublesome after the menopause, which occurred at the age of 49. Warming herself in front of a fire tends to bring on the "flushings," which are often followed by a slight feeling of coldness. No history of hæmaturia. She has had right-sided otorrhœa from chronic middle-ear disease more or less continuously since infancy. At the age of 19 she was laid up for six months with a severe attack of rheumatic fever, accompanied by heart trouble. She had been an out-patient at the Western Ophthalmic Hospital for eye trouble, and I am much indebted to Dr. Rayner Batten, under whose care she was, for information about her. Her sight began to fail in December, 1909. Dr. Batten says that in 1910 he found the right eye myopic, whilst the left eye showed numerous retinal hæmorrhages and irregular beading of retinal vessels. After December, 1909, the patient became subject to recurrent attacks of temporary amblyopia, or even complete amaurosis, chiefly in the left eye, lasting only a few minutes, and sometimes accompanied by a little pain at the back of the eye. She has not had any ocular attacks of this kind recently, but they continued until about six months ago, at which time she almost completely lost the sight of her left eye.

There is no history of syphilis, but she says she has had nine miscarriages, the last when she was aged about 39. She has had no living children.

Present condition: The hands are very red and tend to become cyanosed, but not nearly so much so as the toes, which almost always look bluish, especially the fourth toe of the left foot, from the tip of which one can still see that there has been slight loss of substance. The blotchy mottling of the skin or "livedo reticulata" ("livedo annularis," "livedo figurata," or "cutis marmorata") is very distinct over the whole of the patient's back, but is more striking over the extensor surfaces of the upper extremities (*see* fig. 1), especially on the upper arms near the elbows, and on the forearms near the wrists. In the lower extremities it is well marked on the front of the thighs near the knees (*see* fig. 2), and on the front of the trunk it is most noticeable about the waist. The mottling can be made to disappear temporarily by rubbing the skin when in a warm room. It becomes very much less marked in warm summer weather. The superficial cutaneous hyperaemia which follows venous constriction in the upper extremities (as in the process of ascertaining the brachial blood-pressure) is much greater than in ordinary persons. The white mark left by digital pressure on the mottled skin does not disappear in the extremely rapid way described by Ehrmann as characteristic for his cases of livedo of syphilitic origin.[1] There is no factitious urticaria to be elicited.

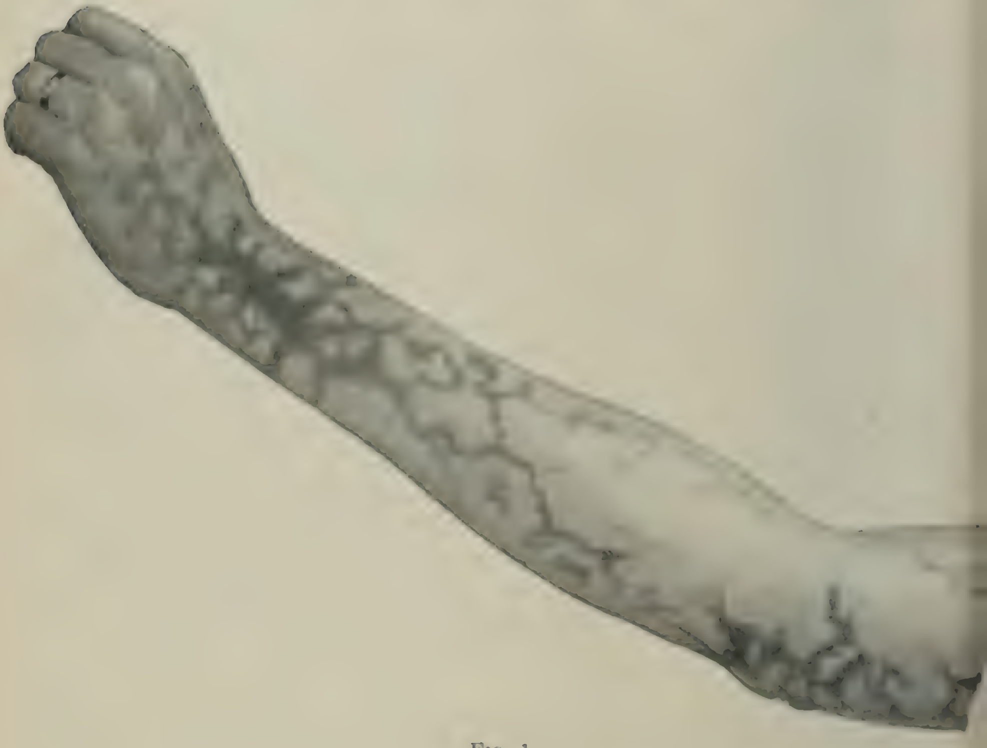

FIG. 1.

Examination of the patient's heart shows that the apex beat is in the fifth intercostal space, in the nipple line; the impulse is very forcible; the first sound at the apex is loud and "thumping," and preceded by a typical presystolic murmur of mitral obstruction. The lungs and abdominal viscera show nothing abnormal to ordinary examination. The liver and spleen are not enlarged. There is no fever. Pulse, 80 to 88 per minute; respirations, 22 to 24 per minute. The brachial systolic blood-pressure is very high; in both arms it measures 240 mm. Hg.

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[1] See S. Ehrmann, "Ein neues Gefässsymptom bei Lues," *Wien. med. Wochenschr.*, 1907, lvii, p. 777.

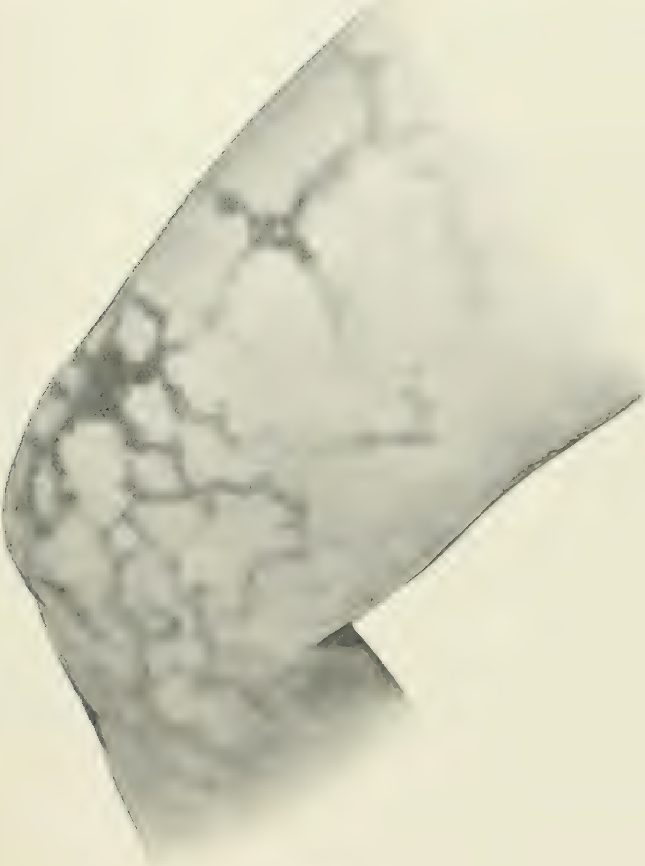

FIG. 2.

The urine (daily quantity about normal) is of low specific gravity (1010) and contains a little albumin; it is clear, pale, slightly acid, and free from sugar, tube casts, blood and pus cells. Blood examination (February 17, 1913): Haemoglobin, 70 per cent.; red cells, 5,160,000, and white cells, 9,800, to the cubic millimetre of blood. The microscopical examination of blood films and the differential count of the white corpuscles shows nothing abnormal. The blood serum (February 10, 1913) gives a negative Wassermann's reaction for syphilis. The knee-jerks are active; the plantar reflexes are of flexor type; the hand grasp is good on both sides, and sensation is normal. At present the right eye shows ophthalmoscopic changes connected with myopia, and with a suitable glass the vision is $\frac{6}{18}$; the pupil reacts normally to light. In the left eye there has been hæmorrhage into the vitreous, and vision is reduced to mere perception of light.

The case is interesting from various points of view. In my opinion both the Raynaud's symptoms and the livedo may be regarded as having developed long ago, probably "on a syphilitic basis," although Wassermann's reaction for syphilis is now negative. The congestive influence of the mitral stenosis on the circulation doubtless favours both the livedo and the Raynaud's phenomena. A certain amount of arterio-sclerosis and chronic interstitial nephritis are almost certainly present, and are possibly also connected with old syphilis.

The early symptoms in the left eye, especially the attacks of temporary amblyopia, were probably in part due to retinal angiospasm, and allied to the Raynaud's phenomena, which were likewise best marked on the left side, notably in the left foot.

In the case of a man, aged 59, with Raynaud's phenomena, observed by Raynaud himself,[1] partial loss of vision followed the attacks of local asphyxia in the extremities. During such attacks of lividity in the extremities, when vision was at its best, the branches of the retinal artery showed partial constrictions. L. E. Stevenson[2] described the case of a woman, aged 25, with Raynaud's disease leading to gangrene of the toes. She suffered likewise from recurrent attacks of temporary complete or partial loss of vision, which Stevenson supposed to be due to spasm of retinal arteries. G. H. Fox[3] narrated two cases of Raynaud's disease, in both of which sudden, paroxysmal impairment of vision was a feature. G. A. Friedman[4] published the case of a young woman, aged 23, with Raynaud's phenomena, in whom ophthalmoscopic examination showed marked contraction of the small arteries of the fundus oculi when the asphyxia of the extremities was most pronounced. Weiss[1] has recently observed the case of a man, aged 54, who is occasionally subject to angiospastic attacks in the left hand. The most interesting feature of the case is the occurrence of transitory attacks of amaurosis, during which angiospastic phenomena have been watched, by ophthalmoscopic examination, in the right eye. The retinal arteries, and then the veins, were seen to empty themselves, so that the vessels came to look like yellowish-white threads. After half an hour the vessels began to refill, at first the small cilio-retinal vessels, then the veins, and then gradually the larger arteries. At the height of the attack there was amaurosis, and the central scotoma remained for half an hour after the ophthalmoscopic picture had become normal again. Several cases of temporary angiospastic amaurosis have been recorded in which the retinal angiospasm was apparently not known to be associated with Raynaud's symptoms in the extremities; in some of them angiospastic phenomena in the retinal arteries were observed by ophthalmoscopic examination. Hans Curschmann[2] narrated the case of a woman, aged 43, who suffered from attacks of angina pectoris, and had temporary right-sided amaurosis, probably due to retinal angiospasm. It must be remembered, however, that some attacks of transient amblyopia in patients subject to angiospastic phenomena in the extremities may be due to temporary circulatory conditions in the brain and not to peripheral intra-ocular causes.[3] With these cases may perhaps be compared that of a woman, aged 51, suffering from well-marked Raynaud's phenomena in the hands, and also from vasomotor aural (labyrinthine) disturbances.[4] One might likewise here call to mind that ocular attacks of a different nature to those already mentioned may occur in subjects of Raynaud's phenomena. Thus, M. Weiss,[5] of Prague, recorded a case characterized by intermittent Raynaud's manifestations, and attacks of certain ocular symptoms, which he referred to the cervical sympathetic nerve and which alternated with some of the vasomotor attacks in the extremities.

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[1] Raynaud, *Arch. générales de Méd.*, Par., 1874, i, p. 8.

[2] Stevenson, *Lancet*, 1890, ii, p. 917.

[3] Fox, *Journ. Cutan. Dis.*, New York, 1907, xxv, p. 336.

[4] Friedman, *Amer. Journ. Med. Sci.*, Philad., 1910, cxxxix, p. 238.

In regard to the livedo in the present case, I would again mention that the mottled skin does not react in the exact way described by Ehrmann in his cases of syphilitic livedo or "livedo racemosa."[1] Yet I suspect that the differences are somewhat arbitrary (chiefly a matter of degree), and that really the livedo in Ehrmann's cases[2] is allied both to the livedo reticulata in the present case, and to the remarkable reticulata occasionally met with in young persons on the extensor surfaces of the upper extremities (especially of the forearms, near the wrists) and on the thighs (near the knees), probably of congenital or developmental origin. The localization is important from a diagnostic point of view. Thus, in a young woman, if cervical ribs are present, patches of livedo reticulata on the forearms may be attributed to the presence of the cervical ribs, but if similar patches of reticular livedo are found near the knees, it becomes very unlikely that those on the forearms are caused by the cervical ribs. The localization alone is sufficient to distinguish the reticular livedo in the cases to which I have alluded from the reticular "erythema ab igne" (erythema figuratum ab igne) and pigmentation over the shins, due to sitting in front of a fire, and from the similar reticular "erythema a calore," and pigmentation due to the prolonged or habitual application of hot fomentations or india-rubber hot-water bottles (on account of pain, &c.), for instance, to the back or abdomen.

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[1] Weiss, "Communication to the Thirty-eighth Congress of the Ophthalmological Society, Heidelberg," *Münch. med. Wochenschr.*, 1912, lix, p. 2074.

[2] Curschmann, *Deutsch. med. Wochenschr.*, 1906, xxxii, p. 1527, Case 3.

[3] *Cf.* Curschmann, loc. cit., Case 2.

[4] H. J. Davis, *Proc. Roy. Soc. Med.*, 1912, v (Otol. Sect.), p. 156.

[5] M. Weiss, "Ueber symmetrische Gangrän," *Wien. Klinik*, 1882, viii, p. 347.

In this connexion it should also be remembered that chronic venous obstruction of any kind may favour or cause the appearance of a local livedo reticulata. Thus, heart disease (especially mitral stenosis), with imperfect compensation, may favour the development of widespread livedo reticulata (this subject I have discussed elsewhere), and chronic obstruction, from any cause, in the great veins leading from the upper extremities, may be associated with reticular livedo and reticular pigmentation in the forearms.

Livedo reticulata, which sometimes very much resembles post-mortem mottling in appearance, though a good deal has been written about it, is, nevertheless, not generally well known. It varies greatly in degree and in extent of distribution. It may be localized so as to form patches (generally symmetrical patches) on the backs of the forearms, about the knees, &c., or it may be widespread, involving not only the extremities, but also the whole of the back and portions of the front

[1] I think it is better to use the term "livedo racemosa" as merely synonymous with "livedo figurata," "livedo annularis," and "livedo reticulata."

[2] Ehrmann, loc. cit. See also Karl Schmidt, "Zur Kenntniss des Ehrmannschen Luesphänomens," *Arch. f. Derm. u. Syph.*, Vienna, 1912, cxiv. p. 191.

of the trunk. Thus, it may be almost universal in distribution, though in such cases the different parts are not affected to the same degree, the colour of the livedo, whether red or bluish, being much more marked in some parts of the body than in others. I suspect that various forms of livedo reticulata and allied cases have been described under many names,[1] including "purpura annularis telangiectodes" ("telangiectatic annular purpura"), and that possibly some other cases described under the latter name may really have been examples of Hutchinson's "infective angioma" ("nævus-lupus," "serpiginous nævus").

DISCUSSION.

Dr. ALFRED EDDOWES thought that certain of the symptoms were as likely to be partially due to phlebitis as to arteritis. It was very likely, in his opinion, that there was resistance on the vein side, due to endo- and peri-phlebitis, a common condition in syphilis.

Dr. PARKES WEBER said, in reply, that there was no doubt about the presence of arterio-sclerosis in this case.

Case of Frambœsiform Syphilide of Palms.

By J. E. R. McDONAGH, F.R.C.S.

S. W., A MAN, aged 22, engineer by trade, contracted syphilis in January, 1912. The chancre on the prepuce was followed by an ordinary generalized maculo-papular eruption, which disappeared quickly under treatment with the exception of some lesions on the face and both palms, which became gradually worse. In spite of twenty-eight intramuscular injections of grey oil and calomel, potassium iodide internally, and the local application of mercurial ointments, the lesions which the patient now presents have scarcely altered since their appearance nine months ago. If antisyphilitic treatment is suspended the lesions immediately begin to increase in size.

This case is not shown so much for its rarity, but more with the idea of contrasting it with the frambœsiform syphilide which most commonly affects the scalp, and which responds to treatment so readily. The behaviour to treatment is no doubt regulated by the blood supply of the part affected, and we have in the frambœsiform syphilide of the scalp and palms an analogy to the soft and hard node. How peculiarly resistant to treatment are also those hyperkeratotic recurrent syphilitic papules which affect the flexor aspects of the palms and fingers, and not infrequently the nails! The lesions usually appear between the second and fourth year after infection, and in spite of the most vigorous treatment they slowly disappear while fresh lesions take their place. During the last three years I have had two cases under my care with the lesions just mentioned, and which were not prevented from recurring by ten injections of salvarsan and continued courses of mercury and iodides.

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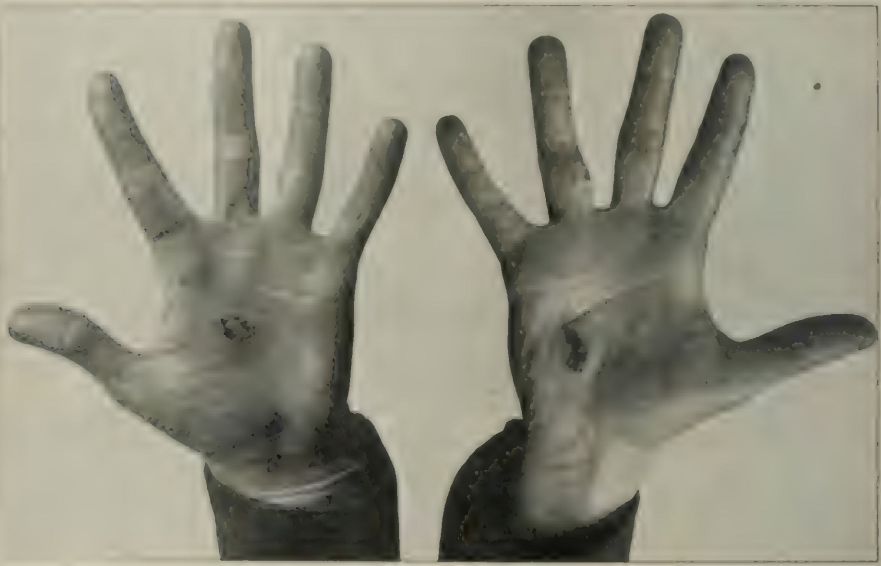
[1] Some of the cases referred to by Sir William Osler, in his paper "On Telangiectasis Circumscripta Universalis" (*Bull. Johns Hopkins Hosp.*, Balt., 1907, xviii, p. 401), may perhaps have belonged to the livedo reticulata group.



Frambœsiform syphilide of palms.

[Later note.—The lesions were aggravated by local mercurial treatment, and are now rapidly disappearing under salvarsan.]

DISCUSSION.

Dr. R. E. Scholefield said that some time ago in private he had an almost exactly similar case, but only one hand was affected. It was on a syphilitic basis, and entirely cleared up under X-rays after being nearly two years in that condition. In that instance ordinary syphilitic treatment had little or no effect, although it had been carried out for a year or more.

Dr. JAMES GALLOWAY remarked that the term "frambœsiform syphilide" seemed to be more aptly applied to the multiple, definitely papillomatous lesions which made their appearance in the early periods of the disease. These lesions produced very little ulceration. They seemed to be of rare occurrence, but he remembered bringing forward a case some years ago of this type of syphilide. The patient was a young woman who was admitted to the hospital with a doubtful diagnosis of lymphadenoma, on account of the great enlargement of the lymphatic glands in the neck. On examination she was found to present the remains of a primary infection of the lower lip; the great glandular enlargement was the consequence of this and was accompanied by a considerable degree of fever and constitutional disturbance. While in this condition she developed a papillomatous frambœsiform syphilide. It occurred, therefore, in an early stage of the malady. The treatment adopted was by means of mercurial inunctions. The eruption rapidly disappeared, leaving practically no ulceration; the glands diminished in size, and the patient, though remaining under observation for some time, had no further manifestations. Ulcerating granulomatous lesions of the palms of the hands, as in the case presented by Mr. McDonagh, were almost always difficult to heal. The position of the lesions and the peculiar characteristics of the epithelium of the palms were probably the chief factors in preventing rapid healing, and it might be well worthy of consideration whether local methods of treatment would be of greater value than too long persistence in general antisyphilitic medication.

Dr. GRAHAM LITTLE had noted a remarkable improvement in a very chronic tertiary syphilitic lesion, resembling somewhat this case, after two applications of freezing with carbon dioxide snow.

Mr. McDONAGH, in reply, said that he did not mean to suggest that all palmar syphilides did not disappear under treatment; on the contrary, most did so, and quickly, and only to those types which remained uninfluenced did he intend to refer. The reasons why he gave the name of frambœsiform syphilide to the lesions of the case presented were: (1) Because they were indistinguishable from the true lesions of yaws affecting the palms; (2) because they resembled almost exactly the more common type of syphilitic lesion affecting the scalp, which went by the name of "frambœsiform"; (3) because, like all frambœsiform syphilides, they appeared very early in the disease.

Case of Dermatitis following Large Dose of Arsenic.

By J. M. H. MACLEOD, M.D.

THE patient was a little girl, aged 7. She was a patient of Mr. Devereux Marshall, at the Moorfields Eye Hospital, for sympathetic ophthalmia, and the exhibitor was indebted to him for kind permission to show the case. She was seen first by the exhibitor at Charing

Cross Hospital in December, 1912, when she presented a profuse eruption, most marked on the trunk and face. Previous to that for two months she had been under treatment at Moorfields Hospital with intravenous injections of salvarsan, and had had 0·4 grm. on October 5, 0·6 grm. on October 22, and 0·6 grm. on November 6. A week after the last injection the eruption appeared. It came out first on the chest, and gradually involved the arms, face, scalp, upper parts of the thigh, and dorsum of the feet, being most profuse on the face and over the abdomen. It consisted of brownish-red macules, circular or irregular in outline, about the size of a pin's head, and tending to coalesce to form small irregular patches. Some of the smaller lesions were follicular in origin. The macules were covered with small, greyish, adherent scales or horny crusts, which did not extend to the periphery of the macule. It was associated with slight itching. Some days after the eruption appeared the skin of the palms and soles became diffusely red, then definitely thickened from hyperkeratosis. This was associated with changes in the nails, consisting of inflammation about the posterior nail wall, hyperkeratosis of the nail-beds, and a raising up of the free border of the nail.

The symptoms gradually subsided under a soothing calamine cream. At the end of January another injection of salvarsan was given *per rectum*, and a fortnight later an eruption similar to the first attack appeared. When exhibited, the second outbreak had almost completely subsided, except a few indefinite scaly macules and pitting of the nails.

It was difficult to make a firm diagnosis from the condition presented at the time of exhibition. It was suggested that the eruption might be psoriasis, which was improbable, as the original outbreak only superficially resembled that disease and differed from it in the brownish tinge of the lesions, the adherent, horny scales, and the diffuse keratosis of the palms and soles.

DISCUSSION.

Dr. Graham Little recalled an example of an acute very extensive vesicular eruption, coming out almost like an exanthem, but without rise of temperature, in a lad, aged 15, who had been given arsenic for about a week previously to the appearance of the eruption.

Dr. H. G. Adamson thought the eruption now present was psoriasis of the punctate type not uncommon in children. He called attention to pitting of the nails, which, he thought, supported the diagnosis of psoriasis.

Dr. F. Parkes Weber said that, of general exanthems resulting from the internal administration of arsenic the vesicular forms were probably severer, or else more acute, than the dry ones.

Dr. ARTHUR WHITFIELD said that he did not agree with Dr. Adamson with regard to the diagnosis of psoriasis. He considered that the whole eruption might be due to salvarsan, and he did not regard the nails as characteristic of psoriasis. The nail plate was pitted like the peel of an orange, and this was a frequent concomitant of eczematous dermatitis.

The PRESIDENT said that he thought this was a case of psoriasis. Psoriasis in a very young child was always very difficult to diagnose. He had seen a very large number of cases of arsenical poisoning, but had never seen one like this.

Three Cases of Tinea Tonsurans cured by X-rays.

By J. M. H. MacLeod, M.D.

THESE cases were brought forward to illustrate a difficulty in connexion with the technique. In two of them the defluvium of the hair had taken place, the exposure having been given a month previously; in the third case, which was only X-rayed ten days ago, the hair had not yet fallen out. The technique employed was the usual Kienböck-Adamson method, the Sabouraud pastille being used to estimate the dosage, and the exposures being given at Charing Cross Hospital by Dr. Maurice Hannay, assistant in the Skin Department. In two of the cases a marked erythema appeared about a week after the exposure, while in the third there was scarcely any perceptible erythema. In the two cases in which the erythema was marked the same tube had been used, in the other case another tube. It has been found that the tube which caused the erythema was capable of doing so with an exposure under a pastille dose, and was, in consequence, a "dangerous tube." Some months ago a tube in use in the department behaved in a similar way and led to imperfect re-growth of the hair.

What it was in the tube that made it dangerous the exhibitor had been unable, so far, to ascertain. It did not seem to be any defect in the position of the anticathode, or difference in the thickness of the glass, and the tube appeared to be identical with one made about the same time, which was safe. It was not a question of peculiar susceptibility on the part of the patient, as whenever a "dangerous tube" is used and a pastille dose given marked erythema and impaired re-growth result.

DISCUSSION.

Dr. J. H. SEQUEIRA said that he had occasionally seen slight erythema following the use of the X-rays in ringworm, but he had not been able to trace it to any special tubes. He had not seen such a condition lead to permanent baldness or to any impairment of the growth of hair.

Dr. H. G. ADAMSON said that a possible source of error was a faulty position of the target. He had had the misfortune to produce a dermatitis for which no reason could be discovered, until it was found that the target was so much advanced towards the cathode that the rays which fell on the pastille had to pass through the thicker glass towards the neck of the bulb. As a result, when the pastille registered the B tint the scalp had already received considerably more than a "pastille dose." Such an error could be avoided in future by carefully testing every new tube with a pastille in the usual position upon the holder and another in the path of those rays which would reach the scalp. They ought, of course, to correspond. An accident of this kind could be also avoided, as Dr. Whitfield suggested, by placing the pastille holder towards the side of the bulb instead of towards the cathodal pole, as was usual in this country.

Dr. S. ERNEST DORE said that at the present time he had a tube which would cause an erythema with half a pastille dose, and had done so in four patients.[1]

Case of Multiple Lupus.

By J. H. SEQUEIRA, M.D.

THE case illustrated three interesting points: (1) The lupus was very widely disseminated; (2) it followed measles; and (3) its dry, scaly character suggested psoriasis and it has been treated as such for several years.

The patient, S. H., aged 11, was an only child. His parents were healthy and there was no history of phthisis or of any cutaneous disease in the family. At the age of 3 some tuberculous glands, some of which had broken down, were removed from the right side of the neck. At the age of 4 the child had measles, followed by pneumonia. On his recovery, some spots appeared on the thighs, face and neck. About six months later these spots had spread into large patches. An attack of "shingles" also occurred about this time, but, after the herpes lesions had cleared up, the other spots still persisted. Ointments were applied without relief. As the eruption continued to extend, the child was taken three years ago to an infirmary, where the eruption was thought to be psoriasis, and treatment by tar ointment and alkaline baths was carried out steadily for six months. Since then, soda baths have been used to keep down the scales.

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[1] Subsequent reference to the notes of these cases showed that the erythema appeared within a week after the exposure, and was, therefore, probably due to static discharges from the tube; such a tube would not necessarily cause permanent atrophy of the hair in treating a case of ringworm of the scalp, although it had not been used for this purpose.—S. E. D.

Recently the boy was seen by another medical man, who thought the case was one of lupus, and he was sent up to the London Hospital for treatment. The boy was well grown, but rather anæmic and rather fat. He has generally good health, but felt the cold very much. There was no evidence of visceral disease, but the bowels were rather inclined to be loose. The eruption had the following distribution: On both sides of the neck and under the chin there were almost symmetrical patches of dry, scaly lupus, one patch being sore and ulcerated from a scratch. On both cheeks there was a small discrete nodule the size of a pea. On the anterior folds of both axillæ and on the posterior fold of the right axilla and on both arms there were many well-defined patches of lupus. The patches were dry, red and scaly, but showed under the diascope characteristic nodules. On both elbows, but more on the left than the right, there were very scaly patches which closely resembled psoriasis. The extensor aspect of the limbs was more affected than the flexor surfaces. On the outer and inner aspects of the right wrist and the outer side of the left wrist there were lesions of a thicker and more warty character. There were a few small discrete spots on the front of the chest. On the front and inner surfaces of the thighs there were more extensive areas, some as large as the palm of the hand, and some extensive areas extending on to the buttocks from the outer surfaces of the thighs. These were all of the dry scaly type seen on the upper limbs. The knees, except for small nodules on the outer aspects, were free. Both calves were the seat of large patches of similar character, but the shins were quite free. In front of the left ankle there were small ovoid areas extending transversely; these had the verrucose character seen on the wrist; in spite of the wide distribution of the disease the back and the scalp were quite free.

The exhibitor looked upon the case as one of the post-exanthematic type, and accepted the explanation which Dr. Adamson had given in similar conditions, that the exanthem, here measles, had caused the breaking down of a previously existing tuberculous focus. In this case the history exactly supported this hypothesis. Manifestly, so extensive an eruption would be difficult to treat. It would be impossible to apply the Finsen light to more than the areas on the face and neck. It was proposed to deal with the limb areas by strong plasters of creasote and salicylic acid, and to direct attention to the patient's general condition.

DISCUSSION.

Dr. ARTHUR WHITFIELD referred to Dr. Sequeira's remarks as to the absence of any patches on the back, and said that lupus very rarely affected the back above the iliac crests. He had photographed a case for Dr. Colcott Fox in which an extraordinarily rapid extension had occurred from a lupus of the face, so that the whole of the front of the body had become converted into lupus tissue, but there was no extension on to the back.

Dr. H. G. ADAMSON said that these cases at an early stage were often mistaken for chicken-pox. He did not suppose that they ever arose as multiple infection of chicken-pox lesions. It was merely an error of diagnosis suggested by the almost sudden appearance of the lesions. He had seen one case in the Hospital for Hip Disease in this very early stage. The boy had had measles a few weeks before admission, and while in the hospital a profuse generalized papular eruption had appeared. This was thought by the sister to be chicken-pox. The papules, however, though small, were distinct apple-jelly nodules. In a few weeks' time many of these nodules had multiplied to form small typical lupus patches, while many others had faded away.

Microscopical Specimens from Cases of Rhinoscleroma.

THE PRESIDENT said that recently he was at Pellizari's clinic in Florence, where three cases of rhinoscleroma were receiving attention. From a culture made from one of them kindly given him by Professor Pellizari, Dr. Dore had made sub-cultures, and these had been brought for inspection at the meeting. The organism in question was, he believed, so close to the pneumo-bacillus of Friedländer as to be almost unidentifiable separately.

Dr. S. ERNEST DORE said he had made sub-cultures from Pellizari's original culture. The organism formed a semi-translucent mucoid growth, tending to become white at the top and edges, on agar and glucose-agar, and small white colonies on gelatine which it did not liquefy. It was a short, coccoid, Gram-negative, capsulated bacillus corresponding in every particular to the bacillus described as the causative organism of rhinoscleroma by Frisch, and closely resembled Friedländer's pneumo-bacillus.

Dermatological Section.

March 13, 1913.

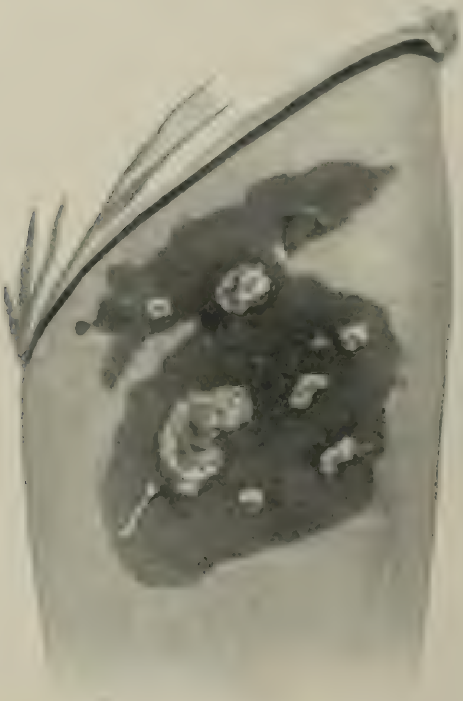
Dr. J. H. Stowers, Vice-President of the Section, in the Chair.

Artificial Skin Eruption.

By F. Parkes Weber, M.D.

The patient was an unmarried Swiss woman, aged 33, a domestic servant in England, who came to the hospital on February 10, 1913, complaining of a large, irregularly shaped patch of erythema over the back of the left foot. On this patch there were several bullæ, and I understood that the patient had had attacks of a similar skin eruption previously, as if she were subject to a kind of recurrent "erythema bullosum." The possibility of artefaction was thought of, but the patient, who seemed normal except for the eruption, had arranged to go back to Switzerland in a few days' time, and as she was apparently anxious that the condition of her foot should not detain her, she seemed unlikely to have produced the eruption herself.

On February 22 she was admitted under my care, as a case of erysipelas, with a supposed axillary temperature of 103·2° F. The foot had in the meantime nearly healed, but on the upper part of the front of the left thigh a large, angry-looking patch of erythema had developed, with several bullæ of various sizes over the bright red (and as I think, swollen) skin. On February 24, however, I ascertained that though febrile temperatures continued to be charted, the patient's pulse did not at all correspond to her temperature. With an axillary temperature of over 103° F. the pulse had been charted as 78 per minute, and with a supposed temperature of 102° F. her pulse was only 68. Since admission her pulse had varied between 68 and 88, and her respiration between 20 and 28 per minute. In the evening of February 24 the temperature *per rectum* was found to be only 99° F. It then became practically certain that the fever had been simulated and that the eruption, first on the foot and then on the thigh, had been artificially produced in some way by the patient herself. Next morning, when the accompanying drawing (*see* figure) was made, there was no fever, the



Artificial eruption on the upper front part of the left thigh. The erythema was fading and the bullæ were drying up at the time of this illustration.

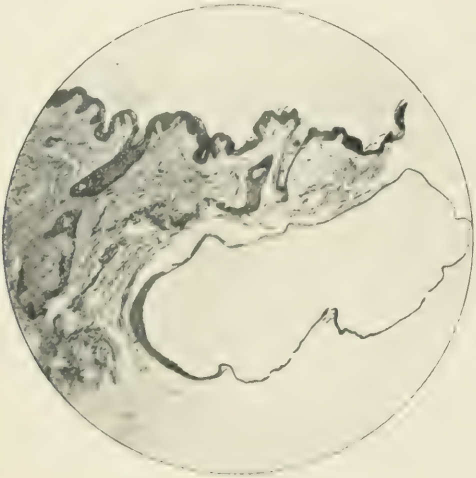
bullæ were drying up, and the erythema was rapidly fading. The patient was able to leave England for the Continent on February 26, but how she produced the eruption was not ascertained.

Case of Hidradénomes Éruptifs (Syringoma).

By J. H. SEQUEIRA, M.D.

THE patient was a girl, aged 23, who enjoyed good health, and, until three years ago had never noticed any abnormality of the skin. The family history was good, and there was no similar eruption in any member.

Three years ago the patient noticed some small "lumps" on the front of the chest. The growths did not itch, nor did they give her any pain. A year ago similar lesions began to appear on the back. Recently a few small growths have appeared on the throat. In the triangle between the mammæ, and in the epigastrium, and on each side of the chest, and on the back, chiefly in the scapular regions, there were many pale, discrete growths, raised above the surface of the skin, and quite tense and hard to the touch. They varied in size from a pin's head to a lentil seed, and a few reached the size of a split pea. Some of the little tumours were yellowish in colour, others paler and of the colour of the skin. On the back there were a few almost linear lesions, while on the front of the neck the papules were pinhead-sized. There was no infiltration about the growths.



Hidradénomes éruptifs (syringoma).

A small growth from the back was excised, and Dr. Turnbull made the following report: "The superficial epidermis is of normal thickness and shows normal inter-papillary processes. Hair-follicles, one furnished with a sebaceous gland, are present in the dermis. There is one large rounded cystic cavity, and a portion of the wall of a second cavity in the dermis. The wall of these cavities is lined by several layers of stratified epithelium. The inner cells are flattened and slightly eosinophilous; beneath this there are a few polygonal cells and a basal layer. The cells of the basal layer are short columnar or cubical. There is no stratum granulosum. The complete cyst lies immediately below the upper extremity of a hair-follicle, and is apparently derived from this hair-follicle or its gland. This origin of the cysts is also indicated by the position of the complete and incomplete cysts relative to the hair-follicle which is furnished with a sebaceous gland."

The case is one of an interesting group to which many names have been applied. The cysts apparently develop from epithelial tubes, but whether these are sweat-ducts or pilo-sebaceous ducts cannot be settled by the sections in this case. Jacquet and Darier's [2] original name of "Hidradénomes éruptifs" was given on the supposition that the eruption started from the sweat-ducts. On the hypothesis that the eruption is nævoid in character, Gassmann [1] applied the name "Nævi cystepitheliomatosi disseminati," which has also been used by Pernet [4] in describing a case. Török [5], McDonagh [3], and others prefer the name "Syringoma," which does not commit one to the type of duct from which the cysts arise.

The eruption is evidently the same as that described by Kaposi as lymphangioma tuberosum multiplex, but it has no relation to the lymphatic system.

(Dr. Sequeira is indebted to Henderson Baird for the opportunity of examining and showing the case.)

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[3] McDONAGH. *Brit. Journ. Derm.*, 1912, xxiv, p. 228.
[4] PERNET. *Brit. Journ. Derm.*, 1907, xix, p. 67.
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Localized Sclerodermia (Morphœa).

By G. Norman Meachen, M.D.

The patient was a girl, aged 14, rather anæmic, who was sent to the Prince of Wales's Hospital by Dr. G. Basil Price. She had had psoriasis eight years previously, and was once said to have had "a slight tendency to chorea." Eighteen months ago the left thumb-nail split without apparent cause, and almost immediately afterwards a white streak was observed upon the back of the terminal phalanx equal in breadth to the radial portion of the split nail. This band spread upwards until at the time of the meeting it had extended as far as the base of the first metacarpal. The surface was devoid of hair, and it was notably thickened. The borders also were slightly bluish. There was no history of injury. The mother had suffered from chorea, and she stated that the girl was of "a very nervous disposition."

DISCUSSION.

Dr. Sequeira said he had now under his care a girl of about the same age with a patch in the cleft between the thumb and forefinger; one band went up the forefinger and another along the thumb, a distribution suggestive of the bifurcation of the nerves. There was no history of trauma in this case. He had applied the X-rays through an aluminium filter, and the girl could now move the thumb and finger better than before. He had had no experience of cataphoresis with salicylate ions.

Dr. F. Parkes Weber said he thought the tendency of localized sclerodermia was spontaneously to leave off spreading after it had existed for a certain time. At present he had a woman under his care in the hospital whom he had seen many years ago with a long stripe of localized sclerodermia on one thigh and with a patch also in the lumbar region. Since that time there had apparently been no extension of the sclerodermia. She was now in the hospital on account of internal cancer. Her present age was 44, and according to her account the sclerodermia had not progressed since she was aged about 18. It formed a hard, depressed band on the right thigh, extending from a spot between the great trochanter of the femur and the crest of the ilium, passing along below the fold of the groin to the inner anterior aspect of the thigh and down the inner part of the front of the thigh to close above the knee. The isolated lumbar portion constituted a hard, depressed plaque, about the size of a five-shilling piece, to the right of the vertebral column, between the iliac crest and the lower ribs. This "zoniform" or "zoster-like" sclerodermia (which Sir Jonathan Hutchinson had termed "morphœa herpetiformis") commenced at about the age of 12, reached its maximum development in about six years (when the patient was aged 18), and since then to the present time had remained stationary, or had undergone involution changes.

Urticaria Pigmentosa.

By G. Norman Meachen, M.D.

The patient was a male infant, aged 2. The condition first began at the age of 2 months as a "gum-rash," but the spots never disappeared. At the present time the eruption was profuse, widely distributed over the trunk, face, and scalp, and was mainly of the macular type with a few papules. Some degree of factitious urticaria was present, and the mother stated that some of the lesions were "still coming out." The child had never been vaccinated, as this had been considered detrimental to its health.

Dr. Stowers said the majority of such severe cases improved in the course of years, but the eruption was essentially of chronic nature, and it was not improbable that the lesions in this instance would persist into adult life. He reminded the Fellows of the valuable paper contributed by the late Dr. Sangster and published in the Pathological Society's Transactions, with illustrations.

Case for Diagnosis.

By G. Norman Meachen, M.D.

The patient was a man, aged 45, a clerk, who had sought advice at the Prince of Wales's Hospital at the end of January of the present year for a "pink discoloration of the roots of the nails" of a fortnight's duration. The redness soon deepened, and the fingers felt sore and itched, after the manner of chilblains. At the same time small red spots appeared upon the back of the terminal phalanx of the left index- and little finger. The fingers were said to "go dead" occasionally and to throb when the hands were held down. The nails were unaffected. Some ulnar deviation was present upon the right side. There was no history of rheumatism or other severe bodily illness. No central atrophy could be detected in the lesions upon the dorsum of the phalanges. At the time of the meeting the condition had improved spontaneously, and the hands were not markedly cold. The pulse was not of high tension.

Case of Folliclis in a Woman, aged 23.

By J. M. H. MacLeod, M.D.

The patient was a tall, rather delicate-looking young woman, a dressmaker by occupation. There was a definite family history of tuberculosis, and the patient herself had tuberculous adenitis with swollen glands on the right side of the neck, and the scars resulting from two operations for tuberculous glands. On the hands and feet there were typical lesions of folliclis consisting of indolent red papules or nodules, slight ulcerations, and numerous white atrophic scars. She had suffered from folliclis for the last five years, the lesions invariably coming out when the cold weather set in in winter, and disappearing in the spring. The object of showing the case was to emphasize the relation of the affection to tuberculosis, for in this case, in addition to the typical folliclis lesions on the extremities, there was a number of brownish papular lesions on the face which underwent necrosis and left a scar. On pressing them with a diascope, a brown stain persisted, indicating a marked cellular infiltration. These lesions suggested a transition between the ordinary folliclis lesions and small foci of lupus vulgaris.

Dr. Whitfield said he had these lesions inoculated into guinea-pigs, but had always got negative results. One observer, however, had inoculated a series and got a high percentage of deaths from tuberculosis among the guinea-pigs. He did not think there was any doubt as to their being tuberculous. Apparently the test must be made as soon as the lump appeared in the skin. The same was true of Bazin's disease.

Case of Favus of the Erythemato-squamous Type.

By J. M. H. MacLeod, M.D.

The patient was a little girl, aged 5, and the favus lesions were present in the form of three patches on the neck. The largest patch was oval in shape and about 1½ in. in its long diameter, was slightly raised, red and scaly. In the centre it was partially involuted and presented, when first seen, a number of pinhead-sized typical favus scutula. The border was studded with minute, deep-seated vesico-pustules. A baby brother of the patient was also affected and had a roundish patch of favus of the herpetic type on the abdomen. Cultures were made from the first case on maltose agar, and grew the white downy culture of *Achorion quinckeanum* of mouse favus.

DISCUSSION.

Dr. STOWERS said that twenty years ago favus was common in London in the East End; he had seen a large number of cases there. It was now very rare, especially in the West of London. Chronic cases occasionally migrated from one hospital to another, giving the impression that the disease was less rare than it is.

Dr. SEQUEIRA said the Favus School, started by the County Council, did much to stamp out the disease in London, and apparently the authorities responsible for the admission of immigrants were now careful to exclude cases of favus. The Favus School had been closed owing to the paucity of cases, but he still occasionally saw cases at the London Hospital.

Alopecia Areata and Tinea Tonsurans.

By E. G. GRAHAM LITTLE, M.D.

THE child had not had alopecia until the ringworm developed. That order of events was common. The alopecia usually lasted for six months, and then the hair grew again. Dr. Little had not known such a case remain permanently bald. He thought the alopecia could arise without there being any definite inflammatory disturbance.

DISCUSSION.

Dr. STOWERS said he published a case some years ago in which the two disorders occurred simultaneously on the scalp of a patient, and eventually coalesced. It was later referred to by Dr. Alder Smith in his treatise on ringworm.

Mr. G. F. HENTSCH said it had been found that 33 per cent. of cases of alopecia had previously had ringworm.

Dermatological Section.

April 17, 1913.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case of Unusual Papulo-necrotic Tuberculide.

By A. WHITFIELD, M.D.

THE patient was a rather stout and quite strong-looking woman, aged 50. She had been seen first by Dr. Whitfield in March, 1912, when he had made the provisional diagnosis of late syphilide, and at the same time a post-graduate working with him had offered the diagnosis of lichen planus. Wassermann's reaction had been sought for on several occasions, but was always absent. In spite of this the patient was put upon a short course of antispecific treatment without modification of the eruption. Some months after her first appearance the patient complained of a swelling in the right side of the neck and it was then found that she had an acutely inflamed and tender gland. A surgical colleague was consulted about this with the special view to the possibility of its being tubercular, and gave the opinion that it was most probably not tubercular, but secondary to carious teeth. The question of a tuberculide, which had already been entertained, was now reopened, and although clinical examination had revealed no evidence of visceral tuberculosis, the fact was now elicited that twenty years before the patient had had an abscess at the bottom of the spine, due to a fall, which had discharged for one or two years and then had completely healed. No scar could be found to indicate the position of the abscess, and it was thought that it might have been a fistula.

As the eruption remained unaltered for so many months the idea of its being a tuberculide gained ground, and finally the patient was taken into the ward for further investigation. A small dose of old tuberculin was injected hypodermically and one of the lesions was excised. There was a distinct rise of temperature within twenty-four hours of the injection and a strong local reaction at the site of injection. The gland in the neck tumefied slightly and became tender, but the eruption showed no change. The result of the reaction was therefore somewhat equivocal, as it proved that the patient was tubercular, but did not prove that the eruption was also tubercular. On examination of the excised lesion, however, it was found that the change was a perifollicular granuloma which had undergone central caseation. No ordinary leucocytic suppuration was present, and although giant cells were few the general arrangement was very suspicious of a tubercular nodule. A large number of sections were therefore stained by the Ziehl-Neelsen method and the tubercle bacillus was demonstrated in one section, lying almost at the edge of the caseated area in close contact with and probably phagocytosed by a large mononuclear cell. (This specimen was shown at the Meeting.)

On exhibition the following condition (which had remained practically unchanged for a year) was observed: The eruption was limited to the sacral region, the buttocks, and the outer sides of the thighs for a small area immediately posterior to the great trochanters. It occurred in what might be described as herpetiform groups with outlying elements, and in one or two places the elements were arranged in the form of a ring. The individual lesion consisted of a small dome-shaped, bluish papule, which, originally rather deeply seated in the corium, slowly rose to the surface and either developed into a very indolent pustule or flattened down to a flat, brownish, shiny papule, and finally disappeared, leaving behind a minute atrophic cicatrix. No "apple jelly" formation was seen on diascopic examination. All these stages were to be observed at the time of exhibition, and it was very striking how great was the resemblance to both lichen planus and a late papular syphilide.

An unusual symptom of the eruption was that the lesions were both painful and very itchy.

Dr. WHITFIELD said that the case was of some interest and importance, for more than one reason. He believed that the senior members would agree with him when he said that it was acknowledged in the old Dermatological Society of London that there were tuberculides that could not be distinguished clinically from syphilides, and that pathological and bacteriological examinations were necessary to make the distinction. Fortunately in this case the chain of evidence was very complete. The most important point, however, was the actual finding of an undoubted tubercle bacillus in situ. He had always fought stoutly against the theory that these granulomatous lesions were toxic rather than bacillary in nature. He did not deny that a "toxi-tuberculide" might exist, but he thought if it did it must partake rather of the nature of an erythema than of a mass of granulation tissue with giant cells. Lastly, the actual finding of the bacillus was a matter of interest on account of its rarity. He was aware that it had been found by Jacobi in lichen scrofulosorum, by Ormsby and MacLeod in a tubercular gumma of a baby, and by a very few other observers in other lesions, but the positive findings were few and far between. He was convinced that all these lesions were due to the presence of bacilli, alive or dead, and he hoped that the expression toxi-tuberculide as applied to these lesions would be dropped.

Case of Neurotic Excoriations.

By HALDIN DAVIS, F.R.C.S.

THE patient, a domestic servant ("mother's help"), aged 25, exhibited on the face and the backs of the hands typical "neurotic excoriations"; shallow, angular abrasions and sores, more or less parallel with one another, while in some places were areas of erythema obviously caused by rubbing and scratching with the finger-nail. About a month previously she had a similar attack, which had cleared up entirely on the patient going for a holiday. An interesting point in this case was the relation which it bore to the working of the National Insurance Act. Although the patient only received 7s. 6d. a week sick benefit, nevertheless the exhibitor considered that this sum very likely weighed with her if she desired to have a holiday.

DISCUSSION.

Dr. SEQUEIRA said he had recently seen in conjunction with Dr. Mackwood a young woman, a domestic servant, who had several times developed an eruption on the face and forearms. When he first saw the patient there was no doubt, from the character and especially from the outline of the lesions, that the disease had been self-produced. After taking the patient into a cottage hospital, watching her, and getting the matron to examine her clothing, Dr. Mackwood elicited a confession that the irritant used was mustard. The girl was an insured person, and, as she was also a member of a club, she drew 19s. 6d. a week when she was sick, and her wages as a domestic were £22 a year. There was no prosecution in this case, but the patient did not get her insurance money.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) remarked that it was often difficult to know what to say in such cases, especially in private—i.e., how far to go in declaring to what the condition was due.

Dr. GRAHAM LITTLE took this opportunity of mentioning that the boy he had shown two meetings ago with ulceration of the left leg which it had been suggested was due to artefact, had completely recovered under treatment with simple occlusive dressings, so that the father said that for four years the leg had not been so well as at present. The boy had been under careful observation in the wards at St. Mary's Hospital: no attempt had been made by him to dislodge the bandages, which had been marked so as to divulge any such attempt had it been made.

Case of Mycosis Fungoides.

By HALDIN DAVIS, F.R.C.S.

THE patient, a woman, aged 56, had suffered from a universal scaly dermatitis for about two and a half years. The whole surface of the body was implicated, including the scalp, which had become almost totally bald. The nutrition of the nails was not disturbed. The patient complained of a certain amount of pruritus, but this had never been particularly intense. In addition to the dermatitis the skin on the face presented a peculiar soft infiltration, darker in colour than the rest of the body. This infiltration began on the left side of the face, but had within the last few months extended to the right side. It was not general, but formed a kind of festooned pattern on the cheeks and also behind the ears. There was also an infiltrated patch on the right calf, and there was beginning at the time of exhibition a patch on the right upper arm. Some warts on the anus had also recently appeared. The mucous membranes presented no abnormality. The exhibitor regarded the case as one of mycosis fungoides just passing from the pre-mycotic stage into the mycotic stage of the disease.

DISCUSSION.

Dr. PERNET considered the case was one of mycosis fungoides. The woman was on the road to a "femme rouge."

Dr. GRAHAM LITTLE suggested the diagnosis of parakeratosis variegata. The blotchy eruption on the abdomen most nearly resembled that disease. The eruption on the face, which was clinically of a totally different character, was probably of different causation.

Dr. MACLEOD thought the face condition suggested a syphilide, and considered it possible that the affection on the face belonged to a different category from that on the body.

Dr. ADAMSON was inclined to regard the case as mycosis fungoides, but thought that lupus erythematosus ought to be considered. The lesions on the face were very like lupus erythematosus in their character and distribution, and the loss of hair would fit in with acute lupus erythematosus, transient alopecia without scarring being a not uncommon occurrence in acute lupus erythematosus.

Dr. PRINGLE said Dr. Adamson's suggestion was at the back of his own mind; it seemed compatible with generalized lupus erythematosus. Possibly that disease and syphilis co-existed in the patient.

Case of Lichen Planus.

By E. G. GRAHAM LITTLE, M.D.

THE patient was a man, a medical agent who was familiar with medical terms, and who gave a very positive statement that twenty-one years ago he was under Dr. Radcliffe Crocker's care, and that Dr. Crocker had then diagnosed psoriasis. The patient thought the present eruption resembled the earlier one in appearance and distribution. He had been free in the interval of any skin eruption until a few weeks ago, when the present condition had developed. He had now quite typical lichen planus of the trunk, limbs and mucous membrane of the mouth. The distribution was rather in the psoriasis area—the back of the elbow and front of the knee being especially involved, but there was no doubt now of the diagnosis of lichen planus. If, as seemed probable, the earlier disease had been psoriasis, it was interesting that there had been apparently complete cessation of that disease and that a totally different disease should have developed in much the same area as had been previously affected.

DISCUSSION.

Dr. STOWERS pointed out that the patient's statement that the late Dr. Crocker had had a coloured drawing made of the interior of the mouth when he saw the patient might be taken as presumptive evidence that he regarded the case as one of lichen planus and not psoriasis.

Dr. PRINGLE thought it quite possible that the man had psoriasis twenty years ago, but he undoubtedly now had lichen planus. He had certainly seen some examples of the co-existence of the two diseases in the same individual. One such case he had had under observation for twenty years, in which the psoriasis element prevailed at times, the lichen element at others. It had been seen by the late Dr. Radcliffe Crocker, who called it "lichen psoriasis."

Multiple Telangiectases.

By J. H. Sequeira, M.D.

The patient, a married woman, aged 55, was admitted to the London Hospital on April 14, 1913. During the past five or six years red spots have appeared upon the face and fingers, and occasionally there has been hæmorrhage from some of the spots. She has six children alive and in good health, and one of her daughters suffers from occasional epistaxis. No other relative has had any similar affection. The patient has usually enjoyed good health. Fourteen years ago she had bronchitis and was anæmic for some months. The catamenia ceased ten years ago; before that the periods were regular, but the loss was excessive. The patient has also suffered from varicose veins for the past ten years.

On April 4 the patient first came to the receiving room at the hospital because a spot on her left index-finger started bleeding and she was unable to control the hæmorrhage. This bleeding recurred several times, and on April 14 she was admitted into the ward. On admission there were many telangiectases on both cheeks. The dilated vessels were not grouped in any particular pattern. In addition to the telangiectases there were many punctate red spots on the cheeks, chin, and left eyebrow. On the dorsal aspects of the fingers and thumbs of both hands and also on the palmar surface of the fingers there were numerous punctate lesions of similar character. The largest of these were slightly raised above the surface of the surrounding skin and the size of a large pin's head. On the tongue, and the mucous membrane of the lower lip, and also on the hard palate, there were numerous punctate lesions, and a large telangiectatic vessel was present upon the uvula. The nasopharynx, so far as it could be observed with the laryngoscope, and the interior of the larynx were normal in appearance. On the lower internal surface of the left labium majus there was a telangiectatic vessel rather suggestive of a spider nævus. About the anus there were several external piles.

There was no definite history of melæna. Nearly every morning for several years the patient had suffered from slight bleeding from the nose. The spots on the left index-finger had bled spontaneously several times during the last three or four years, and also some lesions on the right index- and little-fingers, but the hæmorrhage had always ceased on previous occasions on the parts being bound up. She also stated that there had been slight bleeding from the tongue occasionally.

The patient was somewhat stout and anæmic. The heart's apex was not palpable, but the heart's sounds were best heard in the fifth left intercostal space just outside the nipple line. At this spot a hæmic systolic murmur followed the first sound. The pulse was regular, 72 per minute. The blood-pressure, measured by the Riva-Rocci apparatus, measured 180 mm. The lungs showed no abnormality beyond a moderate degree of emphysema with a little bronchitis. The abdomen was large and flaccid; the edge of the liver and spleen were not palpable. The area of hepatic dullness did not extend below the costal margin. There was no ascites, and the legs did not pit on pressure. The subcutaneous veins of the chest and abdominal wall were rather well marked, there was no grouping of vessels about the umbilicus, but in the middle line of the back just above the level of the scapular spines there was a fan-shaped arrangement of dilated superficial vessels. The subcutaneous vessels round the ankles and on the dorsum of the feet were also well marked. The urine was acid, specific gravity 1025; there was neither albumin nor sugar present. The coagulation time of the blood was three minutes. A blood count gave the following figures: Red corpuscles, 4,300,000 per cubic millimetre; hæmoglobin, 50 per cent.; colour index, 0·6; white corpuscles, 5,000 per cubic millimetre; polynuclear neutrophiles, 55 per cent.; eosinophiles, 0·5; small lymphocytes, 15·5; large lymphocytes, 19; large hyaline, 9; granular basophiles, 1. On ophthalmoscopic examination the retinal vessels were found to be thickened, but there were no hæmorrhages.

The causation of these cases of multiple telangiectases is very obscure. In this instance the high blood-pressure and the thickening of the retinal vessels appear to be the only phenomena suggesting chronic renal disease. There is no evidence of hepatic disease, or of any chronic malignant affection.

[Dr. Sequeira is much indebted to Dr. W. J. Oliver for his assistance in working out the details of the case.]

DISCUSSION.

Dr. PRINGLE said he had seen one case of this kind in which multiple telangiectases of the skin were associated with a large dilated blood-vessel on the epiglottis, which caused repeated and very severe hæmoptyses. Her lungs were many times examined, under the idea that she had phthisis.

Dr. F. PARKES WEBER said that some time ago he described a family group of these cases.[1] In the patient now shown by Dr. Sequeira there were red points of telangiectasis under one finger-nail, and that was a characteristic feature of this type of case. He did not believe the cause of the condition was known, but kidney disease and arterio-sclerosis might in some cases act as predisposing conditions. This woman had not the prominent "stigmata" of the "nævus araneus" or "spider angioma" class, which some of the patients had, especially on their faces. Patients with cirrhosis of the liver sometimes presented similar and still more striking spider angiomata (especially on their faces and hands), in which distinct arterial pulsation could be felt; but such hepatic cirrhosis cases should be distinguished from cases like the one under discussion. Calcium therapy had been tried by Sir William Osler in certain of his cases of bleeding telangiectases.

Multiple Subcutaneous Abscesses in a Young Girl.

By J. H. SEQUEIRA, M.D.

THE patient was brought to the Meeting as a case for diagnosis. She was aged 17, and was engaged in a pickle factory. Her occupation necessitated her standing the greater part of the day. She had never been out of England and only once out of London for a fortnight in Gloucestershire. The family history was negative. The patient complained of being easily tired and short of breath. The bowels were usually confined and she had to take salts frequently. The catamenia had been regular up to December, 1912, when she first noticed any trouble with her legs.

The patient was admitted to the London Hospital, on January 31, 1913. Three weeks before Christmas a "red lump" appeared on the right calf. This was very sore and tender. A little later another bluish swelling appeared on the same leg. On January 23 she attended in the Surgical Out-patient Department, where two abscesses were opened, and a considerable quantity of pus was removed. She was then transferred to the Skin Department and admitted to the ward. On admission the patient was rather anæmic, but there were no indications of visceral disease. The lymphatic glands appeared to be normal. On the right leg there was a deep ulcer over the middle of the tibia, this was about the size of a sixpence and was surrounded by a red infiltration. On the calf there was a large ulcer the size of a two-shilling piece, over $\frac{1}{2}$ in. deep, and with a shelving border. The ulcers were painful and tender. A thin pus exuded from the ulcers. These were the lesions which had been opened. On the calf of the leg and just above the knee behind there were four red hyperaemic swellings varying in size from a florin to a sixpenny piece. These were very painful and did not appear to be breaking down. On the left leg there were two ulcerated lesions which have broken down spontaneously, they were about the size of a shilling. There were also some smaller indurated swellings in the calf and on the inner side of the thigh.

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[1] F. P. Weber, "Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes associated with Recurring Hæmorrhages," *Lancet*, 1907, ii, p. 160.

The patient was kept at rest in bed, and was put on a generous diet. Her general condition improved remarkably, and she put on weight. From that time until she was shown at the Meeting numerous fresh lesions have appeared; some of these have broken down spontaneously, sometimes discharging through several small openings. The Wassermann reaction was negative, and the reaction to Moro's tuberculin test was also negative.

Sporotrichosis was suspected and some of the pus from an unbroken lesion was submitted to Dr. Fildes, who failed to grow any organism. Dr. Adamson kindly saw the patient, and also took some of the pus for examination, and he also failed to grow an organism, though he agreed with the exhibitor that the condition closely resembled sporotrichosis. Agglutination tests were also made in the Bacteriological Laboratory at the London Hospital, but the result was negative. The obvious diagnosis in this was Bazin's erythema induratum, but the character of the lesions, their acute onset, and the fact that fresh foci developed while the patient was at rest in bed, and that her general condition as indicated by increase in weight was steadily improving, were in the exhibitor's opinion sufficiently unusual to render the case worthy of being brought before the Section.

DISCUSSION.

Dr. ADAMSON said that when he saw the case on a previous occasion he felt sure that it was one of sporotrichosis, for the lesions were so exactly similar to those of the case of sporotrichosis which he had had under his care in the hospital and which he had exhibited at a previous meeting. The manner in which the gummata broke through the skin by cribriform openings was exactly that of his own case. He had, however, been unable to grow Sporotrichum although he had inoculated a dozen or more tubes on various culture media, including glucose-agar, and had made the inoculations in the approved manner of spreading a large quantity of the pus over the surface of the medium. The failure to find the fungus certainly made the diagnosis of sporotrichosis doubtful, though he was still inclined to regard it as such. He thought, however, that a tuberculin test by subcutaneous injection should be made.

The President considered that it was a case of persistent Bazin's disease, in spite of the fact that fresh lesions came out while the patient was lying in bed in the hospital.

Case for Diagnosis.

By G. Norman Meachen, M.D.

The patient was a rather pale girl, aged 14. With the exception of an attack of chorea at the age of 4, when she attended the Great Ormond Street Hospital, she has had no other illness. The mother states that six years ago a "small pimple came by itself" in the centre of the right calf of the leg. This has never gone away, but has slowly spread and has remained raised above the surface of the skin. She was only seen by the exhibitor on April 16, at the Prince of Wales's Hospital, when she presented a lesion the size of a two-shilling piece in the middle of the calf, bluish-red, elevated above the surface, and firm in consistence. Surrounding this was a large area of eczematous dermatitis, the result, the mother said, of a kick from a boy three weeks previously. She also showed the remains of a catarrhal herpes upon the left upper lip. Two teeth were badly decayed, and the thyroid gland was distinctly full. Her parents, four brothers and four sisters, were all healthy. There was no history of tuberculosis in the family, nor of any abrasion of the surface at the site of the lesion.

DISCUSSION.

Dr. A. D. Heath said he regarded it as a granuloma, probably of streptococcal or staphylococcal origin.

Dr. Pringle thought that the condition was probably a pus infection, but suggested that the result of treatment would help in arriving at the diagnosis.

Case of Persistent Erythematous Eruption.

By A. M. H. GRAY, M.D.

THE patient was a woman, aged 37, who was in good health till nine months ago. At the age of 13 she had rheumatic fever. She had seven brothers and sisters, three of whom in addition to herself had had rheumatic fever. She had had two children, of whom one was alive and well, the other had died of diarrhœa in infancy; no miscarriages. The present illness commenced nine months ago with two small blisters on the outer side of the left ankle; these spread and formed patches on the outer side of the foot and gradually extended on to the front of the shin. Similar patches appeared, one on the front of each knee and one on each elbow about the same time; a little later the outer side of the right foot and front of the right ankle became similarly affected; six months ago patches appeared on the back of the left hand and wrist and on the calves, and three months ago on the back of the right hand. Quite recently, about a week ago, small spots had begun to appear on the backs of the forearms and arms, front of the legs and outer side of the thighs. The patches were painful and varied from day to day, though they had never disappeared.

The patient had been in hospital two days and the lesions had somewhat subsided, but when seen two days ago they had the following appearance: The newest lesions, seen on the backs of the arms, forearms, front of the shins and outer side of the thighs, were lentil- to pea-sized perifollicular papules of whitish-yellow colour, tender to the touch; in other situations the lesions were larger, attaining the size of a threepenny piece and becoming surrounded by an inflammatory zone; some of them retained their white urticarial appearance, while into others, notably those on the calves, hæmorrhage had occurred and a few had developed bullæ. The inflammatory zone around the lesions on the calves was very wide and purpuric in character, and one of the lesions had been converted into a septic ulcer. The largest lesions, seen best on the front of the left ankle and shin, had been converted into rings with a hard, raised border not more than 0·5 cm. thick, often incomplete, of an ivory-white colour, surrounded by an inflammatory zone and enclosing a smooth, deeply pigmented area. The patches at points of pressure—e.g., over the knees, elbows, and heels—had developed into warty growths not unlike those seen in lupus verrucosus. The lesions were tender to the touch. There was no scarring. The palms, soles, face and trunk had completely escaped, also the flexor aspects of the limbs with the exception of the calves. The patient was pale, but otherwise her general condition was good. Her teeth were in bad condition, and she had pyorrhœa. Urine normal. Periods regular; no vaginal discharge. Wassermann and von Pirquet's reactions were negative. The patient had taken no drugs previous to the appearance of the eruption, but during the last month had been treated with mercury and potassium iodide on the supposition that the lesions were syphilitic. A biopsy had been made on the previous day, but a section had not yet been prepared.

The exhibitor had not been able to group the case, but thought that it most nearly resembled those cases recently described by Favera and Piccardi who had grouped their cases with Crocker's "erythema elevatum diutinum"; the pictures, however, of Crocker's and Bury's cases did not resemble the present case very closely.

DISCUSSION.

Dr. ADAMSON considered it to be erythema multiforme, on account of the distribution and general appearance. It was much like a case shown by Dr. Graham Little, in which the point was discussed whether it was extensive lupus erythematosus or erythema iris.[1] The fact that it got worse when potassium iodide was given was in accordance with the view expressed at a recent meeting by Dr. Pringle that iodide of potassium did harm in such cases.

Dr. PRINGLE favoured the diagnosis of erythema multiforme, and pointed out that the preliminary stage of pallor and vascular spasm had been observed.

Dr. PERNET pointed out the erythema iris type of some of the lesions on the back of the hands.

Erythematous Lesions of the Hands in a Case of Lupus Vulgaris.

By A. M. H. GRAY, M.D.

THE patient, a man, aged 46, had suffered from tuberculous abscesses in the neck from the age of 6 to 16. In March, 1910, he developed lupus vulgaris of the nose, which was treated in the first instance with old tuberculin, but as he developed cough and signs in the chest this was discontinued and X-ray treatment was adopted. He was well for about a year, but at the beginning of 1912 the disease recurred and he did not obtain advice till November of that year, when the disease had advanced considerably. Since that date X-rays and local caustic applications had been used and he had been taking cod-liver oil internally but no other drugs. In March, 1913, his hands became painful and numerous tender, lentil-sized papules, slightly indurated, appeared on the fingers of both hands. These disappeared in about a month without ulceration, vesiculation, or scarring. About a fortnight ago another crop of similar lesions appeared. Patient stated that he had had a similar attack when aged 10. He had also had rheumatic fever when aged 13, but had no endocarditis.

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[1] *Brit. Journ. Derm.*, 1912, xxiv, pp. 119, 270.

The exhibitor considered the lesions to be tuberculides when they first appeared but owing to the absence of ulceration and scarring he had come to the conclusion that they were of the nature of a toxic erythema. Microscopic section showed considerable œdema and leucocytic infiltration into the lower part of the prickle-cell layer and papillary layer of the corium and also to a less degree around the deeper vessels and coil glands.

Case of Rodent Ulcer treated with Arsenic Paste.

By A. M. H. Gray, M.D.

THE patient was a man, aged 38, who had suffered from a rodent ulcer for eighteen years. It began as a pimple over the left zygoma and spread gradually downwards. He neglected it for several years, but some six years ago he was treated by the late Dr. Crocker with X-rays and thorium ionization and the ulcer healed but subsequently recurred. He was then again treated with X-rays but without success, and subsequently underwent treatment at the Radium Institute for nine months, and though the ulcer did not spread it refused to heal. When first seen by the exhibitor in July, 1912, the ulcer extended upwards to a line drawn from the eyebrow to the top of the ear; downwards to the ramus of the jaw; in front to the margin of the orbit and a line drawn straight downwards to the ramus of the jaw; and behind to the mastoid process. The tragus and lobule of the ear had been destroyed and the meatus opened up. The base of the ulcer was covered by a dirty slough and the malar bone and zygoma were exposed. Under an anæsthetic the ulcer was scraped and arsenic paste applied; a second application was made ten days later. After both these applications there was a tendency for the ulceration to extend, so dressings of 2 per cent. formalin were subsequently used, alternated with starch poultices whenever a thick slough formed. Under this treatment further spread ceased and healthy granulations appeared; subsequently a sequestrum consisting of the outer table of the malar bone and the whole thickness of the zygoma separated; the ulcer was Thiersch grafted and healed well. Unfortunately a small nodule has since appeared just beyond the original ulcer in the eyebrow; this has been treated with radium.

The case is of interest, firstly, because the patient was only aged 20 when the ulcer first appeared; secondly, it shows, as Dr. Norman Walker has pointed out, that the old method of caustic applications are still useful in refractory cases; and thirdly, that good cosmetic results can be obtained in these cases.

The PRESIDENT said that, when he was in Vienna, the paste was the systematic treatment for chronic ulcerations of various kinds. In the great majority of cases the result was extraordinarily satisfactory, the chief drawback being the intense pain.

Fully-grown Culture of Achorion Quinckeanum.

By J. M. H. MacLeod, M.D.

DR. MACLEOD showed a culture of mouse favus (*Achorion Quinckeanum*) from the case of favus he exhibited at the previous meeting of the Section.[1]

DR. A. D. HEATH said he had had two examples of favus Quinckeanus. One was on the abdominal wall of a child aged between 3 and 4. In both cases there was a patch of an erythemato-squamous eruption, and one or two very small yellow cups could be seen on each patch.

Congenital Hyperkeratosis of the Hands and Feet.

By H. G. Adamson, M.D.

THE patient was a female infant, aged 4 months. The hands and feet had the appearance of being clothed in black gloves and socks, due to a thickening of the horny epidermis of the palms and soles and

[1] *Proceedings*, p. 121.

backs of the hands and feet. The epidermis was almost black. It formed a kind of dry, wrinkled, leather-like coating which was fissured and separated in parts to expose the smooth, pinkish skin beneath. There was no verrucose appearance. There were similar patches at the tip of each elbow, but no moles or other "birth-marks" elsewhere. The mother attributed the condition to a fright during pregnancy by a dog with "horrible black paws," but she admitted that when the child was born the hands and feet were sodden and white, and only became black some weeks later. There were no other cases in the family.

DISCUSSION.

Dr. ADAMSON thought that the condition could be improved, and the hands and feet kept tolerably smooth by the application of salicylic acid ointment and occasional soaking in warm water. In accordance with the President's suggestion, he would add a little sulphur to the salicylic acid.

Dr. PERNET pointed out that there was a reference to a case of the kind in White's "History of Selborne," the subject being a boy. It was referred to as a leprosy by Gilbert White.

Case for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

THE patient was an elderly woman under the care of Mr. Warren Low, for an ulcer on the cheek, which was almost certainly a rodent ulcer. It had come out on the site of a deep cut from an accident to the cheek six years ago, and had never completely healed. But in addition to this lesion there was a very large number of circular scars chiefly distributed on the dorsum of the feet, the legs, the back of the hands, and the forearms; one or two lesions were present on the abdomen. The history given was that about four years ago there had been a series of scabbed sores in the position of the present scars; these had come out successively and slowly healed. The Wassermann reaction had been taken twice, and each time with a negative result. It was, of course, exceedingly difficult to make a retrospective diagnosis, and the case had been brought up with the view of hearing suggestions.

Multiple "Cold" Subcutaneous Abscesses in a Female Infant aged 11 Months.

By E. G. Graham Little, M.D.

The father of the child was now invalided at home with pulmonary tuberculosis. The swellings were tense and deep-seated, and on incision a thick but fluid pus was evacuated. For the most part the skin over the tumour was normal, but in exceptional instances there was some blueness or redness. They were very numerous, and distributed on the dorsum of the left hand (where was the largest tumour, the size of a small plover's egg), on the face near the right eye, on the buttocks, legs, abdomen, and trunk. Some pus from one of the tumours had been aspirated with a sterile needle and planted on Sabouraud's media with a view to testing the possibility of sporotrichosis being responsible for the swellings, but fourteen days had elapsed without growth on this medium. The case was of a type which is usually described as "tuberculous gummata," but on insufficient grounds. Untreated, the tumours remained indefinitely without absorption and without evacuation, the skin usually remaining unbroken over them.

Demonstrations on the Subject of Malignant and Doubtfully Malignant Tumours of the Skin.

(I) Dr. Whitfield showed eleven lantern slides illustrating the genesis of the soft mole, nævo-carcinoma, and Paget's disease of the skin. The first five photomicrographs were taken from a single section of a tumour from a child's head. The case was a rare one under the care of Mr. Mower White. The whole of the top of the head had been covered since birth with hemispherical tumours of different sizes which had lately begun to grow so that they varied in size from that of a large pea to that of a small plum. The hair had not grown over the site of the tumours, so that the case resembled to some extent those described under the name of withering sarcoma of the scalp. Mr. White had removed the whole area and covered in the raw surface with skin-grafts. The result was perfectly satisfactory, as the child was left with a smooth, healthy scar, and there was no greater area of baldness than had existed before. The slides showed first the acantholysis or loss of prickles at the edge of the growth; in a further stage the cells were seen lying in a sort of lymph space in the lowest layer of the epidermis; further in still they could be seen dropping off into the corium; further still they could be seen lying deep in the corium but still arranged in clumps as they originated from the epidermis; and lastly in the oldest part of the mole they formed lines in between the bundles of fibrous tissue, and this was the stage which had led to the idea that the tumours were lymphangiomatous or endotheliomatous in nature. In this case, however, owing to the activity of growth all stages could be accurately traced from the beginning to the end.

The second series of slides were taken from the case of a woman who had had a mole all her life beneath the breast. This had begun to enlarge rapidly and had been removed by a doctor, but recurrence had occurred in the scar. When Dr. Whitfield saw her there was a large elliptical patch of flat, pigmented infiltration in the sub-mammary fold. There were no enlarged glands and he had removed the growth. Three years after there was no recurrence. The slides showed almost exactly the same process as the previous set, except that there was in some parts pigmented and in others pigmentless downgrowth. The process was far easier to follow in the non-pigmented growth, but he had seen by his method of bleaching pigment that the process was identical in the two cases. A topographical low power specimen was also shown to indicate how superficial was the invasion, though the method of spread was typically malignant.

Lastly, Dr. Whitfield showed a section of early Paget's disease to show how similar was the method of onset in this disease. In this case the acantholytic cells, however, were not limited to the basal epidermis, but occurred in the middle of the epidermis, and were even exfoliated in the horny layer so that one could find them as double contoured bodies (pseudo-psorosperms) by scraping the surface and examining in potash.

(II) Dr. J. H. SEQUEIRA showed by the epidiascope a series of photographs illustrating the usual sites and characteristic development of rodent ulcer of the face. He said that some years ago Mr. Lenthal Cheatle drew attention to certain peculiarities in the distribution of malignant disease, and suggested that neoplasms of malignant type usually developed in Head's maximal points. To determine whether this was the case, the speaker had large diagrams made, upon which he marked the site of origin of over 200 cases of rodent ulcer (*see* figure). These observations showed that in the bulk of the cases the disease started at the inner canthus and about the ala nasi. Some occurred at the outer canthus, a few on the lids, others in or about the ear, and others in different situations, the lips and chin being the least likely to be affected. One interesting point demonstrated by the chart was that a larger number of rodent ulcers developed on the right side of the face than on the left, and this appeared to give ground for the supposition that local irritation—e.g., scratching—had something to do with the development of the tumours, most people being right-handed and probably more prone to irritation of the right side of the face by the fingers. From a study of several hundreds of cases, Dr. Sequeira believed it was possible to group cases of rodent ulcer and to anticipate the direction in which they were likely to develop, an important point both in regard to prognosis and treatment. Illustrations of the development of rodent ulcers in a number of situations were shown, demonstrating the evolution of the process from the early stage to extensive destruction.

Orbital Group.—The early involvement of the lachrymal sac, and spread of the disease to the orbital cavity, ending in a huge excavation, were presented. In the advanced cases the upper nasal sinuses are exposed. Illustrations were also given of the somewhat rare variety of rodent which runs lengthways along the upper or the lower lid. Rodents at the outer canthus usually develop downwards and early involve bone.

Nasal Group.—The common form starting in the angle between the ala and the naso-labial sulcus tends to destroy first the ala and later a large triangular area extending down to the muco-cutaneous margin of the upper lip. A lateral nasal variety, in which the nasal bone becomes involved early and leading to perforation, was shown in different stages.

Frontal Group.—Here the ulcers are usually of the superficial, cicatrizing type. They often start just above the root of the nose and extend upwards in the frontal region.

Maxillary and Malar Group.—The ulceration usually develops transversely, forming an oblong excavation. The bone is involved early and huge tumours may invade the antrum and push up the eyeball and depress the palate.

Auricular Group.—Rodents may start in the concha and then tend to eat out an irregular hole. If they begin at the upper part in front they usually extend on to the temporal region. Pre-auricular ulcers extend forward and a parotid fistula may form, and the facial nerve is often involved. Among the rodents starting behind the ear, photographs were shown of one that remained limited to the retro-auricular sulcus.

Upper Lip Group.—Rodent ulcer of the upper lip is uncommon, but there is a very troublesome form which starts somewhere between the nose and the free margin of the upper lip and if not treated early involves the whole of skin and tissues between the nose and the edge of the lip, ultimately destroying bone and exposing the roots of the teeth.

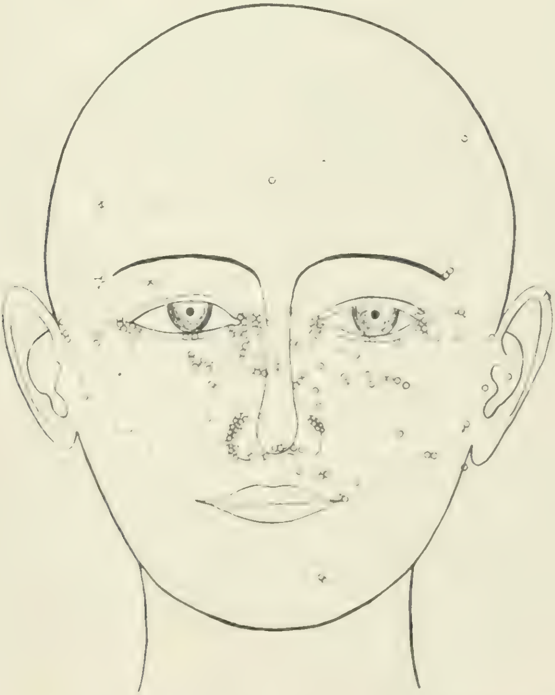

Diagram illustrating the sites of origin of 220 cases of rodent ulcer (basal-celled carcinoma) on the face.

Lower Lip Group.—In the experience of the exhibitor this type is even rarer than the upper lip variety. Here there is also a tendency to resist treatment, and in one case the lower jaw was eroded and the roots of the incisor and canine teeth were exposed.

Mental Group.—These cases usually occur to one or other side of the chin close to the mental foramen. They are troublesome to deal with as they early involve the bone.

In all the cases shown on the screen there was no doubt about the diagnosis of rodent ulcer, as shown by the character of the lesion, the slow development, the absence of glandular involvement even after many years, and in most instances by a microscopical examination.

Photographs of two remarkable cases of multiple rodent ulcer of the face were also exhibited.

The PRESIDENT said that if they were to start a clinical discussion on rodent ulcer it would occupy the entire evening. He would only like to say that he had taught for years—thirty-five at least—the enormous difference, from the point of view of treatment, between the superficial and the fairly advanced cases. At one time no particular attention was paid to rodent in its early stages; it was left to grow until a later stage before any active treatment was instituted. It was only in later years that rodents had been recognized as malignant growths before they involved the periosteum and bone. But such early recognition of their malignancy was a vital matter.

(III) Mr. J. E. R. McDONAGH showed under the microscope several slides illustrating epithelial growths; and also sections of skin and glands from cases of various leukæmic conditions. He said that he preferred to make no remarks upon them, owing to the short time at his disposal and the difficulty of showing the slides by means of the epidiascope.

(IV) Dr. HALDIN DAVIS showed some specimens from St. Bartholomew's Museum, and some slides. Among the former were some amputated fingers of two X-ray operators who had suffered from X-ray carcinoma. Another interesting specimen was an epithelioma which started in a tattoo mark, a part of the tattoo mark being still visible. After the removal of the primary growth the patient had glands removed from the axilla, and ultimately had a recurrence in an inoperable position and died. The slides shown by this speaker included photomicrographic views of two sections, both taken from the same patient but from different tumours. The one showed the microscopic structure of an epithelioma, taken from the back of the thigh of a patient, aged 55, and there was no doubt about the fact that it was an epithelioma, but an interesting point was that on the sole of this patient's foot there was another tumour of quite different structure, which under the microscope more nearly resembled a rodent ulcer than anything else. Finally he showed a case in which the section had been taken from a tumour appearing at the pinna of the ear. To all appearance it was an epithelioma, but quite soft; but under the microscope it appeared only as a suppurating papilloma, and he scraped it away under an anæsthetic. He scraped it again and it recurred, and there was yet another recurrence, but quite small and local. Finally, he excised it completely and cauterized the base with pure carbolic acid, and since then it had healed up completely.

DISCUSSION.

Dr. WHITFIELD referred to the great number of plasma cells in these malignant growths, and said that he had always regarded them as a defensive wall. They were found as a rule surrounding the epithelial downgrowths and the rest of the corium. Moreover, they were not found in the innocent moles. He had cut a good many moles, and except in the cases of those situated in such places as the axilla, where they were irritated by sweat and so forth, he had found no inflammatory action around them. The plasma cells in the malignant growth were evidence, he thought, as in the epithelial tumours, of an attempt at defence on the part of the patient's tissues. If he might add to his remarks on his slides, he would point out that in nævo-carcinoma they were all taught to believe that metastasis was early and fatal. But there was a class of nævo-carcinoma in which the metastasis was not early, and the case he had shown that evening was one of that class. The tumour was excised three years ago, and the patient was still quite well. The criterion appeared to be simply one of depth. The low power specimen he had projected on the screen revealed the extraordinary superficial growth. In one case the growth was excised by the same surgeon three times in seven years, and the third time he succeeding in removing the whole. The speaker believed that it was possible to judge the probable danger to life by microscopical examination. The best cases from the point of view of ultimate complete recovery were those in which there were broad sheets of cells lying horizontally in the corium.

The PRESIDENT asked whether surgery was necessarily the sole treatment for these cases.

Dr. WHITFIELD said that the cases did not yield to X-rays; he could not say about radium.

Dr. SEQUEIRA said that he had one case in which a flat pigmented patch—he supposed it would be called a pigmented mole—had entirely disappeared under radium. It resembled very much the type of case Dr. Whitfield had described. It would be worth while, he thought, to try radium. The pigment in this instance had entirely disappeared, and the scar was left. He was watching it very carefully, but up to the present it could be said that it had disappeared under radium.

Dr. GRAHAM LITTLE put in a plea for treatment of rodent ulcer by freezing with carbon dioxide snow. For nearly three years he had used this method, and was personally satisfied that the cures were as frequent and as permanent as with any other method, and he regarded it as the ideal treatment for the small and early rodent of the skin, where the deeper tissues were not involved. He regarded this treatment and ionization as safer than either radium or X-rays, for one could estimate the depth of the tissue acted upon, which was impossible with X-rays and radium. Advocates of the latter agents especially used arguments which were mutually destructive, for it was claimed that radium emanation penetrated much more deeply (more deeply than was, in fact, desirable in the great majority of cases), and yet did not harm the deeper tissues. The effect was miraculously operative when required and miraculously inert where it admittedly might do mischief.

Dr. WHITFIELD said that he would treat rodent ulcers with X-rays if they did well from the start, but if they did not yield rapidly he would have them widely and deeply excised. He fancied that, in some of the cases of which Dr. Sequeira had shown photographs, a moderate surgical operation would have saved the situation if it had been done when they were first seen. In rodent ulcer a rather free operation should be done. It was not a question of cosmetics, but frequently of the patient's life. A wide and efficient excision was generally satisfactory. Of the few small rodents that he had cut out, he had not seen a single one relapse. He thought that Dr. Gray's case, shown that day, proved that a very nice cosmetic result could be obtained in cases which had entirely resisted X-rays and radium.

Dr. A. M. H. GRAY did not consider that carbon dioxide was always suitable in early cases of rodent ulcer. He had recently had a case, sent to him by a surgeon, of a patient with a small warty growth on the left side of the bridge of the nose. He had applied snow to it for a minute, but ten days later the growth had increased very rapidly in size. It was at once excised and proved to be a squamous epithelioma.

Dr. R. A. BOLAM said that he had treated a fair number of rodent ulcer cases with snow, and had been grievously disappointed in the ultimate results. The relief to the patient was undoubtedly rapid and temporary satisfaction was usually expressed, but recurrence seemed much too frequent. X-rays and radium gave a much better prospect than carbon dioxide snow did, and were to be preferred in that the results were permanent in the majority of cases.

The PRESIDENT said that his experience with regard to freezing—and he had practised the method in several cases—was that one had to make a very careful selection of the type of rodent for the treatment. A slight thickening edge might very often be frozen successfully, but if there was the slightest depth in the growth he would much rather trust to radium or X-rays than to freezing.

Dermatological Section.

May 22, 1913.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case for Diagnosis.

By Sir MALCOLM MORRIS, K.C.V.O., and S. E. DORE, M.D.

MALE, aged 52. Family history: Father died at the age of 64; Mother aged 84, still living; seven brothers and sisters, all healthy. Father and one brother suffered slightly from eczema but no history of any other skin disease. Personal history: General health exceptionally good; has been accepted as a first-class life by several insurance companies.

History of present condition: In February, 1912, he noticed a small red spot on the left side of the face, below the cheek-bone. The patch gradually spread and ulcerated, but no particular attention was paid to it. In the following April a similar patch appeared on the right cheek, which he thought had been inoculated from the other by shaving. At the end of May a third lesion appeared on the bridge of the nose, which became raised, thickened, and ulcerated like the others. In June he consulted a doctor and a zinc ointment was applied. In July, during a holiday, the lesions improved without any treatment and, in fact, nearly disappeared, but after he returned at the end of July, several new patches appeared on the head and on the right shoulder, and these continued to increase in number and in activity. In December, 1912, his blood was tested for venereal disease with a negative result, but in spite of this an injection of salvarsan was given. After the injection his face swelled to twice its normal size within a week and there was acute œdema under the eyes, so that he was only able to see out of one eye, and soon after fresh lesions rapidly appeared on the back, chest, arms and legs. In January, 1913, he went into a hospital and was treated with a tar ointment and a lotion and a mixture. His condition remained stationary, and he then returned home where he continued the same treatment and also took hot sulphur baths. In April he underwent the "Rho Ray" treatment for seven days; this seemed to check the disease for a short time, but there was no permanent improvement. Since this time he has continued to use tar ointment, with the result that considerable irritation of the skin has been set up, but several of the lesions have disappeared.

Present condition: The lesions consisted of oval or circular, slightly elevated plaques, distributed abundantly over the trunk, limbs, face and scalp. They varied in size from that of a sixpenny piece to lesions as large as the palm of the hand, and there were also larger areas due to coalescence of the patches. The most characteristic lesions were of a pink or reddish-brown colour with marked "boss-like" central infiltration, gradually decreasing towards a clearly defined periphery. Some showed evidence of retrogression, one, particularly, having involuted for three-quarters of its extent, leaving a narrow semilunar patch. Another lesion showed a central patch with a concentric ring. A few showed a peripheral ring of scales with their free edges pointing towards the centre, but it was difficult to ascertain how much of the scaling and excoriation was due to the application of strong tar ointment. The face had a bloated appearance, caused by diffuse infiltration and œdema of the skin, and showed superficial ulceration and crust formation. The nose was bulbous owing to the presence of a prominent ulcerating lesion on the tip. The scalp presented a large scarred area on the vertex which was also slightly ulcerated and covered with crusts, and there were smaller infiltrations in other parts of the scalp.

Photographs of the case and microscopical sections will be published at a later date.

DISCUSSION.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) added that there was glandular enlargement in various parts, especially the groins; but the general physical condition of the patient was good. Except for a spot on the shoulder, all the ulceration appeared to be due to the strong tar application he had been using. The itching was only slight. His own opinion was that it was lymphosarcoma, in which case the outlook was not satisfactory. It was an interesting fact that following the one dose of salvarsan which he had there was great exacerbation of the condition. Still, there were apparently similar cases recorded in which arsenic had been beneficial. It was intended to make a biopsy, but he had only that day seen the case for the first time. There was thickening at the centre of the lesions, and withering at the periphery. One member thought mycosis fungoides was excluded because there had been no itching, but he had seen cases of characteristic tumours associated with that condition in which there was no itching, though he admitted such were rare. Hence the presence or absence of itching could not be held to settle the diagnosis. Mycosis fungoides without a preliminary skin lesion was very rare indeed. There had been no form of skin lesion in this case. These tumours began in the face, and they had gone on steadily increasing ever since.

Mr. McDONAGH suggested that the case was one of lymphodermia perniciosa. There was not much itching, and though the glandular enlargement was not at present great, he considered that as the disease progressed the glands would become greatly enlarged, especially the inguinal set. A portion of gland should be removed for microscopical examination, and also the margin of one of the lesions, as this would make a diagnosis certain. With regard to treatment, he recommended a trial of benzol internally, as he had seen marked improvement following its use in these chronic lymphocytic affections.

Dr. WILFRID FOX agreed that a biopsy would be useful, and referred to an apparently similar case in which the diagnosis of lymphodermia perniciosa was suggested; it proved, however, not to be so. But in that case the withering occurred at the centre, leaving a concave lesion. The patient died of sarcoma of the mediastinum three months after he was shown.

Dr. J. J. PRINGLE inclined to the opinion expressed by the President, with some reservation as to the use of the word "sarcoma." Cases of this class were described many years ago by Kaposi under the name of "multiple non-pigmented lympho-sarcomata." But the whole subject of the question of skin sarcoma needed revision. Microscopically, he did not know of any absolute distinctive criterion between skin granulomata and sarcomata. Mycosis fungoides of the Perrin type was a possible alternative diagnosis in the present case. In favour of the President's view was the complete absence of itching, and the fact that many of the tumours had withered spontaneously. The diagnosis might be much helped by the application of X-rays. If the tumours were to rapidly diminish under them, it would, he thought, tend to confirm the diagnosis of mycosis fungoides. A very similar case which he had seen was X-rayed by Dr. Whitfield, and the ensuing improvement had been amazingly rapid and satisfactory; he did not, however, know the ultimate result of the treatment.

Dr. PERNET considered the case was one of mycosis fungoides. In a case of mycosis fungoides d'embleé, a drawing of which he had shown before the Section,[1] the patient was given salvarsan. The patient went from bad to worse, however. He had seen cases in which there was practically no itching. It was the long-standing pre-mycotic conditions that were so pruritic. In mycosis fungoides lesions involuted spontaneously, even quite large tumours.

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[1] *Brit. Journ. Derm.*, 1912, xxiv, p. 318.

Dr. DORE said he remembered a case of multiple sarcoma of the skin which was also seen by Dr. Pringle and Dr. Whitfield, somewhat resembling the present one, except that there was some resemblance to a syphilide. He believed the patient improved on injections of soamin or orsudan.

Dr. MACCORMAC said that if sections of the condition were made, they should be stained for Altmann's granules. These granules were absent in sarcoma of the skin, but were present in mycosis fungoides.

Dr. JAMES GALLOWAY said he was inclined to agree with the diagnosis of mycosis fungoides. The absence of a prodromal dermatitis had been remarked on in this case, but he was not certain that, even if this were so, it definitely excluded the possibility of mycosis fungoides; and on examining the patient, rounded patches of inflamed skin, without noticeable infiltration, could be seen which might well be the prodromal lesions preceding the tumefaction noticeable in so many parts of the patient's body.

Case for Diagnosis.

By J. L. BUNCH, M.D.

THE patient, a man aged about 45, had been in Trinidad, West Indies, for three years. While there he had two similar lesions to those now seen: one on the left thumb, and one on the left leg. They had retrogressed, leaving the slightly indurated, red patches now seen. Since the patient's return from the West Indies six months ago a raised, purplish, somewhat thickened lesion had made its appearance on the right forearm, which had increased in size, until it was irregularly circular, with a diameter of about 2 in. Three small sinuses were now present, two of which had only recently made their appearance, while the patient was taking iodide internally and applying lotio nigra externally. There was also a small subcutaneous nodule above the elbow, which also involved the skin, but there was no reddening or discoloration of the skin over it.

Cultures made from the discharge from the sinuses did not grow a sporothrix, as was thought likely, nor did microscopical examination of the discharge show actinomyces or the bacillus of Hansen. This latter negative finding was of interest, as the patient had been thrown into contact with lepers, and a diagnosis of leprosy had been made by another physician, but there was no anæsthesia, the ulnar nerve was not thickened, and Dr. Bunch did not agree with such a diagnosis.

The possibility of tuberculosis had been considered, but the history did not point to it and the von Pirquet reaction was negative. The patient absolutely denied syphilis, and treatment with iodide and lotio nigra only resulted in the appearance of two fresh sinuses.

DISCUSSION.

Dr. MacLeod did not think a diagnosis could be made without a biopsy. He did not regard it as the grave disease which Dr. Bunch mentioned. The appearance of the case, he thought, strongly suggested some local infection, such as a streptothrix of the actinomyces type. Sometimes in actinomycosis it was extremely difficult to find the ray fungus. Therefore he suggested that in this case a search should be made for it in sections of a recent nodule or at the growing edge. Several varieties of streptothrix had been isolated in lesions of a similar clinical type.

Dr. J. J. Pringle was inclined to agree with Dr. MacLeod's suggestion that it might be a case of actinomycosis: the colour was very significant, and so was the haphazard distribution. It used to be said that actinomycosis was a disease which travelled "across country": it followed no definite distribution of veins or lymphatics. In the present patient there were two patches on the arm and leg in no anatomical relation to each other. Dr. MacLeod's other remark that it was often difficult to prove streptothrix invasion by microscopic examination was also notoriously true, especially in patients who had been under treatment.

Dr. Adamson did not think the case could be actinomycosis, because it was very unusual for that disease to begin in the skin; it practically always involved the skin from some deeper structures. He thought it possible that it was a case of sporotrichosis; there had apparently been an abscess which had broken through the skin with multiple openings, in a manner very characteristic of sporotrichosis. He did not think a culture could now be obtained from the lesion on the arm, but it might be possible to get one from the unbroken nodule on the upper arm. The man might have been taking iodide of potassium, which would account for the healing up of the lesions on the leg.

Dr. Pernet said he did not regard the case as one of leprosy. The possibility of a streptothrix was on the cards. In a case of the late Dr. Radcliffe-Crocker's, Dr. Pernet had found streptothrix from an infiltrated lesion with soft fluctuating points over the left hip. The streptothrix was characteristic, viz., mycelial elements, very numerous, aggregated here and there into felted masses, but there were no rosettes of clubs. Dr. Pernet exhibited the Gram eosin-stained preparation before the Dermatological Society of London in 1905.[1] If the nodule in the present case were examined, something helpful to diagnosis might be found in it.

Dr. BUNCH replied that he had thought it was likely to be sporotrichosis, but he was disappointed to find the cultures did not grow. He proposed to excise a portion of the growing edge of the nodule. He gave the man iodide of potassium for a fortnight, without any appreciable effect, and this fact was not in favour of a diagnosis of sporotrichosis. He had never looked on the case as one of leprosy.

Congenital Hyperkeratosis of the Hands and Feet, &c.

By J. H. SEQUEIRA, M.D.

THE patient was shown as a companion to a similar case exhibited by Dr. Adamson at the last meeting of the Section. The child, a female, was aged 4, and the condition of the skin had been noticed soon after birth. There was an almost exactly symmetrical hyperkeratosis, chiefly affecting the limbs. The lesions were not scaly and there was no general ichthyosis. Both palms and soles were dry and of a brownish-black colour, the epidermis was obviously thickened, and the normal fissures of the skin were exaggerated. The pigmentation of the palms was deeper than that of the soles. The lower halves of both legs and forearms were similarly affected, the hyperkeratosis extending on to the dorsal aspects of the feet. Patches of normal skin were visible between the thickened areas on the extremities. The skin of the buttocks was dry and rough and slightly hyperkeratotic, and a similar condition extended for a short distance on to the thighs. Both sides and the back of the neck showed similar areas of horny thickening. It was noteworthy that the flexures of the axillæ and the flexor aspects of the elbows and the popliteal areas were all affected. The lower part of the back was dry and the skin was of a brownish colour. The front and back of the chest, the abdomen and the face were normal. The scalp was covered with fine brownish branny, rather adherent, scales and the hair was thin and scanty.

The child was very small for her age, and her muscular development was poor. She had never walked and was unable to stand. Her mental condition appeared to be normal. The abdomen was protuberant, but the liver and spleen were not palpable. The cutaneous condition has rapidly yielded to inunction of equal parts of lanolin and olive oil, and frequent baths.

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[1] *Brit. Journ. Derm.*, 1905, xvii, p. 265.

It is interesting to note that the patient's mother, an unmarried woman, was under the care of the exhibitor for secondary syphilis in November, 1912. She also suffered from infancy from an abnormally dry skin, the note made when she was under treatment being: The skin of both palms is dry and cracked, and the skin of the soles is also thickened and covered with tough, horny scales. Both legs from the knee to the ankle are covered with dirty brown to blackish scales suggestive of ichthyosis hystrix. The edges of these patches fade away into the surrounding skin, which is xerodermatous. The elbows are covered with similar scales, and also the shoulders and anterior axillary folds. The flexures of the elbows, the loins, buttocks, and the spinal furrow are also dry and xerodermatous. The scalp shows a tendency to scaliness.

So far as could be ascertained no other relatives are affected.

The points of interest in this form of hyperkeratosis are the symmetry of the lesions, the involvement of all four extremities, and the marked predilection of the flexures. In these respects the condition differs essentially from ichthyosis hystrix or nævus unius lateris, and from the universal form of ichthyosis which affects the flexures less than other parts.

DISCUSSION.

Dr. ADAMSON said the case he showed at the last meeting[1] had one or two patches on the body also. These cases of localized ichthyosis he regarded as different from linear nævus: they were not warty but scaly. They differed also from generalized ichthyosis: in ichthyosis the palms and flexures were smooth. In this child there were patches on the flexor surfaces of the arms. He did not think the fact that the mother had ordinary ichthyosis meant that the daughter had the same complaint. Neither did he consider these cases of localized hyperkeratosis the same disease as congenital hereditary palmar hyperkeratosis (malady of Méleda). They need not be limited to nor even affect the palms or soles.

Dr. MACLEOD said that he considered that these cases were allied to congenital hyperkeratosis of the palms and soles, and that they belonged to a different category from ichthyosis.

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[1] See *Proceedings*, p. 136.

Case of Lupus Erythematosus.

By J. H. Sequeira, M.D.

The patient, a married man, aged 43, presented the common lesions of lupus erythematosus in the butterfly-shaped patches across the face, with central cicatrization and red, scaly margins. There was also a small patch of exfoliation on the lower lip. The backs of the hands and the knuckles were the seat of red, raised, chilblain-like patches, which were always worse in the winter, and sometimes disappeared in the summer months.

The case was brought to show the extensive involvement of the lymphatic glands of the neck. In this region there was a chain of very large, softening glands extending almost from one ear to the other. There were also numerous scars of operations upon previously affected glands. In addition to this the patient had a dull patch over the left scapular region, and deficient breath sounds were audible in the region. Two years ago he had hæmoptysis. There was no evidence of active phthisis now, but the patient had a slight nocturnal rise of temperature and sweating. The lupus erythematosus had been present since the patient was aged 16.

DISCUSSION.

The PRESIDENT remarked that if lupus erythematosus was a toxæmia, the toxin was as likely to be that of tubercle as any other. But those who did not agree as to its tuberculous nature did not believe subjects of lupus erythematosus were all tuberculous. At the time the Koch craze was at its height he gave many injections in cases of lupus erythematosus, but a local reaction was never produced. One might see enlarged glands in this disease, but they were not necessarily tuberculous. It must be admitted that the pathology of lupus erythematosus was not yet properly understood.

Dr GALLOWAY said that he thought that the soundest position to occupy in the vexed question of the ætiology of lupus erythematosus was that the lesions of the disease could be produced by more than one toxæmic process. Still, it was not to be denied that the coincidence of tuberculosis and lupus erythematosus in the same individual had impressed many observers strongly with the idea that a causal connexion between the two states of disease always existed. But lupus erythematosus was associated with many other diseased states than tuberculosis. A case similar to the patient shown by Dr. Sequeira had been under his care till his death, which occurred a few weeks ago from subacute pulmonary tuberculosis. He had noticeable, though not extensive, lupus erythematosus of the face and hands associated with great enlargement of the lymph glands in the neck, axilla, and elsewhere. The glands remained firm and large for so long that the case afforded a good text for frequent demonstrations of the differential diagnosis between Hodgkin's disease and enlargement of the glands due to chronic tuberculosis.

Dr. PRINGLE said he was, and had long been, of the opinion that the association of tuberculosis and lupus erythematosus was much closer than most dermatologists could be brought to admit, and he was fully aware of the numerous and cogent arguments against his view. It had, however, been brought home to him, many years ago, by the greatest tragedy which had occurred to him in his professional life. During the early phases of the Koch craze dermatologists were maintaining that lupus erythematosus was not tubercular or connected with tuberculosis. So repeated and assertive were the statements on the point that he gave a subject of the disease, an apparently healthy boy, an average dose of Koch's tuberculin, merely with the object of a negative demonstration to his class. As the result a latent focus of tuberculosis in him became extremely active and the lad died of acute tuberculosis in a few weeks. The impression made upon his mind was, naturally, a very deep one, and his subsequent experience had convinced him that his belief was not merely based on "the evidence of things not seen." If the ætiology of lupus erythematosus could be reduced to one sole cause, he could not but believe that this cause was tuberculosis, although he had no theory to advance as to the intimate nature of the relationship.

Dr. AGNES SAVILL said she had had a case almost similar to that mentioned by Dr. Pringle, about six years ago. It was that of a patient at hospital with a very severe and extensive lupus erythematosus on the face of many years' duration. Treatment by various physicians had done very little good. She heard of tuberculin as a method of treatment and began with giving every two or three weeks for three months $\frac{1}{1000}$ mg. The lupus erythematosus cleared up like magic; but at about the sixth injection the girl manifested acute tuberculosis, for which she was taken into Brompton Hospital; she was dead in four months. The speaker had not used tuberculin since for lupus erythematosus.

Dr. WILFRID FOX remarked that several people had written papers on the subject in which they pointed out that they did get reactions with Koch's original tuberculin. He agreed with Dr. Pringle that the tuberculous was the most important and most frequent toxin found in connexion with the disease. There was now a case at St. George's Hospital of lupus erythematosus which had been treated in various ways by Dr. Freshwater. After two months the patient returned with a large gland, which on palpation was suggestive of a tubercular gland.

Dr. ADAMSON did not consider the evidence for the tuberculous nature of lupus erythematosus convincing. Both diseases were so common that it was not surprising that they sometimes occurred together in the same patient. The association of tuberculous lesions with lupus erythematosus was certainly not so frequent as with the eruptions which we called tuberculides, acne scrofulosorum, acnitis, Bazin's disease, &c. In a fatal case of acute lupus erythematosus at St. Bartholomew's Hospital there was no post-mortem evidence of tubercle, and the patient died of acute pneumonia.

Dr. MACLEOD said that it seemed to him that the evidence was insufficient to establish a direct causal connexion between lupus erythematosus and tuberculosis, and believed that the association of the two was a coincidence, though he admitted that being a weakening disease, tuberculosis might be a predisposing factor. He considered that there was nothing in the histology of lupus erythematosus to suggest tuberculosis of the skin. Tubercle bacilli had never been found in the tissue, and inoculation experiments had invariably given negative results. This alone showed that it was not due to tubercle bacilli in situ. Nor was he convinced of the toxi-tuberculide theory of its origin.

A Peculiar Case of Lymphangioma Circumscriptum in a Girl, aged 6.

By J. M. H. MACLEOD, M.D.

THE lesion was situated on the chest beneath the right breast and consisted, at the time of exhibition, of a slightly raised, rounded, smooth swelling, about 2 in. in diameter, the skin over which appeared to be normal; it was not definitely demarcated, but faded into the surrounding skin. On the lower part of the swelling there was a number of small clear vesicles varying from a pin's head to a split pea in size. These were irregular in outline and tended to form small clusters which had coalesced in one situation into a small bulla about the size of a large pea, which was uneven on the surface, as if it were multilocular. In the bulla and also in some of the vesicles the contents had become hæmorrhagic.

The history of the condition was as follows: The diffuse rounded swelling was noticed soon after birth, and had not increased to any extent with the growth of the child. Some time ago, before the patient came under the observation of the exhibitor, an incision had been made into the upper part of it and an attempt was made to scrape it out. In this way the size of the lesion had been considerably reduced. The contents were said to be of a "fibro-cystic" character, but unfortunately had not been examined microscopically. There was no bleeding from the tumour. The description of the contents was suggestive of a cavernous lymphangioma. Until two years ago the nævus had given no trouble. One day about that time it felt hot and painful, and a crop of vesicles appeared, some of which in a few days became hæmorrhagic. In about a fortnight these had completely dried up, and formed scabs, which came off without leaving scars. A week later another attack occurred, and it has been going on recurring almost every three weeks ever since.

The child's general health seems fairly good and there is no other congenital anomaly present, nor history of such in the parents.

DISCUSSION.

The PRESIDENT considered that it looked like lymphangioma. There did not seem to be any erysipelatoid condition.

Dr. F. PARKES WEBER thought that the subcutaneous tumour of the right mammary region, which was said to have a cystic fibromatous structure, was in reality a cystic lymphangioma. The cutaneous vesicles in the neighbourhood were probably superficial manifestations of the same lymphangiomatous growth. Hæmorrhage often occurred into the minute lymphatic vesicles of lymphangiomata. In this case the hæmorrhage into the superficial lymphatic vesicles was perhaps the cause of the coagulation and scab formation. When the scabs thus formed were cast off, a fresh "crop" of superficial lymphatic vesicles would soon appear, which, in their turn, would be also transformed into scabs and thrown off, and so on, in periodic cycle.

Dr. DORE said he had treated a case of lymphangioma circumscriptum (for Sir Malcolm Morris) with X-rays and it cleared up completely after several pastille doses.

Case of Pigmented Nævi-like Freckles in a Girl, aged 16.

By J. M. H. MacLeod, M.D.

The nævi were like large brown freckles, irregular in outline, about the size of a lentil, and distributed on the left side of the trunk, from the axilla to the buttock. They appeared five years ago. In addition to the freckles there was a faint superficial capillary nævus on the right wrist and back of the hand, which had been noted at birth.

DISCUSSION.

Dr. Adamson said he had shown an almost identical case of unilateral freckling, involving the same area, and had published a photograph of it. In that case there was a supernumerary nipple on the opposite side, a circumstance not very uncommon with unilateral pigmentary nævus.

Dr. F. Parkes Weber considered that the marbled red appearance of the skin at the back of the patient's right wrist (an appearance which could be made to temporarily disappear by friction) was an excellent example of congenital local "livedo annularis" ("livedo reticulata"), a condition intimately allied to ordinary capillary hæmangiomata (telangiectatic areas) of the "port-wine nævus" kind.

Dermatological Section.

June 19, 1913.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case of Recurrent Nodular Eruption of the Hands and Face for Diagnosis.

By H. G. ADAMSON, M.D.

THE patient, A. S., was an unmarried lady, aged 57. The eruption had first appeared twenty years ago, rather suddenly, during a visit to Oxford, and she had then attributed it to the place not agreeing with her. But the eruption had continued for twenty years, with intervals of freedom. It had always been better in the winter, and about ten years ago, after extraction of many stumps of teeth, it had disappeared entirely for two years. It usually got better when she took a mixture containing mercury and iodide of potassium. She had recently had swelling and pain in the left knee which had been diagnosed as rheumatoid arthritis and which had got well with ionic medication.

The eruption now present consisted of numerous dusky red nodules on the face, neck, and hands and fingers. The nodules were scattered irregularly over these parts. They varied in size from that of a hemp-seed up to disks about $\frac{1}{4}$ in. in diameter. There were about a dozen on each hand and the same number on the face and neck. Each nodule was raised (up to $\frac{1}{8}$ in. for the larger nodules), firm, dusky red, with smooth, shiny surface. On pressure the redness disappeared, leaving the nodule momentarily of a pale yellowish colour. There was no appearance of vesication, necrosis, pustulation, or crusting. On close inspection some of the larger nodules appeared indefinitely lobulated as though made up of separate smaller nodules, and some of them had a suggestion of a "ringed" appearance (like granuloma annulare, but by no means so obviously ringed as the lesions of that disease). The nodules did not itch and they were not tender. Each nodule lasted about three weeks and then gradually faded, leaving no trace. There were no lesions in the mouth, but on the outer side of the left sclerotic there was a wide leash of congested blood-vessels passing over the cornea and dotted here and there with pinhead-sized opaque spots. This condition suggested a phlyctenular conjunctivitis, or possibly a miliary tuberculosis of the conjunctiva. Such appearances in the eye, sometimes in one eye, sometimes in the other, had been frequent, and until this attack they had disappeared on taking the mercury and iodide mixture. One other point to be noted was that the patient still had several teeth in the lower jaw which were loose and showed marked pyorrhœa at their bases. The exhibitor had never seen an eruption quite like this and was unable to recall any description of a case which corresponded with it. Perhaps the two eruptions which it most resembled were granuloma annulare and erythema elevatum diutinum of Crocker, and he was inclined to put it into the latter group, although the lesions in the case depicted by Dr. Crocker were much larger. He thought a drug eruption could be excluded. It seemed possible that both the "rheumatoid arthritis" and the eruption might depend upon absorption from the affected tooth sockets, and this view was supported by the fact that the eruption had previously disappeared for two years after removal of diseased stumps. But against this was the fact that pyorrhœa was so common, and this eruption was certainly unusual, if not unique.

Additional Note.—Since the patient was seen at the meeting of the Section the eruption has almost disappeared and the conjunctivitis has cleared up. This took place after taking six doses of the mixture containing liq. hydrargyri perchloridi, 20 minims, and potassii iodidum, 2½ gr., to each dose. After the first three doses the eruption began to get pale and there is now only a faint brown stain at the site of each nodule. This mixture was first prescribed six years ago, and it always has the effect of at once clearing off the eruption. Dr. George Pernet has kindly furnished the exhibitor with the note that Dr. Radcliffe-Crocker's diagnosis in 1893 was erythema papulatum. Dr. Pernet saw the case in 1896, when the eruption was practically the same as when the case was exhibited at the meeting, and there was a phlyctenular conjunctivitis of both eyes.—H. G. A.

DISCUSSION.

Dr. WILFRID FOX thought it was a likely suggestion of Dr. Adamson that the condition was due to absorption of toxic products from defective teeth. He had seen a case with a similar kind of condition, long-lasting, and with a somewhat similar distribution, in a patient the subject of chronic staphylococcic bone disease, with slow necrosis and the formation of a sequestrum.

Dr. WHITFIELD said this case presented a new clinical picture in his experience; he had not seen anything precisely like it. If it had been acute he would have thought of erythema papulatum.

Nævus Unius Lateralis and Unusual Effects of Solid CO_2 (Dermatitis Repens).

By W. KNOWSLEY SIBLEY, M.D.

PATIENT is a well-developed, healthy-looking girl, aged 18, who had an attack of scarlet fever when aged 5, and after this her mother said she first noticed some "warts" on her right hand and side of the chest, which have persisted ever since and gradually increased. The lesions tend to form lines and the distribution is distinctly asymmetrical, the right side only being affected. A large group exists over the right breast and forms an irregular circle around the nipple. Considerable groups are present in the right axilla, from which three distinct radiating lines descend vertically downwards. Irregular groups are present below the inferior angle of the scapula and over the ribs; a very distinct band runs over the deltoid in a wavy curve across the scapula to about the middle line and in front extends over the deltoid down the upper third of the arm. Other less distinct bands are present about the anterior bend of elbow.

Under the microscope a section from one of the growths taken from the axillary region shows hypertrophy of the prickle cell layer, and the blood-vessels are surrounded with some cell infiltration. There is no thickening of the stratum corneum—that is, no hyperkeratosis: a typical acanthoma.

On March 18, 1913, a stick of solid CO_2, measuring 3 in. by ½ in., was applied for one minute to a group situated on the right breast, and afterwards the end of this stick was applied to four or five small spots in the front of the chest, and in the axilla for a similar time. The usual blebs appeared a few hours afterwards and everything went on normally for the next week or so. On March 25 the patient had an attack of tonsillitis with a temperature of 103·6° F., and on March 26 there appeared a sudden and rapid extension of the ulcerated surface wherever the solid CO_2 had been applied. The wound in the breast now presented a gaping, angry-looking mass of exuberant, fungating, granulation tissue measuring some 5 in. by 2 in., and those in the axillæ had become very deep and the muscles were exposed in the bases. The tip of a finger could be inserted for ½ in. or more. The condition remained very resistant to treatment and took some ten weeks to heal up.

Cultures from the tonsils showed streptococcus and from the ulcer of breast *Streptococcus pyogenes longus*, and she had some injections of an autogenous vaccine. No growth could be obtained from the blood, and a differential leucocyte count did not reveal anything abnormal.

As to the meaning of this extensive ulceration. The patient shows some obvious neurotic phenomena—a certain amount of anæsthesia is present in the palate and some paranæsthesia over the regions where the lesions are present. She was at the time unable to differentiate when touched with the points of a pair of compasses, except at a distance of some two inches. I venture to suggest that this condition might be a "dermatitis repens," as first described by Radcliffe-Crocker, and that here it has followed the same pathological conditions as a burn, merely a freezing process. It was believed that this disease results from peripheral nerve irritation, and that there is a secondary parasitic involvement of the part, an infective dermatitis, the traumatism being simply an initial factor of the process. The scars are now becoming of a keloid nature.

DISCUSSION.

The PRESIDENT (Sir Malcolm Morris, K.C.V.O.) said that he was inclined to think the case was one of congenital linear nævus.

Dr. WHITFIELD said he thought the condition was a systematized nævus, not warts, and he thought part of the result must be attributed to the very large area which was frozen by the snow at one time, and the unusual time during which the snow was in contact with the part. To apply a rod of such large area for a minute to degenerate tissue was asking for necrosis to ensue. In the case of small hairy moles he had sometimes intentionally so applied the snow as to get a commencing necrosis, but he would not think of using such a large area for so long a time on any skin, except, perhaps, the palms of the hands or the soles of the feet, where the epidermis was especially thick. In this case he did not doubt that the freezing had been overdone. It was difficult to remove the papillary form of mole without causing severe scarring, because it was true papilloma.

Dr. SEQUEIRA said he was inclined to agree with Dr. Whitfield, both in regard to the diagnosis and the explanation of the phenomena which followed the treatment. There were points which Dr. Sibley raised in his remarks about the case which required some explanation. It was stated that the patient suffered from localized anæsthesia and from anæsthesia of the soft palate. He presumed the suggestion was that the patient was hysterical, and that probably the destructive nature of the lesion which followed the application of the snow was of a neurotic character. He would like to know whether that implied that there was a possibility of an artefact element in the case, artefact from the patient's point of view. The streptococcal lesion in the throat was an unfortunate coincidence, but he did not think it could have any particular effect on the nature and the healing of the wound. He agreed with Dr. Whitfield as to the extraordinary difficulty in removing these conditions by means of CO_2 snow; if that were used, it must be pressed to a considerable extent, so as to produce deep reaction. He rarely advised such treatment for these conditions.

Dr. SIBLEY replied that the mother declared that nothing abnormal appeared there until she was aged 5, but of course such conditions were sometimes present without their having been noticed. Previously to this application for one minute, the area was painted with ether and CO_2, and with practically no result. One minute he did not regard as severe; for lupus he had applied the snow for two or three minutes and over very much larger areas than in this case, using quite firm pressure, yet he had never before had such a result as seen here. On a future occasion he would be pleased to show a small boy with lupus in whom large areas had been frozen for three minutes, and with most satisfactory results. He considered there was something very unusual in the condition of the tissues of this patient.

Psoriasis and Pityriasis Rubra Pilaris.

By W. KNOWSLEY SIBLEY, M.D.

ON March 20, 1913, a boy, aged 6, was sent to me by Dr. McHugh with a history that he had had a rough, dry skin for some five weeks, and that the present condition has been present for three weeks and came on more or less suddenly. On inspection the whole face and scalp were covered by a mass of heaped-up dry scale, of such a thickness that the boy could not open his mouth and hardly separate his teeth. There was a fairly well defined margin to the exuberant scaly condition, and the excessive dry, heaped-up scale formation was present to the same extent over the whole scalp. The arms, forearms, hands, thighs, legs, and feet were all covered by scale of moderate degree, which was especially abundant and heaped-up over the patellar regions. The palms and soles were also affected, and showed a general thickening or keratosis, rather than any distinct eruption. What appeared to be a distinct guttate psoriasis in quite an early stage was abundantly present over the trunk. With the exception of the scalp there did not appear to be any marked affection of the hair-follicles.

Under the microscope a section of one of the lesions taken from the dorsal surface of the forearm showed a fairly typical psoriasis lesion, marked hypertrophy of the horny layer, with flat, horny cells (parakeratosis), and atrophy of the stratum granulosum. The stratum mucosum was very much thickened, the hair-follicles were filled with horny material, the papillary blood-vessels were dilated, and the other vessels were surrounded with round cells and leucocytes.

The child looked distressed and ill; the temperature varied from 99° to 100° F., and he complained of pains, especially about the flexures of the elbows.

In a few days the whole body presented the appearances of an acute exfoliative dermatitis, or pityriasis rubrum, and in some two or three weeks the excessive scaly condition had separated from everywhere with the exception of the scalp, where the presence of the hairs prevented its removal. At this time the scalp presented a very unusual appearance of deep furrows and heaped-up ridges.

DISCUSSION.

Dr. Sibley added that the first diagnosis he made was psoriasis on an ichthyotic skin. A section under the microscope at this stage was of typical psoriasis. The section was taken from the dorsal surface of the boy's forearm, and it did not show any marked cornification in the hair-follicles; the present marked prominence of the hair-follicle on the dorsum of the finger had appeared since he last saw the case about a fortnight previously. There was some thickening of the palms and soles.

Dr. J. J. Pringle could not accept the exhibitor's diagnosis of the case, which he considered to be a typical example of pityriasis rubra pilaris in process of spontaneous recovery.

Dr. ADAMSON agreed with Dr. Pringle in the diagnosis of pityriasis rubra pilaris. It was a typical example of pityriasis rubra pilaris. The boy also had psoriasis. As the speaker had pointed out on several occasions, the association of psoriasis and pityriasis rubra pilaris was not infrequent. Indeed, he believed that pityriasis rubra pilaris was really a phase of psoriasis—a follicular psoriasis. The case of F. C., aged 7, shown by Dr. Adamson in May, 1911, and again by Dr. Sequiera in June, 1912, was an example of alternating psoriasis and pityriasis rubra pilaris.[1] Dr. Whitfield had also shown a case in a child, aged 4½, in which the eruption at one time took the form of follicular psoriasis and at another that of pityriasis rubra pilaris,[2] and Dr. Little had mentioned a case of pityriasis rubra pilaris in which the sister had psoriasis.[3] The behaviour of pityriasis rubra pilaris was like that of psoriasis in its tendency to spontaneous cure with relapses.

Dr. WHITFIELD was in partial agreement with Dr. Adamson's remarks. He said that some years ago Dr. Poynton showed two cases of pityriasis rubra pilaris, other members of the same family having had psoriasis. Several such cases were on record. Pityriasis rubra pilaris did not react to treatment like psoriasis, and although he thought the two diseases were related, he did not consider them identical.

Dr. J. M. H. MACLEOD expressed his agreement as to the combination of the two diseases in one patient, but he regarded pityriasis rubra pilaris as a separate disease from psoriasis.

Dr. GRAHAM LITTLE had had a case of pityriasis rubra pilaris of many years' standing, in a young girl, whose sister he had also seen: the latter had typical psoriasis. Dr. Little could not agree with the opinion that pityriasis rubra pilaris and psoriasis were the same affection: their behaviour under treatment and the distribution and character of the lesion were markedly dissimilar.

Dr. PERNET also considered that pityriasis rubra pilaris and psoriasis were different conditions. This was especially so in adults, he considered.

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[1] *Brit. Journ. Derm.*, 1911, xxiii, p. 181, and 1912, xxiv, p. 280.

[2] *Brit. Journ. Derm.*, 1902, xiv, p. 470, and 1904, xvi, p. 462.

[3] *Brit. Journ. Derm.*, 1900, xii, p. 92.

Neuroma Plexiforme.

By J. H. Sequeira, M.D.

The patient, a well-grown lad, aged 13, came to the London Hospital on account of numerous small swellings on the left hand. The parents, who are intelligent people, were certain that the swellings had not been noticed until about six years ago. The first area observed to be affected was the left thenar eminence, and at intervals similar lesions have been observed, first on the left index, the dorsal aspect of the thumb, and during the last twelve months others have been noticed on the second and little fingers, and on the flexor aspect of the wrist. There has been no pain or tenderness, but the spots are said to itch occasionally. There was no history of injury, and no other skin trouble.

The eruption consisted of small flat elevations, the colour of the normal skin, hard to the touch, quite movable, and varying in size from a pin's head to a split pea. On the left thenar eminence there was a ring of nodules the size of a two-shilling piece. On the left thumb there was a collection of small nodules on the flexor aspect at the junction of the first and second phalanges, and on the dorsal surface there were a few similar elevations just above the nail, and some discrete, linearly arranged lesions on the dorsal aspect of the second phalanx. On the left index-finger there was a line of small nodules extending from the metacarpal to the tip of the finger. On the radial and dorsal aspects there were small groups also. On the left middle finger, at the base of the first phalanx on the palmar surface there was a small circular group of small nodules. On the little finger there was a nodule on each side of the palmar surface of the distal phalanx. On the ulnar side of the front of the left wrist there were three discrete nodules.

An accurate diagnosis was impossible, and one of the small growths was excised and sent to Dr. Turnbull for microscopical examination, and to him Dr. Sequeira is indebted for the following report: "The epidermis is quite normal, and shows short interpapillary processes. There is an abundant dense fibrous dermis, and in this are many large and several small, well-defined nerves. The nerves have a distinct peri- and endo-neurium. The endoneurium is frequently thicker than normal, but the nerve-fibres are well developed and numerous. The arrangement of the nerve-fibres is more irregular than normal. The majority of the nerves are cut transversely. The arrangements of the segments of nerve in section suggest that the segments are sections of one or more tortuous nerves from which branches are given off."

The case is an unusual one, and appears to belong to the group associated with the name of von Recklinghausen, but the anomaly is here localized. It seems almost certain that it is of congenital origin, and this would account for the absence of pain in the lesions.

The PRESIDENT remarked on the absence of tenderness.

Case for Diagnosis.

By J. H. SEQUEIRA, M.D.

THE patient, a lad, aged 13, came to the London Hospital on account of swellings on the third finger of the left hand. The finger appeared to have been normal to all appearances until the boy was aged 5, when a small, wartlike lump was noticed on the dorsum of the left third finger. This swelling was painted with iodine but did not disappear. Since then fresh lesions have appeared from time to time, and some even within the last twelve months. There was no history of injury and the lesions have been painless and free from itching. On the third finger of the left hand, the skin over the second phalanx was slightly red and glazed, the whole phalanx being spindle-shaped. On palpation the swelling was found to consist of several small hard nodules, varying in size from a lentil to a pea. Three of these were on the ulnar side of the articulation of the first and second phalanges. Four nodules were grouped about the head of the second phalanx, and one large nodule at its base. Three nodules lay along the radial side of the same phalanx, and one on the palmar aspect. The nodules were distinctly subcutaneous, the epidermis over them was not movable apart from the nodules and had a stretched appearance. The nodules did not appear to be fixed to the bone. The general appearance of the finger suggested strumous dactylitis, but there had been no suppuration, and the swellings were discrete and hard. There was no enlarged gland at the angle of the jaw on the left side, and the boy had had double pneumonia when he was aged 7, but the swelling had been present before this. An aunt on the father's side had died of phthisis.

The exhibitor believed that the growths were fibromata, and this view was shared by several members present. A biopsy has been made, but the report on the specimen is not yet to hand.

A Case of Lichen Planus of the Tongue and Lips.

By A. Whitfield, M.D.

The patient, a young man, first noticed a small grey patch on his tongue at Christmas, 1912. This spread until the whole surface of the tongue was involved, and two months later he developed "eczema" of the arms and legs. When shown, the tongue was evenly grey in colour to the naked eye, but with a lens this greyness could be seen to be formed by fine white stippling. The insides of the cheeks showed the common white "embroidery" of lichen planus and there was a similar condition of the dry mucous membrane of the lips. On the flexures of the elbows, the groins, the outer sides of the trochanter and and the body of the penis (not the glans) there were numerous typical domed papules of lichen. Some, but not all of these, were circumpilar in origin and their grouping was rather unusual in character, being distinctly corymbose in arrangement.

Dr. Whitfield said that lichen on the mucous membrane of the cheeks was very common, but he thought such extensive involvement of the tongue and the dry part of the lips was unusual. The rash was not very irritable and was already undergoing involution after a three weeks' arsenical treatment.

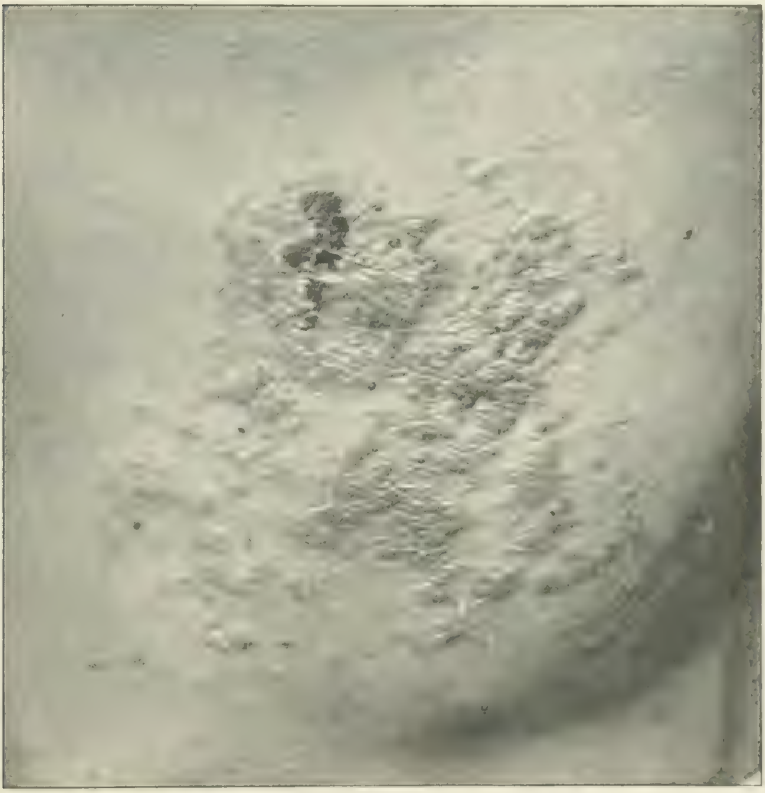
The President remarked that the appearance of lichen planus on the mucous membranes before coming on to the skin was commoner than was generally supposed. And one could not safely generalize on the symptom of itching, because in some undoubted cases itching was severe, whereas in others there was scarcely any irritation.

Case of Lymphangioma Circumscriptum.

By H. MacCormac, M.B.

The patient was a male, aged 18. He had been born with a condition similar to the present, but this had been excised. Four months after the operation the disease had recurred, and had gradually increased and spread forwards. It now occupied a position over the left lower ribs measuring $4\frac{1}{2}$ by $3\frac{1}{2}$ in., and consisted of closely set vesicles from a pin-head to a hemp-seed in size. There were no associated blood-vessel changes, but a few warty thickenings of the skin were to be seen. There was a history of recurrent erysipeloid attacks.

Microscopical sections demonstrated that the condition was almost entirely confined to the papillary layer, where numerous large cavities lined by a single layer of endothelium were found. No relationship seemed to exist between these lymphatic spaces and the blood-vessels of the part.



Case of lymphangioma circumscriptum.

The PRESIDENT said all the cases of the condition which he had watched—some of them for a considerable time—had gradually spread, and even surgical removal did not seem to be always efficacious. He saw Sir Jonathan Hutchinson's original case.

Case for Diagnosis.

By H. MacCormac, M.B.

The patient was a healthy man, aged 73. Fifty years ago he had had gonorrhœa, but there was no history of syphilis. He had been married forty-six years and had two healthy children. Fifty years ago he was frost-bitten. Two years later he had an attack of what he calls scurvy, the left foot breaking out in ulcers and patches about the ankle; at the same time his gums were spongy, red, and bleeding easily. No drugs were being taken at this time. He recovered from this in about fifteen months, the ankle and foot healing completely, but leaving some scarring and pigmentation behind. The present condition began four years ago, when "little patches came out on the foot and ankle, with redness and swelling, and a discharge of black water." During the last year there has been a considerable amount of pain.

At the present time the foot and ankle are considerably swollen, and there are numerous sinuses from which a thin fluid flows, containing small masses of yellow pus. On the inner side of the foot there is a considerable thickening of the skin, with some deep ulceration; there are also many warty-looking papillomata. The whole condition bears a close resemblance to Madura foot, but no streptothrix has been seen in stained preparations, nor has any been grown. An X-ray photograph shows that the bone has not become involved. The blood count showed some secondary anæmia. Wassermann reaction strongly positive.

Case of (?) Syphilide.

By S. E. Dore, M.D.

The patient was a middle-aged woman, sent by Dr. Travers Smith. She had had the eruption for nine years. There was no history pointing to syphilis; she was the mother of six healthy children. Dr. Dore thought that it was a nodular serpiginous syphilide, but the Wassermann reaction was negative. He invited opinions as to whether this was one of the tertiary cases in which the Wassermann reaction gave a fallacious reading, or whether the diagnosis of syphilis was incorrect. That reaction was so often a reliable guide that when it failed it was apt to lead to difficulties in diagnosis.

Dr. MACLEOD suggested that Wassermann's reaction might be tried again after an injection of salvarsan. He did not think the condition was tuberculous.

Case of Erythema Induratum giving no Evidence of Tuberculosis.

By JAMES GALLOWAY, M.D.

THE question whether a constant relationship exists between the cutaneous malady recognized by the name of erythema induratum and tuberculosis has been a frequent and instructive subject of discussion. In recent years the opinions of Audry, Thibierge and his collaborators, of Boeck, Phillipson, Walther Pick, and Kraus, may be referred to as having influenced the discussion in various directions. The whole subject has been carefully passed under review by Professor Jadassohn,[1] who has enriched his monograph with extensive references to the literature of the subject, and by Dr. J. H. Sequeira, who presents the different aspects of the subject in a very succinct form.[2]

In our own Society the subject has frequently been discussed. The views of Dr. Colcott Fox, based on a wide experience and formally expressed at the International Congress of Medicine in Paris, 1900, and many times at our own meetings, have had great influence on our opinions. Dr. Arthur Whitfield's instructive papers read before the Society in 1905 and 1909 are also prominently in our minds, as they present a different aspect of the subject.[3]

In 1899 I ventured the opinion that the cases of erythema induratum included more than one type of disease, and that, at any rate, two clinical types should be distinguished: one of them characterized by a breaking down of the infiltrated tissue, and by the production of ulceration, the other in which inflammatory œdema of the skin was the more prominent lesion, and which rarely and only accidentally ulcerated. The opinion so stated was given in a somewhat dogmatic form, but it expressed the result of my clinical experience at the time, that all cases of erythema induratum were not of tuberculous origin, and that different causes produced the different clinical types of the disease.[1]

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[1] Jadassohn, in Mracek's "Handbuch der Hautkrankheiten," 1907, iv, pt. 1, p. 116; also Wolff, *ibid.*, 1902, i, p. 577.

[2] Sequeira, in Allbutt and Rolleston's "System of Medicine," 2nd ed., 1911, ix, p. 502.

[3] Whitfield, *Brit. Journ. Derm.*, 1905, xvii, p. 241, and 1909, xxi, p. 1.

The case reported in this paper is of interest because of the evidence it affords that, even when lesions of erythema induratum contain areas of cellular infiltration, the newly formed inflammatory tissue need not be of tuberculous structure nor of tuberculous origin.

The patient, the subject of the following observations, is a woman, A. S., who came under my care on admission into the wards of Charing Cross Hospital on April 17, 1908. She has therefore been under close observation for five years; during this time frequent opportunities have occurred of observing the course of the cutaneous disease and the condition of her health in general. On admission the history obtained was as follows. Her age is 36. She is a short, rather stoutly built woman of medium brunette complexion. She complains of being easily tired and of suffering from palpitation and giddiness when attempting to do any household work involving slight exertion. She married at the age of 19, and during the whole of her married life she says she has been anæmic, and has had bad health.

On investigation the history of ill-health resolves itself into a description of almost constant difficulty in digesting food, and of troublesome constipation. For the relief of these symptoms she has frequently been under treatment by diet and by internal medicine, including the administration of purgatives. She states that three years previous to admission she had what was described as a gastric ulcer, and lived for three months on milk and light food. The evidence in favour of actual ulceration of the stomach is doubtful. Twelve years ago she had what was described as rheumatism, but this also seems to have been of rather indefinite nature. The patient has had no children, and, so far as can be judged by her history, has not been pregnant.

On physical examination she is noted to have the appearance of a considerable degree of anæmia, the pallor of her skin contrasting forcibly with her dark hair and the bright red cheeks, which are characteristic of her complexion and normal in her case. In spite of a long history of ill-health she is stout rather than thin, and has not lost weight recently. The heart is slightly enlarged transversely, and a systolic murmur is heard over the cardiac region. This murmur was at first considered to be due to old endocarditis; it proved to be of functional nature, and disappeared entirely as the patient's health improved. A very slight degree of varix of the veins in both legs was noted. The urine was found to be normal on several examinations. No albumin or other morbid contents were present at any time.

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[1] *Brit. Journ. Derm.*, 1899, xi, p. 206.

Shortly before admission she commenced to develop purple patches and nodules on the legs and lower portion of the thighs, varying in size, but rarely more than 1 in. in their longest diameter. These caused some discomfort and pain, were slightly tender on pressure, and one or two about the knees, which had been rubbed by the clothing, showed superficial excoriations, but no deep ulceration. The patient was kept in bed, and under appropriate dietetic and nursing care and the administration of iron rapidly improved.

The following were the results of the examination of her blood on four occasions during her stay in hospital:—

April 18: Hæmoglobin, 48 per cent.; colour index, 0·57; red blood cells, 4,220,000 per cubic millimetre; leucocytes, 4,000 per cubic millimetre.

April 28: Hæmoglobin, 58 per cent.; colour index, 0·68; red blood cells, 4,290,000 per cubic millimetre; leucocytes, 5,400 per cubic millimetre.

May 5: Hæmoglobin, 74 per cent.; colour index, 0·7; red blood cells, 5,200,000 per cubic millimetre; leucocytes, 6,600 per cubic millimetre.

May 12: Hæmoglobin, 70 per cent.; colour index, 0·7; red blood cells, 4,860,000 per cubic millimetre.

The patient was discharged on May 17, greatly improved in her health, and more capable of doing work without being tired. She is able to digest food without pain; the constipated condition of the bowels was cured; the eruption of painful nodules on the lower extremities had ceased for the time being; those previously existing had healed or become absorbed.

This patient has remained under observation ever since leaving the hospital. Fortunately she has been able to live an easier life. Her husband obtained better remunerated employment, and consequently she did not require to work so hard. The tendency for the erythematous patches and nodules to appear has not ceased entirely, but this condition has never been so severe as before admission to the hospital in 1908. Occasionally, after attempting to do some more arduous household work than usual, one or two reddened, slightly painful and tender nodules will make their appearance under the skin of the calves or of the lower portions of the thighs. These will gradually disappear when she rests. Even a few days' rest in bed will cause the tenderness and pain to vanish from a recently formed nodule. The disappearance of the induration and staining, on the other hand, may not occur for some weeks or months. Within the last year the appearance of these lesions has become less and less frequent. It is now a rare thing for her to develop a definite hypodermic nodule, though patches of purplish erythema may be observed from time to time, lasting, it may be, for some weeks.

The patient has been admitted as an in-patient on two separate occasions subsequent to her first residence in hospital. On March 16, 1912, she was admitted. A few nodules had recently appeared on the legs, and with the patient's consent it was decided to remove one of them for purposes of histological examination. One of the nodules was removed from the left leg, including the skin and subcutaneous tissue. She was again admitted on November 18, 1912. On November 21 a von Pirquet's test was applied to the skin, giving a very slight positive reaction. On November 26, $\frac{1}{1000}$ c.c. of old tuberculin was injected hypodermically at mid-day, the temperature being then 98·4° F. Hourly temperatures were recorded, showing a rise to 98·8° F. at 3 p.m., which fell to 98° F. at midnight. No constitutional or other disturbance was noted. On December 2, $\frac{1}{2000}$ c.c. old tuberculin was administered at 10 a.m., and two-hourly records of the temperature kept for twelve hours. The temperature, which had ranged between 98° and 99° F. on the previous day, had the same range on the day of inoculation and on the day following, the lowest record being 98° F., the highest 99° F. On December 6, $\frac{1}{1000}$ c.c. old tuberculin was injected. She was under close observation for the whole day, and no rise of temperature or constitutional reaction was observed. It should also be noted that no appreciable local reaction took place on any one of these three occasions at the point of injection.

The tissue which was removed on March 18 was divided into two portions, one of them being used for histological examination: the other half was sent to the clinical laboratory of the hospital for experimental inoculation. Dr. Topley has sent me the following report:—

Report, July 2, 1912.—"On March 18 I inoculated a 450-grm. guinea-pig with 1·5 c.c. of a saline emulsion of the tissue removed from Mrs. S., using the intraperitoneal route. The animal gained in weight, and never showed any sign of disease. I killed it to-day, fifteen weeks after inoculation, and made a post-mortem examination. The organs were all perfectly healthy and there was nowhere the least sign of tuberculosis."

When removing the piece of tissue from the leg it was noted that the portion solidly indurated was small in comparison with the total area of tissue apparently affected by the lesion. The larger part of the lesion corresponding to the discoloured area evidently consisted of slightly congested and œdematous tissues.

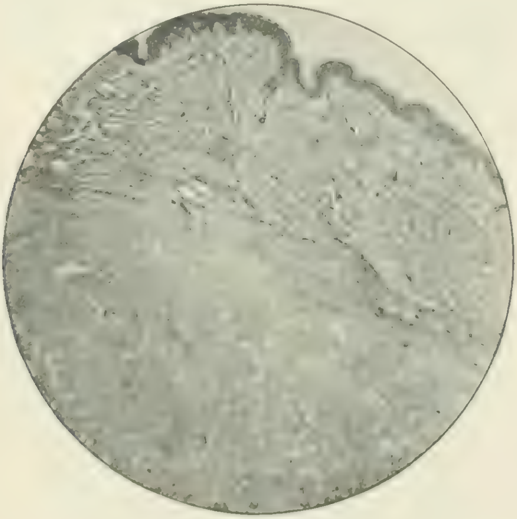

FIG. 1.

Low power view, showing the nearly normal skin, the area of lesion in the hypoderm, infiltrating the subcutaneous fat, the giant cells in groups, and an almost obliterated blood-vessel.

On microscopical examination the area of cellular infiltration is seen to be situated mainly in the lower portion of the true skin and extends downwards into the fat-containing areas of the subcutaneous tissue. One or two thin strands resembling in their structure the cellular characters of this "granuloma" extend upwards towards the epidermis, apparently along the lines of vessels or sweat-ducts; but the epidermis itself and the whole upper portion of the cutis is quite free from the underlying granulomatous mass.

The cells of the granuloma are distinct, well formed, and mononuclear, with a fair amount of surrounding protoplasm corresponding in type to "plasma cells." These cells may be seen passing downwards into the subcutaneous tissue surrounding and causing absorption of the fat cells; in the densest parts the fat of the subcutaneous tissue has entirely disappeared. In the looser areas the spaces occupied by fat cells can be easily seen, giving a spongy texture to the infiltrating mass. Very few, if any, polymorphonuclear cells can be observed in or surrounding the granuloma, and there is no sign of suppuration.

The tissue is noteworthy on account of the numbers of giant cells it contains. These cells appear to occur in groups in certain areas of the granuloma, especially in the middle and lower parts, but they may also be observed scattered sparsely throughout. The giants cells are large, exceeding well defined, and contain large numbers of nuclei, which take the stain firmly and distinctly. These nuclei are frequently seen forming a girdle round the outer part of the cell. The well-defined margin of the giant cells and their distinctness is a remarkable feature of the granuloma. The cells seem almost to be cyst-like, and in some places the manipulation necessary in preparing the microscopical sections seems to have detached the giant cell from the surrounding tissue, turning it out of its place, or leaving a clear area between the wall and

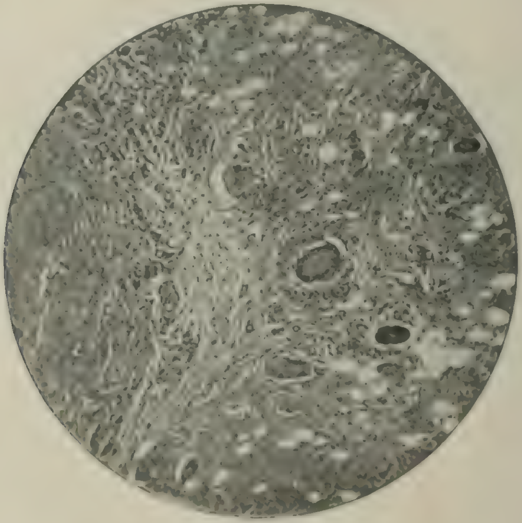

FIG. 2.

Higher magnification, showing the density of the infiltration, the large size of the giant cells, and their loose attachment.

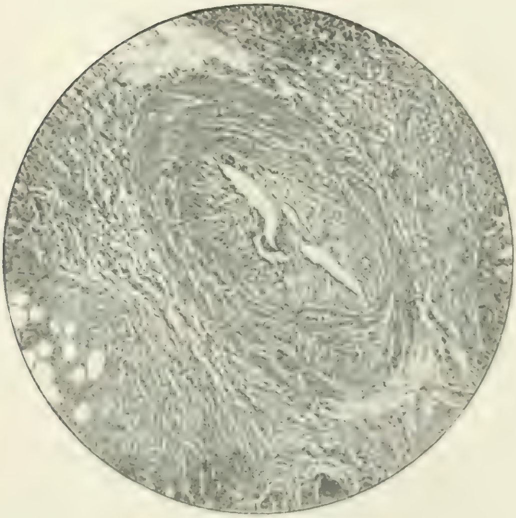

FIG. 3.

An arteriole, showing infiltration of its outer and middle coats by the cells of the surrounding "granuloma" and proliferation of the inner coat.

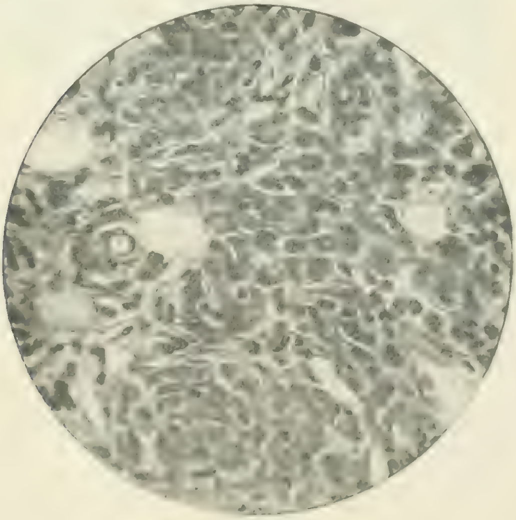

FIG. 4.

A higher magnification, showing the density and the character of cells in the newly formed tissue; the absence of caseation is noteworthy and characteristic.

surrounding plasma cells. The cells, therefore, are evidently bound down very slightly to the surrounding tissue and do not form the centres of a system of epithelioid and other cells such as is so clearly the case in the giant cell systems of the tuberculous granuloma. Throughout the whole area there is no sign of caseation and very little evidence of any other form of necrosis, even in the central protoplasmic areas of the large giant cells.

The vessels in the immediate neighbourhood or involved in the granuloma show remarkable occlusion of their channels, owing to excessive thickening of the vessel walls. The impression produced at first sight suggests the obliteration occurring in severe degrees of syphilitic arteritis obliterans. The process of obliteration, however, differs from that of syphilitic disease in various respects. The thickening of the walls of the vessels is due mainly to a general infiltration of the external and middle coats by the same type of cells as is seen in the surrounding infiltration. These cells may be traced passing between the fibres of the vessel walls in continuous lines from the tissue without. A considerable degree of proliferation of the intima may also be observed. The process is of more acute character than that seen in other forms of specific

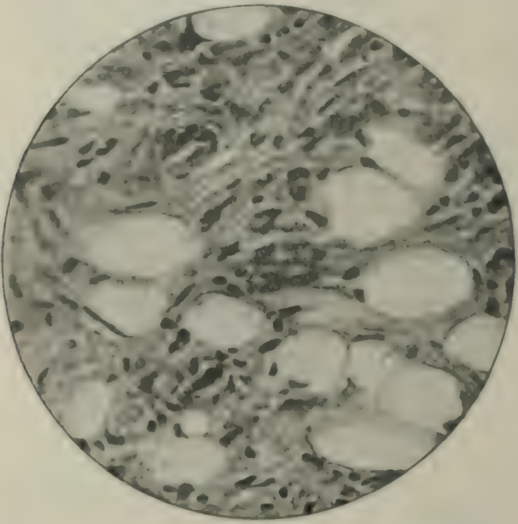

Fig. 5.

Shows the infiltration and absorption of the subcutaneous fat by the cells of the "granuloma."

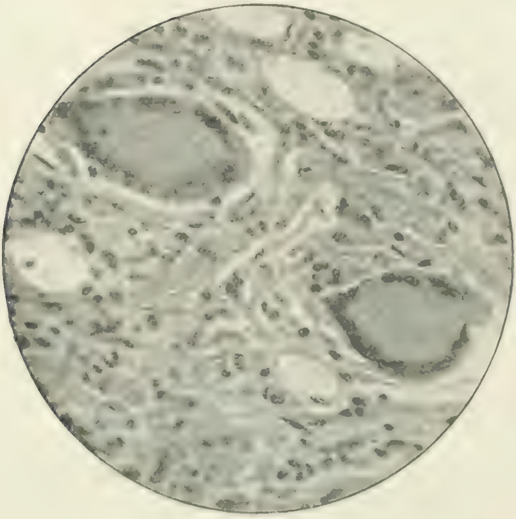

Fig. 6.

Shows two of the well-defined characteristic giant cells and the position and arrangement of their nuclei.

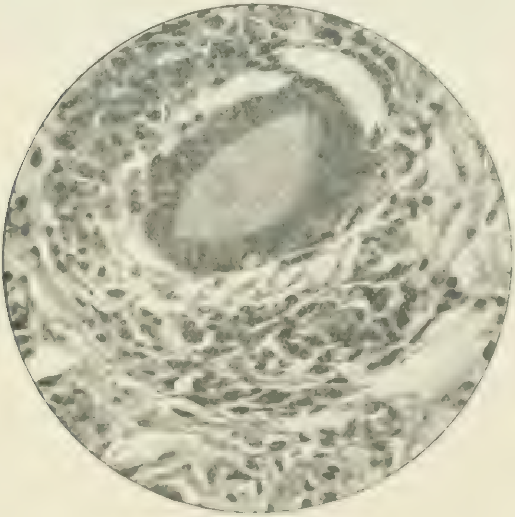

Fig. 7.

A giant cell with surrounding cellular infiltration, showing its loose attachment.

obliterative endarteritis. The venules are probably affected as well as the arterioles.

A number of sections were stained, and carefully examined for the presence of tubercle bacilli by Dr. Walter MacLeod and myself, but none were seen.

There is no history nor clinical evidence of syphilitic infection, and the blood gives a distinct negative complement deviation reaction to syphilis (Wassermann).

From the clinical aspect the following features are noteworthy in this case: An eruption of erythema induratum appears on the lower extremities in a woman, aged 36, following a period of depressed health, of which the main feature is distinct anæmia of the chlorotic type. Notice must also be taken of the fact that indefinite, painful attacks, suggesting rheumatism, have occurred. This rheumatic condition is of doubtful ætiology, and no evidence of acute or simple rheumatism has been noted while under observation. The lesions, ten or twelve of which may have been reckoned at any one time during the earlier stages of the disease, are situated in the cutis and in the subcutaneous tissue. They are distinctly painful and tender on pressure. They are not only indurated but show a considerable margin of purple, congested skin. Ulceration does not occur as a rule, and then only accidentally as the result of superficial abrasion. The ulceration does not pass deeply into the tissues; it does not spread, but tends to heal. The lesions disappear, leaving small areas of pigmented skin, but scarcely any visible cicatrization.

Under favourable conditions, especially of complete rest in bed, the eruption rapidly vanishes, and as the result of her general improvement in health the tendency for the eruption to recur has gradually become less and has now almost disappeared. No physical sign of tuberculosis has been observed, although the patient has been watched for over five years and has been carefully examined many times. A doubtful von Pirquet's cutaneous reaction was given, but a series of diagnostic tests carried out by means of Koch's old tuberculin gave an entirely negative result. There was no disturbance of temperature, no local reaction at the point of inoculation, and no reaction in the lesions of the lower extremities. The patient is now in better health than at any time since she first came under observation.

From the histological point of view the lesion shows in the centre of an area of slightly œdematous and congested skin a small mass of newly formed tissue of the nature of a true granuloma. This tissue is mainly composed of mononuclear cells infiltrating and causing absorption of the fat in the subcutaneous tissue, pressing on, but passing only slightly into the cutis vera. The cells of the new tissue resemble mainly the mononuclear plasma cell type; larger cells, however, may be observed and among these are unusually large, well-formed giant cells. These giant cells resemble protoplasmic cysts and contain large numbers of nuclei, which are often arranged in a very characteristic form around the periphery of the cells. A considerable degree of fibrosis occurs surrounding and throughout the granulomatous area. The appearance of the granuloma differs in its general aspects from tuberculous tissue. The character of the giant cells especially is different. They are evidently loosely attached to the surrounding granulomatous mass and are unusually large and sharply defined. No tubercle bacilli were seen, and a considerable portion of the tissue inoculated into a guinea-pig did not produce tuberculosis.

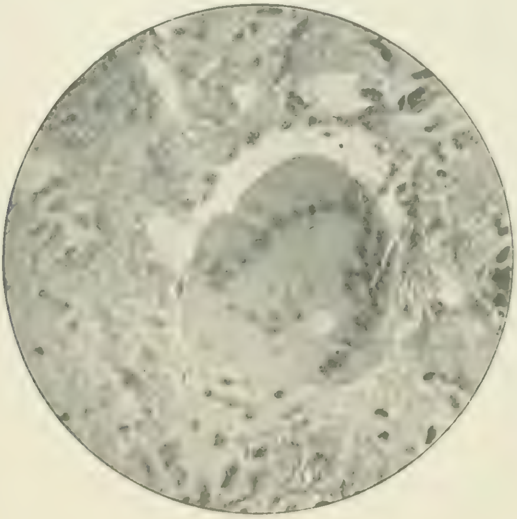

FIG. 8.

A giant cell of a type less frequently present in the "granuloma," showing its loose attachment and easy displacement.

The evidence therefore obtained by observation of this patient supports the contention that, in the malady usually described as erythema induratum—Bazin's disease—there seem to be at least two groups of cases: One of them definitely tuberculous, giving tuberculous reactions, presenting or developing other signs of tuberculosis, and producing tuberculosis on inoculation into susceptible animals.

The second group of cases is not so well defined. Of these at least some, such as the case on record, develop a granuloma with certain peculiar features in the local lesions. The giant cells, which form a striking feature of this granuloma, closely resemble those giant cells described as being of the "irritation or foreign body" type, which may be seen in conditions having no connexion with tuberculosis, and which appear also to be formed by the experimental injection subcutaneously of certain irritative materials, such as fatty acids and cholesterin.[1] The blood-vessels in these cases undergo inflammatory thickening of their walls with occlusion of their channels, resembling the obliterative endarteritis of syphilis and other specific granulomata; the vascular changes in the lesions now under discussion are, however, of a more acute character.

It is probable that other cases of erythema induratum occur in which the local vascular changes are the most prominent features, and that these cases form connecting links between erythema induratum on the one hand and the acute malady erythema nodosum on the other. Such cases would present a close analogy to persistent forms of erythema nodosum.

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[1] Stewart, M. J., "On the Occurrence of Irritation Giant Cells, in Dermoid and Epidermoid Cysts," *Journ. of Path. and Bact.*, 1913, xvii, p. 502.

Dermatological Section.

July 17, 1913.

Sir MALCOLM MORRIS, K.C.V.O., President of the Section, in the Chair.

Case of Acute Lupus Erythematosus with Lupus Vulgaris (or (?) Lupus Pernio).

By H. G. ADAMSON, M.D.

THE patient was a gentleman, aged 50, who had spent twenty years in India. While in India he had had ague. He had also had leucodermia, of which there was now no trace. A few months ago a little sugar was found in the urine and this had disappeared on dieting. He was otherwise apparently in robust health. The eruption on the face and scalp consisted of four red, raised, sharply circumscribed patches: one on the right temple (2 in. by 1½ in.) and involving the right upper eyelid; one on the forehead above the inner end of the left eyebrow; one on the right cheek, and another on the right side of the scalp, each of these about the size of a shilling. The patch on the scalp and that on the cheek had the characteristic stippled appearance of lupus erythematosus, but the two on the forehead did not show this. The patches had appeared almost suddenly three months ago, and had gradually increased in size. On the right side of the abdomen there were two large oval patches, red-brown in colour, and showing the typical "apple-jelly" nodules of lupus vulgaris. One patch had been present two years or longer, and was scarred in the centre; the other had appeared within the last six months. He showed these cases on account of the interest of the association of acute lupus erythematosus with lupus vulgaris, but he was prepared to question his earlier diagnosis, and to ask, Was this a case of lupus pernio and not an association of what he believed to be two distinct affections—lupus erythematosus and lupus vulgaris?

Additional Note.—Since this case was exhibited the plaque on the forehead above the left eyebrow has begun to show, buried in the more uniform dusky red area, separate red-brown nodules suggestive of lupus nodules, and this appearance has still further inclined the exhibitor towards the diagnosis of lupus pernio. The case has given a positive von Pirquet reaction.

Dr. PRINGLE accepted Dr. Adamson's view that the patches on the abdomen were tubercular lupus, but regretted that he could not accept Dr. Adamson's diagnosis of the lesions on face as being true erythematous lupus. They might perhaps be early mycotic tumours as some Fellows suggested; but he was more inclined to regard them as examples of what had been described by Radcliffe-Crocker as the *nodular* type of erythematous lupus, which was in reality a distinctly tubercular disease. In two cases of this rather rare condition observed by himself and accepted as such by Radcliffe-Crocker, and in one recorded by Dr. Liddell, the tubercular nature of the disease had been established microscopically. A peculiar and confusing point about the condition was that it affected the same distribution as typical erythematous lupus, as did the lupus vulgaris erythematoides of Leloir. Dr. Pringle suggested the desirability of a biopsy or of a test tuberculin injection.

Case for Diagnosis.

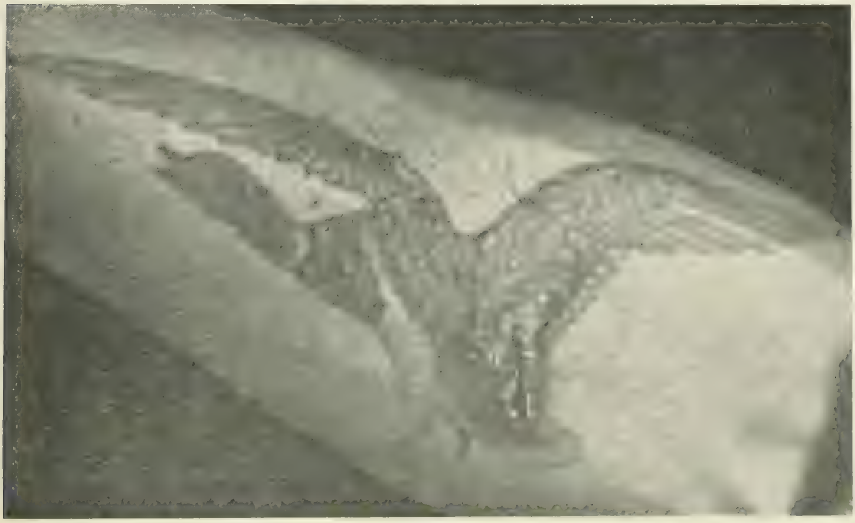
By T. P. BEDDOES, F.R.C.S.

THE patient was a middle-aged Italian, who had been in this country two years. Seven months ago the condition now seen appeared on one left toe; a month afterwards it appeared on the hand, and at the same time there had been lesions on the penis, and one week ago in the throat. A Wassermann reaction had not been done, and he had had no specific treatment. The toe was swollen, suggesting cellulitis, negatived by the limited area and the tint. There was a little enlargement of the inguinal glands on both sides. On the glans penis there was localized erythema, not indurated or raised. On the back of the hand an area with 1 in. diameter was red with an irregular edge: no scaling. On the right side of the soft palate were irregular bright red, not raised, isolated spots.

Case of (?) Tuberculous Infection of Tattoo Marks.

By S. E. DORE, M.D.

THE patient, a man aged 29, was seen in consultation with Mr. Percy Sargent. Five years ago he had been tattooed on both forearms. Three years later he went to the same operator to have the work touched up. About two months ago a small swelling appeared on the right tattoo mark which had gradually increased in size. It was now a flat, raised patch with a well-defined edge and soft to the touch, measuring 1 in. in length and ½ in. in breadth. There were also about a dozen small conical elevations, smaller than a pea in size and some having a slight central depression not unlike tuberculosis, which had appeared soon after the larger lesion, in the part which had been touched up, and three or four similar papular lesions of more recent occurrence in the mark on the left forearm. Owing chiefly to the history, a tentative diagnosis of keloid had been made at first and X-ray treatment suggested with free excision as an alternative.



? Tuberculous infection of tattoo marks.

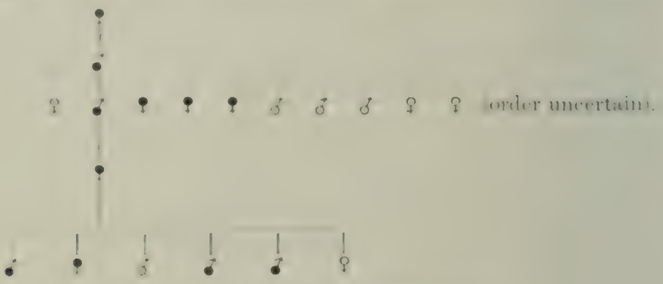
Five Cases of Epidermolysis Bullosa.

By H. MacCormac, M.B.

The patients exhibited, a mother and four children, were sent to the Middlesex Hospital by the school medical officer. The mother was quite familiar with the course of the disease, as she could trace it through five generations (*see* Table).

In all cases the affection commenced in infancy, tending to become less marked in adult life. Both sexes were involved; the family history is good, although the mother has a cured lupus vulgaris.

The complaint becomes most troublesome during the summer months, large blisters appearing where any pressure has been applied, the feet being especially involved, a condition resembling a severe dysidrosis resulting. A tight collar or garter will cause a bulla to appear in from twelve to twenty-four hours, preceded by some tingling and pain. Nikolsky's sign is not present. The bullæ leave no scars. No epidermic cysts are present, and the nails have not been involved. A blood examination in two cases showed a perfectly normal condition. There was no eosinophilia.



Pedigree of cases of epidermolysis bullosa.

Pigmented Tropical Skin, with Multiple Epitheliomata of the Rodent Type, also with Barcoo Rot, in a Man, aged 53.

By Sir Malcolm Morris, K.C.V.O., and S. E. Dore, M.D.

Family history: Father and mother were healthy. Four brothers and two sisters are alive and enjoy good health. There is no history of any skin disease in the family.

Personal history: The patient has always been healthy with the exception of a severe attack of influenza. His mother noticed that he had a particularly white thin skin.

History of present condition: The patient went to West Australia thirty years ago. Soon after his arrival he became intensely sunburnt, and frequently had blisters on his neck and was obliged to wear a chamois leather cap over his nose. His face was protected by a large broad-brimmed hat. He often went out in a singlet only in order to acclimatize himself to the sun. About eight years ago small warty growths began to appear on his face and arms. These began like inflamed warts and ended in ulcers. Some of them disappeared as the result of the application of silver nitrate, but in some cases this treatment seemed only to irritate the growth. Six of these tumours were excised from the neck and face, one large tumour on the side of the neck being grafted. After microscopical examination the tumours were said to be rodent ulcers. The patient attributed several small scars on his arms to Barcoo rot, a condition well known in Australia, and apparently due to abrasions of the skin followed by septic infection. These never ulcerated, and he considers them quite distinct from the ulcers on the face and neck. X-rays were tried in Perth without any good result.

Present condition: The skin of the neck, shoulders and upper part of the chest and back was deeply pigmented, the pigmentation chiefly consisting of small closely aggregated macules, some of which were darker than others. Around the neck the skin was not only mottled from a fine pigmentation but rugose and somewhat thickened. There were several congenital pigmented moles on the abdomen and back. The forearms were also deeply pigmented, especially on the extensor surfaces, and covered with long hair, the pigmentation ending abruptly just below the elbows and at the distal ends of the metacarpal bones. On the face there were several large, smooth scars where the tumours had been excised, and on the forearms numerous thickened, warty patches, also leaving scars.

Case of Pigmented Tropical Skin with Multiple Epitheliomata.

By Sir MALCOLM MORRIS, K.C.V.O.

A SECTION was exhibited under the microscope by Dr. MacCormac. The disease was something like the veldt sore of South Africa, and he had seen a somewhat similar condition in the skin of labourers in this country. Dr. Ernest Black had written an account of the condition, and it was mentioned in the book by Castellani and Chalmers, the statement made being that it was a streptococcic infection, though of what form was not clear.

DISCUSSION.

Dr. ADAMSON regarded the case as an example of the disease met with on the face and hands in elderly persons whose occupation exposed them to sun and light—the affection known as "keratosis senilis," or by the name given to it by Unna—viz., "sailor's skin." He did not agree that the warty ulcerating growths were true rodent ulcers. They differed in appearance; in the fact that, unlike rodent ulcer, they arose upon a previously damaged skin; in that their distribution depended upon the distribution of the original damage—they were present, for instance, upon the hands; and above all, in that they were liable to become carcinomatous and infect the glands, &c. Microscopically, they were sometimes squamous cell epitheliomata; sometimes, as in the present case, basal cell epitheliomata. Usually these basal cell epitheliomata showed also some prickle cells, cell nests, and a tendency to invade, break through the palisade layer and invade the lymphatic spaces. In the sections under the microscope these features were but little marked and he admitted the close resemblance to rodent ulcer, but would prefer to describe it as a basal cell epithelioma.

Dr. MacLeod said that it was possible that the chronic dermatitis due to the actinic rays of sunlight might predispose to "Barcoo rot," but the condition was extremely like veldt sore, and was doubtless a microbic infection. From the latter condition Bishop Harman had isolated a diplococcus, which he did not believe to be an attenuated form of *Staphylococcus aureus*.

Dr. Wilfrid Fox said he did not think acquired Kaposi's disease was very uncommon; he had seen two cases in one family, who had lived in Honolulu. Both acquired it in adult life. They were ladies, aged 35 and 38 respectively.

Dr. Bolam said he had seen an analogous condition in three or four paraffin workers; they got a similar pigmentation, with epithelial growths of the same microscopical characters as in this case, and there were also sores which healed readily under suitable treatment.

Further Report on a Case for Diagnosis.[1]

By Sir Malcolm Morris, K.C.V.O.

The case when previously shown was thought by several members to be mycosis fungoides, and further investigation had shown that they were right, for Dr. Whitfield had examined the growth microscopically and found it was not sarcoma. He now showed the case to indicate the benefit which had been derived from X-ray treatment, for the mycotic changes had practically disappeared.

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[1] Shown at the meeting on May 22; see *Proceedings*, pp. 145-8.

Case of Acute Lichen Planus treated by Lumbar Puncture.

By G. PERNET, M.D.

THE patient was a woman, aged 52, who attended at the West London Hospital on June 24 with an acute lichen planus which had been present three weeks. It was spreading very quickly and the pruritus and irritation were extreme. He had brought the case to show the result of treatment. She was admitted and he asked the house physician, Mr. Hammond, to do lumbar puncture. At 11 o'clock next morning this was done, 7½ c.c. of cerebrospinal fluid being withdrawn. From that time the pruritus began to yield, and by 4 o'clock the same day it had practically ceased. No other treatment. No pruritus since, and the condition was now involuting. She was given mist. sacchari usti. The cerebrospinal fluid did not show lymphocytosis, but, as is usual, it reduced Fehling. Two years ago he had had a similar acute case in private in a male patient, but there were difficulties about the patient entering a nursing home to have it done. But the result of lumbar puncture was good. In this case the cerebrospinal fluid did not reduce Fehling. In a case in which the puncture was done and the patient allowed to go home very severe headache ensued and lasted five or six days; and he concluded it was far better to have the patient under observation. The sooner the puncture was done for acute lichen planus the better the result *qua* pruritus. Ravaut, of Paris, had worked at this subject for three years, and his published writings should be consulted for further details.

Case of Acne Varioliformis.

By G. PERNET, M.D.

THE patient, a man, aged 70, was now almost well as a result of treatment. He had first attended the West London Hospital for an eruption occupying the upper part of the trunk, in front extending below the transverse nipple line. The outbreak was acute and the characteristic lesions closely aggregated. On the scalp the lesions were older. No local application was ordered, but simply mist. ferri perchlor. t.d., and a week later the eruption had cleared up.

Case of Mycosis Fungoides.

By J. J. Pringle, M.B.

This was a well-marked example of mycosis fungoides in a male patient, aged 41, by occupation a warehouseman, and of pure English stock, who had come under observation in the Middlesex Hospital Skin Wards on June 24.

History: One brother had died of tuberculosis, but there was nothing either in his family or personal history to throw any light upon the ætiology of the case. The eruption apparently showed itself about four and a half years ago in the form of insignificant rough and slightly irritable patches on the thighs and forearms. No importance was attached to these by a medical man who had examined him for life insurance. Soon afterwards a red rash appeared in the same localities; this itched considerably and a red swollen patch appeared on the dorsum of the left foot, which "broke and discharged." Three and a half years ago red and hard lumps came out on the penis, which soon broke down, leaving deep ulcers. He was treated for syphilis for a year with mercurial pills. During this time his skin was being gradually invaded, becoming "thickened, lumpy and breaking down" over wide areas. For two years previously and up to his admission to the Middlesex Hospital he had been attending as an out-patient at a skin hospital; he states that his disease was there called lichen hypertrophicus and the only treatment adopted was by arsenic internally, not apparently in large or increasing doses. His general health had only been impaired in the last six months; he had, indeed, been able to follow his work and said he "only felt 'rather weak'"!

Present condition: As the general characters of the eruption were typical and familiar to all Members of the Section no minute verbal description was necessary or desirable. The accompanying schemata roughly indicated the distribution of the disease in all its stages:—

(1) Variously sized patches of well-defined erythrodermia and scaling, roughly circular in outline.

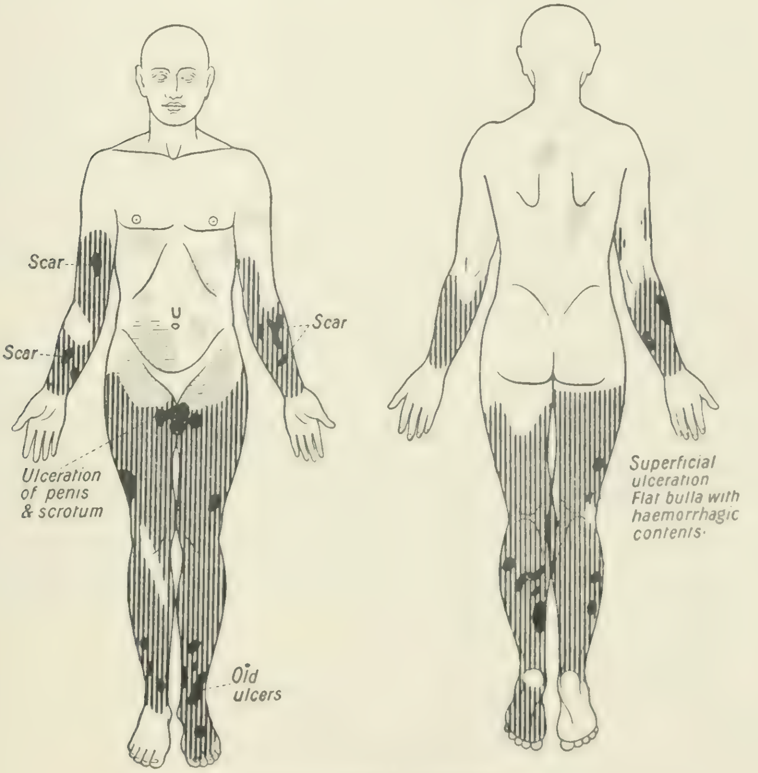
(2) Huge areas of soft doughy infiltration with very coarse lichenification.

(3) Large masses of tumours arising from infiltrated skin.

(4) Very numerous large ulcers resulting from the necrosis of growths and of the rupture of *large hæmorrhagic bullæ* which were

very abundant, and constituted one of the most prominent and the only unusual clinical feature of the case. Numerous deep white scars are also indicated in black.

The spleen was palpably enlarged, as also the inguinal glands, but there were no other lymphatic enlargements. Repeated blood examinations showed no tangible deviation from the normal; there was no anæmia, no leucocytosis, and no eosinophilia. Blood cultures were sterile. Examination of the urine revealed no abnormality. The Wassermann reaction was negative.


Scar
Scar
Scar
Ulceration of penis & scrotum
Old ulcers
Superficial ulceration
Flat bulla with haemorrhagic contents

Case of mycosis fungoides.

☐ (1) Premycosic erythrodermia.

▥ (2) Various stages of infiltration and lichenification.

☐ (3) Tumour formation.

■ (4) Ulcers and scars.

Dr. Henry MacCormac epitomized the microscopical appearances of excised portions of skin as follows:—

“(I) Premycotic Lesion.

“(1) *Low Power.*—The epithelium is seen to be intact; in many places it exhibits a considerable œdema, while here and there collections of cells have strayed into the Malpighian layer. The round cell infiltration is confined very sharply to the papillary and sub-papillary layer.

“(2) *High Power.*—A dilatation of the vessels and lymphatics of the papillary and sub-papillary layers is to be seen. The cells forming the infiltration are of no very definite type, but rather characterized by multiformity. No giant cells are to be found.

“(II) Advanced Lesion.

“(1) *Low Power.*—The striking appearance is the dense infiltration of the skin with ‘round cells.’ No very marked alteration of the epidermis except an almost complete disappearance of the papillæ can be detected. Scattered throughout the deeper parts of the infiltration are numbers of giant cells.

“(2) *High Power.*—The epidermis is seen to be reduced to a thin rind, with an almost complete absence of papillæ. Well-marked prickles can be observed in the Malpighian layer. The infiltration presents cells of a very multiform aspect. Besides those of a round form there are many with an angular, oval, or irregular outline. The giant cells are of rather irregular outline and formation, and in many cases have become fused together. There is a proliferation of the endothelium of the smaller blood-vessels. Some of the larger arterioles show distinctly thickened walls.”

Dr. Henry Beckton reported that Altmann's granules were present in great abundance in all cells composing the growth. This fact, according to Dr. Beckton's researches, demonstrated the non-malignant nature of the tumours.

The principal method of treatment employed had been prolonged immersion in warm starch-boric-cyllin baths, under which a large number of the ulcers had healed with surprising rapidity and kindliness. In the intervals the ulcers were treated with diluted boric ointment. Pastille doses of X-rays were being applied to the prominent tumours, which had certainly become softer, less prominent, and less well defined since admission. The patient had also received a full intravenous injection of salvarsan without appreciable effect, either local or general.

Case of Urticaria Pigmentosa.

By H. C. Semon, M.D.

The patient was a child, aged 2, and eighteen months ago spots first appeared on the back, and later spread round to the front, and were now present on the neck. When scratched they showed factitious urticaria. A similar case was shown by Dr. Meachen in March last.[1] There was nothing in the history to throw any light on the ætiology, and the child was in all other respects well nourished and healthy. The case belonged to the macular type, and there were no nodules noted at any time. On diascopy of the round, brownish, slightly infiltrated macules slight staining was manifest. The lesions were not irritable.

Case for Diagnosis—Parapsoriasis.

By W. Knowsley Sibley, M.D.

A fairly healthy looking girl, aged 17, who for at least ten years has suffered from a more or less persistent eruption over many parts of the body. There is nothing of interest in the family history. She had a slight presystolic cardiac bruit and some displacement of the apex beat, and shows evidence of a chilblain circulation, with rather cold and slightly cyanosed fingers and hands. She suffers from pernio on the dorsum of the fingers in cold weather.

The eruption first appeared on the legs and knees, and now is most abundant on the forearms, dorsum of hands, on the thighs and legs, present over the olecranon processes and patellæ, and slightly on the face, especially in the left eyebrow. A few scattered lesions are present on the neck, and abundant circumscribed, sharply defined patches are present on the sides over the lower ribs and costal cartilages, both anteriorly and posteriorly. They have only appeared on the trunk this year; formerly they were confined to the limbs. At one time the lesions appeared in a line on the dorsum of the hand after a pin scratch.

The eruption consists of scattered, variously sized, scarcely elevated plaques, which are mostly well defined and of a rose-red colour, except those on the forearms and hands, which often have a blue or violaceous tint, the surface in some places slightly scaly; in others desquamation is absent, and the surface has a somewhat marked and reticulated appearance. The palms are free from eruption, but it is slightly present in the soles.

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[1] *Proceedings*, p. 120.

The finger-nails are markedly glistening and pitted in several places. The teeth are well preserved, and the mucous membranes normal. Symptoms are completely absent; the evolution has been very slow and the eruption has been most rebellious to treatment. Some eight months ago I had her in the hospital, and considerable improvement was obtained by Dowsing's radiant heat baths and chrysarobin ointment (4 per cent.), but she soon relapsed after leaving the hospital.

The patient says the eruption is generally most abundant in the spring, and does not differ much in summer or winter.

Histological report of a section of a lesion taken from the dorsum of the right hand in June, 1912:—

(1) Stratum corneum shows thickening, with a fine granular appearance in the hair-follicles; no horny cells can be seen.

(2) Hair-follicles are dilated and filled with horny material.

(3) Stratum granulosum is thickened.

(4) Stratum mucosum shows a slight thickening in places, and there appears to be an œdematous condition of the prickle cells.

(5) The papillary spaces appear to be dilated and also their blood-vessels.

(6) Blood-vessels in the dermis show slight dilatation, and are surrounded with round and spindle-shaped cells, and there appears to be some œdema of the connective tissue.

Parapsoriasis, lichen variegatus (Crocker), psoriasiform and lichenoid exanthem. Dermatoses psoriasiformes (Jadassohn), resistant maculo-papular scaly erythrodermias (Colcott-Fox and Macleod). Erythrodermie pityriasique en plaques disseminées (Brocq).

Brocq divides cases into three groups:—

(1) Parapsoriasis guttata—like psoriasis.

(2) Parapsoriasis lichenoides—intermediate between psoriasis and lichen.

(3) Parapsoriasis in patches—like seborrhœic psoriasis.

The present case reveals features of all three groups: (1) The marked distribution of the lesion over the elbows and knees, and a few weeks ago of a typical guttate psoriasis over the abdomen and back; (2) many lesions, especially those on the forearm and hands, are intermediate between psoriasis and lichen; and (3) the lesion on the left eyebrow is one of seborrhœic psoriasis.

I suggest that this case shows (and she has some seborrhœa capitis in addition) a fourth variety, one of an erythema, especially marked in the lesions on the dorsum of some of the fingers, which at times resemble those of lupus erythematosus, and which have left some superficial scarring, and are the seat of lesions of a chilblain nature during the cold weather.

Dr. ADAMSON considered the case one of lichen planus of the annular type. There were everywhere typical flat-topped angular papules, and here and there atrophic patches and pigment stains from faded lesions.

Case of Multiple Lupus Vulgaris treated with CO_2 Snow and Zinc Ionization.

By W. KNOWSLEY SIBLEY, M.D.

THE patient was a boy, aged 9, who had had thirteen patches of lupus which came on after an attack of measles, occurring in March, 1909. The first lesions appeared on the cheeks, then on the trunk, buttock, back of thighs, front of the right leg, the dorsum of the left wrist and elbow.

On June 15, 1911, under a general anæsthetic, a circular stick of solid CO_2, measuring 2 in. in diameter, was applied for two and a half minutes with firm pressure to a lesion of this size on the posterior of the right thigh and two smaller ones to the lesion on the left cheek for two minutes. On subsequent occasions for similar or lesser time solid CO_2 was applied to some of the other smaller lesions. The scar left on the thigh is seen to be a fine and regular one, measuring some 1¾ in. in diameter, and with the possible exception of a minute granuloma left at one spot in the margin the disease was cured by this one application, and this lesion has not been treated since.

Three or four other quite pale, almost invisible scars are seen over the lumbar region, where also one application of the CO_2 has eradicated the disease.

Similar scars are present on the thorax and abdomen, and one over the dorsum of the left wrist, which was treated in two areas of one minute each.

The lesion on the anterior of the right leg has been treated by zinc ionization, and shows a very satisfactory pale scar in the central regions, with a few semi-quiescent granulomata at the periphery, which are now being treated by application of a mixture of acetone and CO_2.

Dr. DORE asked how many cases Dr. Sibley had treated in this way. He had himself been disappointed with the use of the snow in lupus vulgaris. He had seen cases in which severe applications had caused necrosis of the tissues and destruction of some of the lupus tissue, but such heroic treatment seemed uncalled for, and it seemed much better to excise the patches. He would like to hear whether in all the cases of the condition so treated by Dr. Sibley, similar smooth scars had resulted.

Case of Dermatitis Herpetiformis.

By D. KING-SMITH, M.D. (Toronto).

HISTORY: Mr. J. B., aged 34, born in England; duration seven years. Seven years ago patient felt a tenderness of the mouth, especially so on the taking of hot drinks and food. In a short time blisters appeared in mouth, leaving a raw condition. About four months after large blisters appeared on various parts of body, some being of the size of an orange. During the outbreak of bullæ patient lost many pounds in weight and was quite prostrated. The attack gradually subsided and he regained his lost weight and felt in fairly good condition, but soon an attack similar to the first one appeared.

Three years ago he came under my observation. He was then bedridden, and had been so for several weeks.

On examination, mouth showed a macerated appearance of epithelium, many denuded areas and exfoliations. There was great salivation, so much so that patient lay with his head hanging over edge of bed so that saliva could run freely away. There was marked pyorrhœa. The body presented in axillæ, umbilicus and groins vegetating lesions, giving the appearance as if they had spread from a centre outwards, leaving in their tract a marked pigmentation. Scattered here and there were many pustules, which seemed to be the beginning of the vegetations. Urinalysis negative. Blood examination: Slight increase in eosinophiles, not marked. Otherwise examination was normal. Pus from pustule showed ordinary *Staphylococcus albus*. Scrapings from vegetations did not reveal any fungi. Wassermann was negative, and from inquiry patient had been given anti-luetic treatment without any benefit.

By continuous irrigation of mouth with weak permanganate solution and removal of teeth the buccal condition improved markedly. Patient was soon able to take nourishment and gained rapidly in weight. The vegetations never entirely disappeared, although much less at certain periods. During past three years he had a number of attacks, but none quite as severe as when I first saw him.

The diagnosis of *Pemphigus vegetans* was made.

Vaccine therapy was tried. No improvement noticed.

DISCUSSION.

Dr. SEQUEIRA said he had had a similar case in a woman who had been sent to him at the London Hospital by Dr. Cursham Corner. He thought it was generally recognized that there was a variety of dermatitis herpetiformis with vegetative lesions. The prognosis in such cases was not so serious as in pemphigus vegetans proper.

Dr. PRINGLE remarked that the interrupted course of the case exhibited with marked periods of comparatively good health scarcely accorded with the accepted or classical notions as to pemphigus vegetans, which was generally a rapidly progressive and fatal disease. That, at least, was his experience of all three typical cases he had personally attended. He had, however, at the present time under his care in hospital a case which exemplified the now generally acknowledged fact that the various pemphigoid conditions might merge clinically one into another. This patient was a middle-aged Jewish woman who had suffered for years from attacks of typical dermatitis herpetiformis, but within the last year she had developed lesions in the mouth, about the vulva, in the groins, and in the axillæ, exactly like the patient exhibited, and the objective resemblance to pemphigus vegetans was most striking. The condition had cleared up to a surprising extent under prolonged antiseptic baths in January of the present year, but had relapsed in the unhygienic conditions of her home after discharge from hospital. She was now in hospital again for a severe return to her previous condition and was making satisfactory progress again under the same treatment as before, and this in spite of the fact that the disease was unfortunately complicated by pernicious anæmia.

Case of Lupus Erythematosus.

By A. WINKELRIED WILLIAMS, M.B.

THE case was one of skin lesions on nose and ears—typical lupus erythematosus—but the eruption inside nose was rather suggestive of tubercular lupus. A patch on the nose was treated with 5 per cent. tuberculin ointment. In twenty-four hours there was no reaction; in forty-eight hours there was slight but distinct reaction. The patch was more raised, redder, and had an areola, round which showed slight vesication. The same jar of ointment had been used on a typical lupus vulgaris, which reacted in twenty-four hours distinctly and had a most intense reaction in forty-eight hours; a non-tubercular case of sebaceous hypertrophy showed no reaction with the same jar in forty-eight hours.

Case for Diagnosis.

By A. Winkelried Williams, M.B.

Patient, a young lady, secretary to a medical man, had a hairy nævus on loins at birth. It was now strangely altered; she had been seen by Sir Cooper Perry and Dr. Radcliffe-Crocker in the past, and from her account no very definite diagnosis was made; she had been treated by high-frequency current and X-rays.

Present condition: An irregular, somewhat lumpy patch, which crossed the middle line, measuring in its longest transverse diameter 21 cm., and 15 cm. in longest vertical line. The hair is now limited to borders of patch. It varies from intense white to reddish-brown and in few areas dark brown colour. It is irregularly raised above skin level, but by sense of touch it can be detected as extending much more deeply in the skin. It is arranged in irregularly circular lobules. In centre there is a hard, white, sclerodermia-like patch, depressed below general level and surrounded with a pinkish-brown raised border. Under diascope a moderate amount of brown pigment, collected in some places in more intense masses, is seen. To the extreme left a dome-shaped, raised mass, about size of a shilling, firm to the touch and separated by normal skin from the rest of the growth.

It has of recent years become painful at times; the pain varies in intensity and is shooting in character; the patient always has irritation and has got into habit of knocking it with her fist. During the past six months it has extended considerably—i.e., at least 5 cm. more to the right.

Dr. Williams felt very anxious about the case, as he feared it was developing a malignant phase.

Dr. Adamson said he had shown a case almost exactly similar, of which a photograph appeared in the *Proceedings*[1]—"pigmented vascular sclerosing nævus."

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[1] *Proceedings*, 1911, iv, p. 98; *Brit. Journ. Derm.*, 1911, xxiii, p. 179.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE SIXTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1912-13

ELECTRO-THERAPEUTICAL SECTION

LONDON
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ELECTRO-THERAPEUTICAL SECTION.

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Electro-Therapeutical Section.

October 18, 1912.

Dr. Reginald Morton, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Arthritis.

In reviewing the past work of this Section and its parent—the British Electro-therapeutic Society—it is noticeable how few papers have had any reference to the subject of arthritis. There would seem to be a danger of neglecting these less "dramatic" diseases in our study of the application of radiology to internal medicine and surgery. The excellent work done in these branches is worthy of all praise, and will go on developing and advancing for a long time to come, but the study of arthritis is not an unprofitable occupation for us. It presents itself in many phases and varieties, in the diagnosis of which the X-rays can be of great help, and our methods of treatment of much value in giving relief and occasionally a cure of the affection.

Arthritis in its many forms, though not possessing a high mortality, is one of the most distressing crippling conditions, and one that is widely distributed. It is found in all classes of society, but more in the less favoured ones—that is, those who are least able to afford it. It is thus a disease of great public importance, and it is for all these reasons that I venture to bring it forward on this occasion, and I hope in this way to stimulate a little more active interest in a subject that deserves it. To anyone devoting special study to the subject of arthritis, probably one of the first problems that will confront him will have to do with those two conditions known as rheumatoid arthritis and osteo-arthritis. If we consult various text-books we find most commonly that these two terms are used very indiscriminately and most often as synonyms of one and the same condition, or as different phases of the same disease. As we dip more deeply into the literature of the subject, especially into the most recent writings, we at once find ourselves in a field of active controversy where the arguments put forward by the different schools are so varied, and each so convincing in their own way, that it is difficult not to side with one or the other with a genuine enthusiasm.

The view likely to be adopted by any one of us will in all probability depend very much on previous training and inclinations. While nearly everyone will agree that rheumatoid arthritis is primarily due to a toxaemia, the neurologist will take the view that the effect on the joints is secondary to the influence of the toxin on the nervous system, and he would have very sound and substantial reasons for this attitude. The bacteriologist, on the other hand, would probably conclude that the effects on the joints were due to the direct action of the micro-organism or its toxin, the nervous symptoms being merely incidental though produced by the same agency. The pure clinician, if such now exists, would ignore these points and look upon osteo-arthritis as a chronic, or perhaps hypertrophic form of rheumatoid arthritis, and all of them mere phases of "rheumatism." Or again, we find an eminent pathologist in the person of Goldthwait, of Boston, making a classification as follows:—

(1) *Infectious Arthritis.* This term "is used to designate a joint disease resulting from the presence within the body of some infectious organism, the symptoms being due either to the presence of the organism itself within the joint, or to some toxin produced by that organism in some other part of the body." This group is, of course, a very wide one, and includes the more acute cases of rheumatoid arthritis as well as all those of Still's disease. He points out that in the early stages of the cases that come within this group there is active leucocytosis with a high percentage of hæmoglobin. Later on these conditions disappear and secondary anæmia develops.

(2) *Atrophic Arthritis.*—In this group he includes those cases that have a gradual onset and run a chronic course without any pyrexia. In the early stages we have the spindle joint, to be followed later by an atrophy of the whole of the peri-articular tissues as well as the synovial membrane, articular cartilage, and the ends of the bones. This class

includes practically all those cases that are usually described as rheumatoid arthritis.

(3) *Hypertrophic Arthritis.*—This group is characterized by hypertrophy of the cartilages and ends of the bones making up the affected joint, and is identical with what is usually called osteo-arthritis.

Such a classification as this has much to commend it, and coming from an authority of such high standing it is deserving of our serious consideration; but to many of us, I venture to say, it will not be altogether satisfying. For one thing, the first group is rather too comprehensive, in that it includes all forms of arthritis due to the action of perhaps many different kinds of micro-organisms; and further, it places the more acute cases of rheumatoid arthritis into this first group, while the less acute and more common variety is placed in the second group. I think it will be found that most observers are unwilling to consider these two conditions, which we term the acute and subacute varieties of rheumatoid arthritis, as if they were separate and distinct diseases.

One noteworthy feature of this classification, to be found also in that of Nathan, of New York, is that rheumatoid arthritis and osteo-arthritis are clearly differentiated from each other, both clinically and pathologically. Other classifications have been made by Přibram, Hoffa, and possibly some others, but in all these mentioned we find this tendency to treat osteo-arthritis quite separately from any other form. Garrod, in the second edition of Allbutt's "System of Medicine," frankly divides arthritis into two main classes, rheumatoid arthritis and osteo-arthritis, and the same arrangement is adopted by Llewellyn Jones, from whose writings I have derived both pleasure and profit, and to whose valuable work I wish to pay the highest tribute.

This classification into two main divisions of arthritis, which might be called the English classification, has not only the virtue of simplicity, but seems to be an eminently suitable one in the present state of our knowledge of the ætiology of arthritis. It would, of course, be very satisfactory if we could classify our cases according to the particular organism responsible for the mischief, and also separate a true rheumatoid from other forms of infectious arthritis; but as soon as we attempt to do this we find ourselves in difficulties through lack of accurate knowledge. While recognizing that different micro-organisms play their part in the forms of arthritis, the fact remains that so far all efforts to establish the constant association of one particular organism with a particular form of arthritis have met with only partial success. The different forms of arthritis glide into one another almost imperceptibly, and it is probably on this account there have been so many difficulties in nomenclature, leading to an apparent confusion between the writings of various authors. It is to the bacteriologist to whom we now look to lead us out of this Egyptian darkness, and it is not too much to hope that ere long our expectations will be realized. Though, as we have seen, the bacteriological evidence so far available is incomplete in places, there is enough at our disposal to justify the very general belief that these diseases are primarily due to the presence of micro-organisms. After all, it is difficult to see how the changes observed in the diseased structures can be due to any but the action of a chemical substance which has a disintegrating action on articular and peri-articular tissues. That these changes are not due to inflammatory reaction is clearly shown by microscopical examination, and if we accept the theory that they are the result of the action of a chemical substance or toxin, it is a fair assumption that micro-organisms are responsible for their production. These ideas are more applicable to the rheumatoid variety than to osteo-arthritis; the latter condition is most frequently seen in subjects that are well nourished and not favourable to any form of bacterial invasion; but in spite of this, and looking at the matter in a broad way, I am disposed to believe the ultimate cause of osteo-arthritis will be proved to be of a bacterial origin.

Before leaving this part of the subject it should be noted that the bacilli described by Schuller as "short plump rods showing polar granulation" as the ones most frequently found in rheumatoid arthritis, are seldom found in the fluid within the affected joint, but much more commonly embedded in the fringes and tissues round about. This is of importance when we come to consider the question of treatment. There is another point that gives rise to differences of opinion. Do the bacteria invade the joint and produce the toxin in situ, or is the latter produced at some distant point and carried to the susceptible joints by the circulation? Here again we find authorities divided into opposite camps. If the bacillus as described by Schuller is the one responsible for rheumatoid arthritis, it would appear that the local production of the toxin was the correct view; but on this point we are not yet on certain ground, and as there is considerable evidence pointing to the production of the toxin at a distance, we cannot regard this as settled.

Let us now go into the question of the identity or otherwise of the two conditions, rheumatoid arthritis and osteo-arthritis, from other points of view; and while here, as in most other instances, it is impossible to lay down any hard and fast rules, I think it will be agreed that we have ample grounds for considering them as quite distinct diseases, having few points of similarity, and in many ways divergent. Indeed, we may go so far as to say that in typical cases the only points of resemblance are that they both attack joints and both cause deformity; and even here the deformities are of quite a different nature—atrophic in one, hypertrophic in the other. If we take a general view of rheumatoid arthritis we see that we have all the conditions necessary to make up a constitutional disease, with important local manifestations. It frequently runs a course that is quite typical of a toxæmia in which the nervous system takes part. At times the onset may be acute with fever and sweating: also vasomotor disturbances such as coldness of the fingers and local congestion. We may also find anæsthesia and loss of power. These symptoms are seen most frequently in young female adults whose constitutions have been enfeebled by influenza, puerperal troubles, pyorrhœa alveolaris, or other bacterial invasions of the digestive system and respiratory passages. Mental worry and anxiety are factors in the predisposition to the disease. Locally, we find that the small joints, notably those of the hands, are the favourite objects of attack, and in the fingers these take the spindle form of swelling that is so characteristic. These swellings, at first covered with a glossy skin, in time tend to diminish in size by atrophy. Nothing corresponding to Heberden's nodes are ever found in this disease.

In contradistinction to this picture let us examine the various points that go to make up the disease we term osteo-arthritis. Here we find it is the large joints that are most frequently attacked, and the subjects are for the most part well-nourished males of middle or advanced age. The onset is never acute, there is no pyrexia, no sweating, no vasomotor disturbances, no loss of power, or muscular wasting. The glossy skin is not seen over the swellings, which are of irregular form and tend to increase with time, becoming more irregular and more deformed. Heberden's nodes are a frequent accompaniment of the condition. In short, osteo-arthritis is really a joint disease with no constitutional symptoms of any importance.

Further differences are to be made out by means of radiography, notwithstanding the fact that most writers tend to discount the value of this method in the differential diagnosis. While it is admittedly difficult at times, especially in the early stages, to make a definite and accurate diagnosis by the X-rays alone, this is becoming easier with improvements in apparatus and technique, and in nearly every case it can give much valuable direct evidence not attainable in any other way. If, as has been claimed by some authorities, radiography cannot effect any saving of time in making a diagnosis over ordinary clinical methods—a claim with which I do not entirely agree—it can at least afford corroborative evidence at a very early stage, and it can demonstrate differences between the conditions known as rheumatoid arthritis and osteo-arthritis to such an extent as to make it difficult for anyone to believe they can be merely phases of one and the same pathological condition.

In the course of my investigations into this interesting question I was fortunate enough to come across a case of more or less general osteo-arthritis involving the small joints of the hands among other parts. I am thus in a position to show these two conditions affecting the same anatomical part, and this makes it much more easy to appreciate the essential differences between the two diseases. Fig. 1 is from the hand of a young female adult who had been under observation for about a year, suffering from rheumatoid arthritis. One of the first points to be detected by this method is the irregular narrowing of the spaces between the affected joints, which even in a comparatively recent case may have gone so far as to give the appearance of the articular ends being in actual contact. Here it is very clearly seen in the metacarpo-phalangeal and the first row of interphalangeal joints—the latter showing the outline of the spindle-shaped swelling, due to peri-articular thickening that is so characteristic of this disease. Erosion of the articular ends of the bones is well shown in some of the joints, notably the first interphalangeal joint of the forefinger and the distal ends of the fourth and fifth metacarpal bones. In addition to this we find, more or less constantly, signs of absorption of the cancellous tissue near the articular ends of the long bones, and in spongy bones such as the carpus. This is well shown at the carpus and adjacent ends of the metacarpus in another plate which I show you. Of course it is well known that a temporary suspension of function will bring about a certain amount of thinning of the trabeculæ, and while this is undoubtedly a factor here, I am convinced that in rheumatoid arthritis we get a degree of this not attainable by rest alone in the same time and in a healthy joint.

Speaking generally, the disease seems to have a preference for the site of an epiphysis, and in the case of those long bones having but one, such as the metacarpals, it is most active at this end. Similarly as regards the formation of osteophytes: these are rare in rheumatoid arthritis, but when present they are found close to the ends

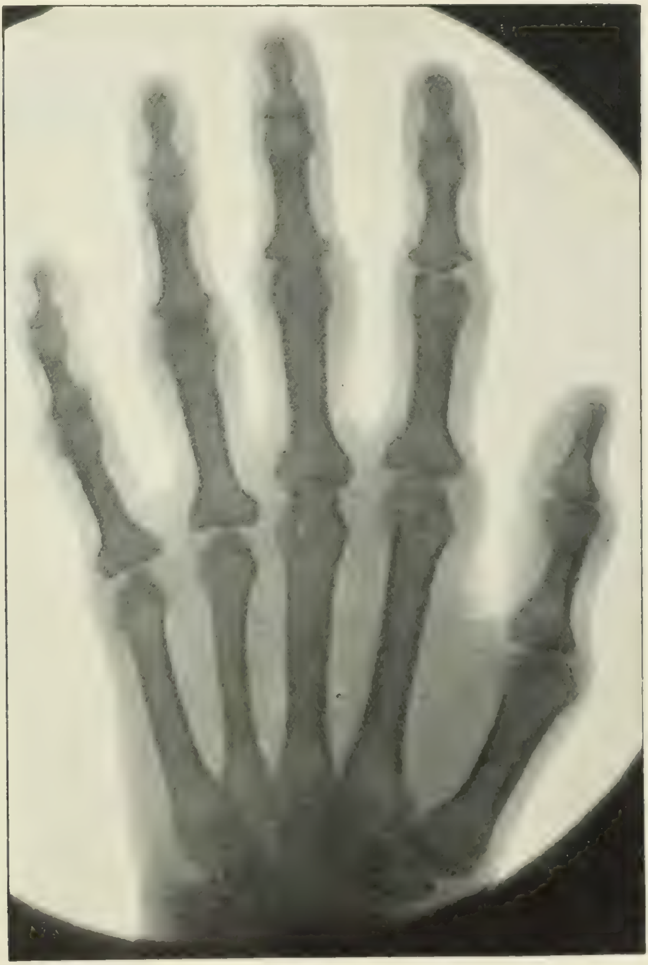


FIG. 1.

of the bones. The next plate is from the hand of a middle-aged man who has suffered from rheumatoid arthritis, for many years, in a chronic form. The absence of articulate cartilage is well seen, and osteophytes are at the extreme ends of long bones. Apart from this very occasional production of new bone on a very elementary scale it is quite evident that in rheumatoid arthritis we have a disease that is essentially destructive to joint tissues, and one that shows only the slightest tendency to attempt any sort of repair; and this is found only in mild cases that run a very chronic course.

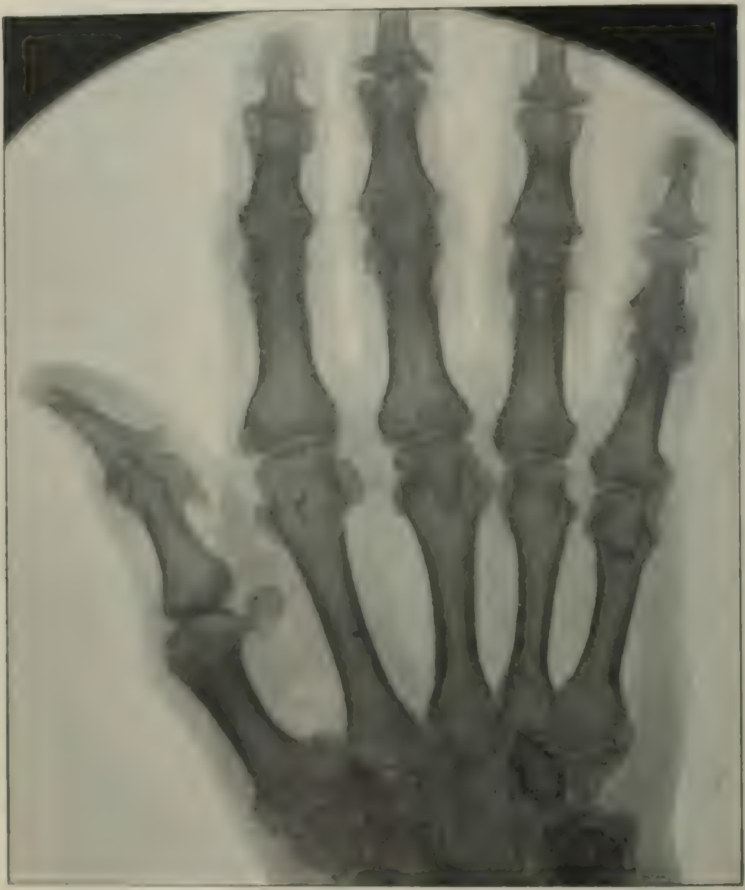


FIG. 2.

Fig. 2 is from the hand of a patient with osteo-arthritis of many years' standing. Here we see that while the joint spaces are narrowed, they are only partially so, and the spacing between the articular ends is more or less even. There is no erosion of the ends of the bones and there are no signs of absorption of the cancellous tissue. Of course, we know there are serious cartilaginous changes in osteo-arthritis, but they are in the nature of fibrillation and partial disintegration, and not carried away by absorption as in the rheumatoid form. The formation of osteophytes is an important feature of osteo-arthritis, and they are found not only at the articular borders but also along the shaft. To show these differences still more clearly I have prepared two slides by contact from the original negatives of figs. 1 and 2, showing the first interphalangeal joint of the first and second fingers. Fig. 3 is the rheumatoid case, and fig. 4 osteo-arthritis. From the examination of these plates it is reasonable to express the opinion that while both these diseases are destructive in character, we see more effort at repair in osteo-arthritis than in the rheumatoid form, and as evidence of this we have the active osteophytic process which tends to support the joint, notably in the case of the hip, the increased density of the bones, and the eburnation that takes place on the articular surfaces as the cartilage gets worn away. The destructive character of rheumatoid arthritis is also seen in the shoulder, where we have the high position of the head of the humerus due to the absorption of the articular cartilages and the subacromial bursa; thinning of the trabeculæ of the upper end of the humerus is also evident. I look upon the shoulder as being very susceptible to this form of infection.

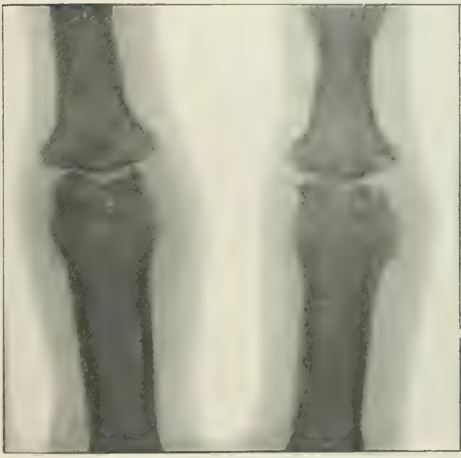


FIG. 3.

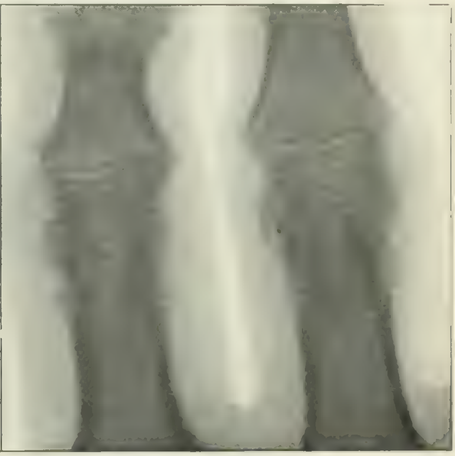


FIG. 4.

especially after injury. In figs. 5 and 6 are shown these diseases as they affect the knee, and on comparing these, which are taken from quite ordinary cases, it is difficult to understand how they could have been classed as different phases of the same disease. Fig. 6 shows the "lipping" at the edges of the articular surfaces that is so characteristic of osteo-arthritis. As the cartilage gets fibrillated and softened it is pushed out from between the bones and undergoes ossification. This is never seen in the rheumatoid form. I now show a plate from an old case of rheumatoid arthritis where the ligaments had suffered severely, and with the aid of pressure from the boot severe deformities have been produced. The other foot had suffered in the same way.

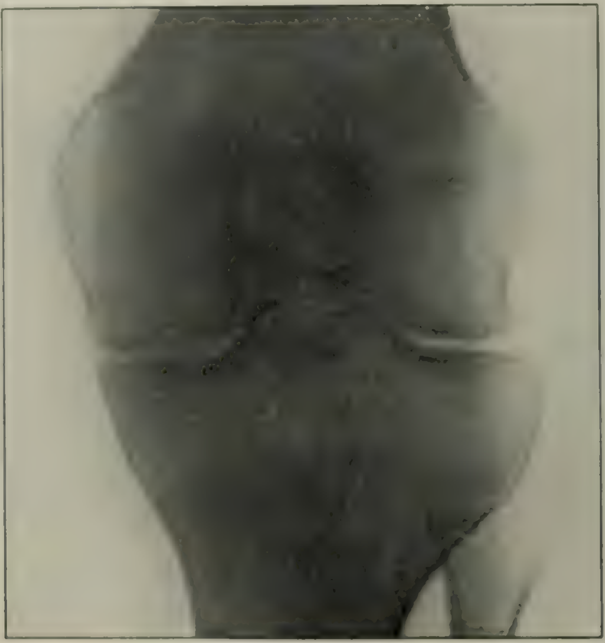


Fig. 5.

It is hardly necessary to pursue this part of the subject any further just now. Enough has been said to show what an interesting controversy is going on regarding the nature of the different forms of arthritis, particularly the two forms I have specially mentioned. I also hope I have succeeded in showing how much can be done by radiography in elucidating many doubtful points, even to the extent of making the distinction between two varieties formerly grouped as phases of the same condition so clear as to be quite unmistakable.

It now remains for us to show whether radiography can anticipate a clinical diagnosis, or at least corroborate it as soon as made. Up to the present most authorities have accorded to radiography a very secondary place as a diagnostic agent in arthritis, and this may have been deserved. I feel sure, however, that at the present time we are in a very much stronger position than ever before; we have better appliances, more skill, and more experience. So far as I can gather, cases of arthritis are submitted to X-ray examination more than they used to be. This is a significant feature as well as an evidence of an increasing confidence in the method. The changes in the early stages of some forms of arthritis may be unobservable by radiography, and when present may be so slight as to be easily missed, but errors are rare if care is taken and a comparison made with the sound side or a corresponding normal part. One source of confusion is the very similar appearances in gouty rheumatoid and arthritis. So far as the bone changes are concerned, in a paper by Koehler, of Wiesbaden, in

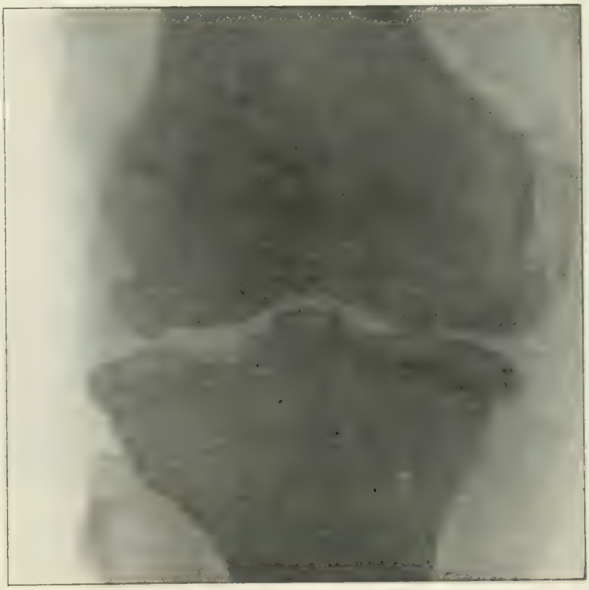
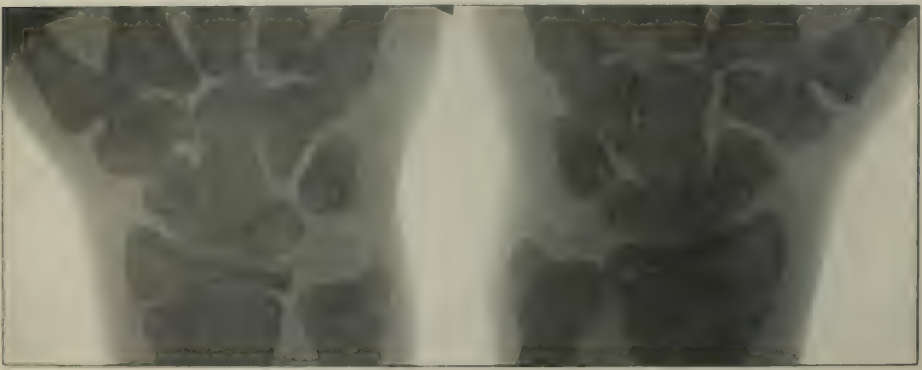


FIG. 6.

the February number of the *Archives of the Röntgen Ray*, the diagrams illustrating the changes in gout might easily pass for rheumatoid arthritis. But being aware of the similarity, and having regard to the history of the case, we are not likely to be led into any grievous error. In any form of arthritis it is important in the highest degree that an accurate diagnosis should be made at the earliest possible moment. This can only be assured if every method of removing uncertainty has been employed, and for this radiography is entitled to a recognized place. In support of this I may mention the following case. A boy, aged 14, was sent to me by Dr. Wingrave, suffering from pain in the right wrist. It was most noticeable when attempting to use the wrist for any sudden or twisting movement, and he was unable to play cricket or tennis on this account. It appears that some months previously he put out his hand to open a swing door, and at the same moment it was pushed against him by someone coming through from the other side. A sharp pain was felt at the time, which gradually subsided but never entirely disappeared. Massage, iodine and so on were used without any definite improvement, and in consequence of this an X-ray examination was advised. The two wrists are shown together in fig. 7. The two skiagrams were made at the same time and as nearly as possible under the same conditions. It will be observed that the right carpal bones do not come out so clearly as the left, and there is a distinct narrowing of some of the joint spaces around the os magnum, particularly on the side next the unciform bone. These changes are not unlike those seen in rheumatoid arthritis, and



Left. FIG. 7. Right.

if that was the correct diagnosis, the persistence of the symptoms is explained. It is generally admitted that all of us harbour tubercle bacilli at some time or other, but they cause no inconvenience until something happens to provide a nidus, such as an adherent pleura for instance. The same may be said of many other forms of bacteria, including those responsible for these varieties of arthritis. It is not unreasonable to suppose that here the injury to these carpal joints, though not very severe from the point of view as a casualty, was sufficient to provide the nidus necessary for the micro-organism to obtain a footing. However this may be, the case was dealt with on this basis, and I am glad to say there has been a very satisfactory improvement.

The lesson to be learnt from this case is an important one for all of us. The clinical signs were so indefinite that no clear diagnosis could be made. The first positive opinion that could be expressed was founded on the X-ray appearances, and improvement set in very quickly after the commencement of a line of treatment suggested by those appearances. Unfortunately we do not often see many cases at this early stage, and herein lies the difficulty of establishing our point, that all cases of doubtful joint mischief should be examined radiographically early, and at intervals if necessary, until the nature of the trouble is made clear. When this is done consistently there will be no doubt as to the value of radiography in the early recognition of arthritis, even to the extent of discriminating between the different varieties.

The subject of the treatment of arthritis generally, and especially of the rheumatoid form, is too vast to include in this address, but there are one or two points to which I would like to refer before I close. Believing as I do in the bacterial nature of most forms of arthritis, any treatment to be successful must have regard to this, and any site of bacterial invasion must be sought for and dealt with accordingly. I believe that vaccines are of value and that they will be more valuable as this branch of therapeutics is developed. I know that the system has its detractors, and no doubt mistakes have been made, but we all know that sneering at the early errors of any new form of diagnosis or treatment is a very economical form of self-indulgence.

Of the methods practised by ourselves, the one I have the most faith in is ionization, and a solution of iodide of lithium the most useful, though the salicin ion is probably equally good. I do not believe in ionization through solution as in a Schnee bath, since the current appears to enter almost entirely at the surface of the liquid. Pads made of a dozen or more thicknesses of lint soaked in a 2 per cent. solution are the best, and each joint, or group of small joints such as the carpus or fingers, should be treated individually, one pole on each side, and alternated at each sitting. Lithium iodide is a very deliquescent salt and difficult to keep dry after the bottle is once opened. I now get it in hermetically sealed tubes containing 100 gr. One of these dissolved in 10 oz. of hot water gives a solution of approximately 2 per cent. strength. Large currents are preferable to small ones, but what I consider to be most important of all is that the application should be long—not less than an hour at each sitting—and that they should be given frequently—daily if practicable. I consider this necessary because the ions do not penetrate the tissues very rapidly, and many of them are washed away by the circulation. Though it has been difficult to carry out these ideas in many cases, where I have been able to do so the results have been distinctly better.

If the bacillus of Schuller is the one responsible for rheumatoid arthritis as is probably the case, from the fact of its being buried in the tissues, it is not enough to get a few ions through the skin; we must endeavour to suffuse the joint with ions, and thus render the site unsuitable to the development of the organism, as well as to stimulate the tissues to increased activity and resistance. The use of high frequency currents, as ordinarily understood, is to all intents and purposes, futile. I am hoping that diathermy may yet prove of some benefit here, but so far as we can judge at present, it looks as if surgery would benefit by diathermy more than medicine.

DISCUSSION.

Dr. IRONSIDE BRUCE said he was glad that the President had chosen arthritis as the subject of his address. The pathology of the associated diseases—osteo-arthritis, gout, and true rheumatoid arthritis—seemed at the present time to be rather confused, and the corresponding appearance of these diseases on X-ray examination not at all definite. Dr. Strangeways, dealing with this subject, went into the differential diagnosis of these conditions by X-ray examination, and concluded that it was a very difficult matter to distinguish one from the other. One point was mentioned by the President—namely, the obliteration of the joint spaces—but he (the speaker) had found that he had difficulty in deciding whether flexion of the joint had not to do with this appearance rather than destruction of the cartilages.

Dr. G. B. BATTEN desired to thank the President for his historical review, as he was himself one of the original members of the British Electro-Therapeutic Society. He was very pleased to hear Dr. Morton's contribution on the subject of arthritis, especially as relatives of his had suffered from various forms of rheumatic affections, and had been treated by vaccines and ionization with good results. With regard to the differentiation between rheumatoid arthritis and osteo-arthritis, he remembered the case of a lady who, he did not doubt after seeing the photographs, had rheumatoid arthritis. Medical men who had seen it clinically differed in their views from one another. For two years the lady was so crippled with the condition that she had to be carried up and down stairs. Her fingers and toes were drawn up, and she did not go six weeks without having either an inflamed bunion or tonsillitis. Recognizing that it was toxæmia, he had her teeth taken out, after Mr. Goadby had prepared a vaccine from them. That vaccine was used in September, 1910, and continued regularly for eighteen months at increasing intervals. The following July she walked 10 miles, and she had increased 2 st. in weight. She had had no illness since. The skiagrams were the same kind as those shown, with absorption of cartilages and thinning of bone. Skiagrams of her hands a year afterwards showed only a slightly increased density of bone. There was no other change in the bones, though her hands now were quite supple.

The PRESIDENT (Dr. Reginald Morton), in reply, said that with regard to the differentiation of the varieties of arthritis, he fully recognized that there were cases in which no dogmatic statement could be made; there were cases in which there was a mixed infection, and one form might be merged gradually into another. The cases which he had depicted that evening were ordinary cases, the only exception being the deformed toes, which were included as curiosities. In the case of osteo-arthritis of the hands, which had existed for some years, the patient could write and do various things notwithstanding, and could also put the hands down flat. The skiagrams were not made with the hands in a position of flexion. When making a contribution he liked to take up a strong position; it encouraged attack and helped the ultimate arrival at the truth. In reference to Dr. Batten's remarks, he had come across cases of long standing where a diagnosis was made by X-rays, whereas, clinically, one could not say whether it was one variety or another. He was not clear about the early stages of the disease, but when it was well marked it was easy to establish the academical point that osteo-arthritis was distinct and separate from rheumatoid arthritis. He was glad to hear Dr. Batten speak of the success of vaccine treatment, in which he had great faith. One should abstain from condemning the method if it failed in some cases; much had yet to be learned in regard to it, but he felt sure it was a valuable treatment.

Electro-Therapeutical Section.

November 15, 1912.

Dr. REGINALD MORTON, President of the Section, in the Chair.

Electrical Accidents from the Clinical and Forensic Standpoint.

By Dr. S. JELLINEK (Vienna).

THE accidental contact with a wire carrying an electric current may be followed by serious consequences. The principal condition which determines the danger is, of course, the voltage, but this is by no means the only factor. In some cases a wire at 20,000 volts or more has been touched without any disturbance beyond some local injury, whilst in other cases contact with a wire at only 100 volts, or even less, has been followed by instant death. The fact is, that the degree of injury depends on a number of contributing circumstances. These we may call the *conditions of accident.*

These conditions of accident fall under two heads—the *external* conditions of the electrical installation, and the special or individual conditions which are peculiar to the victim. Under the former we may include voltage, amperage, and the phases and character of the current; while under the latter we include the electrical resistance of the victim's body, his physical and psychical condition, and the path of the current. In pathological experiments we have also to take into account the species of the animal under examination. Each of these factors plays a rôle of more or less importance, which can only be determined by an analysis of the mechanism of the accident, and with due consideration of the disturbance of function.

The most important factor is the voltage. If the electrical tension is below a certain minimal value, the danger may be regarded as *nil.* What is the exact minimum is still a matter of dispute. The point at which the voltage begins to be dangerous is usually fixed at 300 volts for an alternating current, and 500 volts for a constant current. Our experience shows that the danger-point should be fixed very much lower.

We have ourselves seen several fatal cases with an electrical tension of only 110 volts; and a fatal accident is recorded from touching a wire carrying only 65 volts. This was the case of a workman who was removing the crust from a steam-boiler, and who came into contact with an electric lamp. It is therefore a safe rule to regard every electrical installation as a possible source of danger.

With regard to voltage, it is interesting to note that there is an upper as well as a lower limit of danger—the lower one a minimum of a few volts, and the upper a maximum of many thousand volts, as in the high-frequency currents from a Tesla transformer.

The next factor of importance is that of amperage. Here also there is no danger unless the intensity of the current is above a certain minimum. This minimum is also a matter of dispute. It is the general opinion of electricians that a man can bear $\frac{1}{10}$ ampere without fatal results. This, too, is contrary to our experience. We have seen a number of fatal accidents with an amperage much less than $\frac{1}{10}$ ampere. On the other hand, cases are recorded in which a man has taken 1 ampere or more without serious consequences. The result, in reality, depends on the combination of these two factors—voltage and amperage—and on the physical and psychical conditions of the victim.

An accident is usually caused by the victim touching one pole of the circuit, since most electrical installations are earthed, and the circuit is completed through the victim's body if his feet or other parts are in good contact with earth. With a low voltage, however, an accident can only occur if both poles are in contact with the victim.

With regard to the different varieties of current, it would seem that the continuous current is more dangerous than the alternating. The longer duration of action of the continuous current apparently produces more serious effects than the intermittent action of an alternating current of the same voltage and intensity.

Among the individual factors, the most important is the varying resistance of the human body. The skin is the great protection against the entrance of electricity into the body. The resistance of a dry and healthy skin is so great that, in comparison, the electrical resistance of the internal organs is quite negligible. From actual measurement, we find that the resistance of the integument in different individuals varies between 30,000 and 100,000 ohms, and may even be as high as 1,000,000 ohms for the dry, hard skin of a labourer's hands. The more delicate the skin, and the more freely it is supplied with capillaries and sweat glands, the less is the resistance. On this account the skin of the female is usually of lower resistance than that of the male. The natural protection given by the skin is reinforced by that of the clothing, more especially that of boots and stockings. This protection varies greatly according to the nature and condition of the clothing, whether they are wet or dry.

Since most accidents are caused by the earthing of a high-tension wire through the victim's body, the state of the ground on which he is standing is a most important factor.

Among floors which are *absolutely dangerous* I would mention those of bath-rooms, cellars, and mines, since they are usually made of conducting material, and are often wet. The danger of electrical installations in such places is therefore greatly increased. Floors which are *comparatively safe* are those which are covered with a carpet, or otherwise well protected. These, however, may be rendered dangerous by the presence of waterpipes, gaspipes, or other good conductors, leading to earth. Sometimes an old gasolier is adapted for the electric light, and if this has not been properly insulated, danger may arise from touching the electric lamp socket with one hand and the gasolier with the other. Several fatal accidents have occurred in Vienna from touching the ordinary electric lamps when the victim has also been in contact with a metallic conductor leading to earth.

I will show you pictures of how such accidents are produced. Here is a picture showing a man touching an old gasolier ; the current passed from his right hand through his body to the left hand, and so from the gasolier to the earth. The boy shown in the next picture wished to try what would happen if he touched the electric wire ; he was instantly killed. The next picture shows a boy carrying bread through the garden to the house ; he touched with his left hand the iron railing, which carried electricity. The day before a mechanic was working on the roof, and when leaving threw away a piece of wire, which remained connected and carried electricity to the trellis work. This was the cause of the boy being killed. The next shows another kind of fatal accident which we have had in Vienna. The man was employed to take out from a cellar an electric arc lamp. The lamp did not light, but in spite of that he was killed. It was a one-pole switch, and the lamp was in continuation with the other wire, so that when the man touched the electric lamp the current from this one pole passed through his body to the earth, and he was instantly killed. The next slide shows an accident which often happens—namely, the breaking of a telephone wire in the street. These, of course, have electric tension, and unless people are careful they may easily be struck and wounded or even killed. It is difficult to free such a person, because the telephone wires are very hard, elastic and tough. In Zürich some rescuers tried to set free a man so caught, and in doing so the wire got round the man's neck, with the result that his neck was cut through—burned through by the enormous heat generated. On the screen I show you some small pliers which I have constructed for the purpose of freeing such wires. They are handy and can be carried in the pocket. The ordinary implements are enormously long.

The condition of the earth plays a very important rôle. All these accidents which I have shown you were produced by earth circuit. Another factor which plays a very important rôle is the particular path which the current takes in the victim's body. Many rescuers have been killed from touching such a victim. When the body is struck the electricity passes along two paths—one on the surface and one penetrating the body. In the Physiological Institute of Professor von Tschermak in Vienna we have made experiments on horses, which are very suitable because their bodies are long and the horse is susceptible to electricity. If we subject a horse to 100 volts, we can prove that part of the current goes through the body itself and part through the skin. I think this will enable us to account in some measure for the failure in some cases of electrocution in America. I shall mention this again later.

The mental condition is a most important factor. If you touch an electric wire intentionally, you can bear 100-volt, 200-volt, or even 300-volt currents. At the beginning of my studies I myself touched a 500-volt current, and I know an engineer in Vienna who has touched a 1,000-volt current, a voltage which may easily be fatal. There have been cases in which electricians have been used to receiving charges of electricity and have often intentionally touched conductors, but on doing so once *accidentally*, have been killed. Most of the victims we have observed in Vienna have been persons of experience, some of ten or fifteen years' experience, and yet they have been killed.

A similar phenomenon is observed in electrocution as practised in America. The prisoner is fully prepared for the passage of the current, and therefore the moment of surprise or shock which produces paralysis is absent. Of thirty-six criminals electrocuted in America, in no single instance was there instantaneous loss of consciousness followed by immediate death as so often happens in electrical works. This lack of success in the American judicial electrocution is, in my opinion, due in great part to the absence of this factor of surprise; the failure is also due in part to faulty technique, which causes the path of the electrical current to be partly deflected. When the human body is introduced into an electrical circuit the current takes two directions—a direct path through the body, and a side circuit over the surface of the body. It is not the shortest way, but the way of least resistance, which is the most important. When the skin is covered with perspiration or moisture, a large portion of the current passes over the surface of the body, and the internal organs do not receive sufficient density of current. Moreover, in electrocution it is usual to employ a metallic helmet filled with cotton-wool drenched with salt solution; this solution runs down over the prisoner's neck and body, thus affording a path of low resistance, which allows the electricity to stream over the surface of the body instead of penetrating it.

Another of the individual factors which determines the gravity of an accidental discharge is that of species. Not only are various types of men very differently affected by the electric discharge, but different species of animals vary greatly in susceptibility. Among the animals on which I have experimented, I found the horse and the white mouse most sensitive to electricity. A horse is readily killed by a current of 100 volts, and a white mouse by 35 volts or less. Dogs and guinea-pigs show much more resistance, nearly as much as fishes and pigeons. On the other hand, I was quite unable to kill either frogs or tortoises by electricity, short of actual burning by the electric light arc.

From numerous experiments I have concluded that death by electricity is really in the first place merely suspended animation, which is later changed into real death.

I now want to show you some of the changes produced by electricity, for they are important not only from the scientific and clinical points of view, but because it is necessary to study them from the forensic standpoint. Electro-pathology plays a very important part in social life. The physician is called upon to give his opinion about an accident, whether it is grave or slight. He may be required to give evidence in the case of a claim being brought by a labourer, and sometimes his advice is sought in cases of self-mutilation in order to get compensation. Electricity is used for suicide and for murder. I show you illustrations of lesions produced by electricity on the skin and tissues. The current also damages bones and viscera. In Vienna we have an electro-pathological museum in the Institute of Medical Jurisprudence, which we find of great use. These are the remains of the clothing of a boy who was struck by lightning, though he was not killed nor even rendered unconscious. Here are two splinters of glass from a window, which were found in the heart of a girl who was said to be killed by lightning. But it was only a so-called "false action" of lightning. In the case of accidents from electricity we have been able, owing to the material we have in this museum, to reconstruct the accident. Once they wrote to us at Vienna, from the University of Lille, for advice in regard to an accident to a boy who was found at a distance of 10 metres from an electric plant, and was said to have been killed by electricity. We were able to answer that this was possible, and our answer was based upon our experiments upon animals. Later our opinion was strengthened by the result of our own experience of accidents. The next picture shows a man working with a bare, uncovered electric wire. He omitted to take out the safety fuse, and so handled a wire under electric pressure. Most of the accidents happened through carelessness, recklessness, and thoughtlessness. You see in this way he made a short circuit. The metal he had in his hand not only melted but volatilized and fell on to his face. You see the effects it produced. At first sight it looks like a very severe injury, but it turned out to be very slight. The same changes are seen on his hands. It is an impregnation of the superficial layers of his skin by volatilized metal (figs. 1 and 2). In three weeks he had recovered. The next shows the case of a girl who was killed in her bath. She touched with her hand the lamp, which was not properly insulated (fig. 3). When we came to see the body in the bath, the electric lamp lay on the body and was alight. When we lifted the lamp from her body the light went out, and when

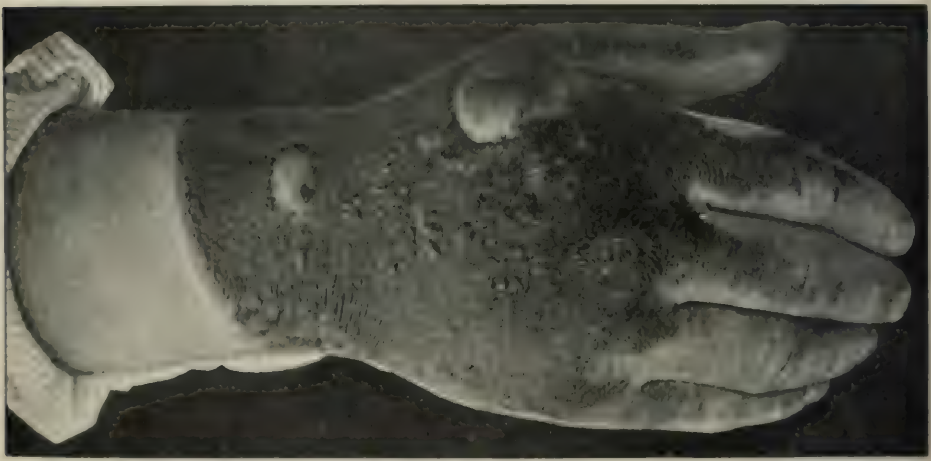


FIG. 1.

Metallic impregnation of the skin of the hand.

we put the lamp back it was again lighted. This diagram shows the method of connexion with earth. In the next case a man, employed in electrical work, was standing on a ladder, and gripped the wire; he immediately fell down dead. Notice the changes on the skin of the sole and the hole burned through the sole of his boot. This was the first fatal accident in Vienna from electricity. In the next case an electrician was employed on a transformer and wore gloves to protect his hands, but the glove did not reach high enough and, his forearm touching the electric conductor, the current passed through his forearm to his axilla. He leaned on the earthed railing and, the current passing through this, all the rest of his body escaped. I show you the changes in the tissue of the forearm and axilla. The next slide shows the fingers of the left hand of the man whose left side was degenerated and atrophied, but three months later regeneration occurred and he recovered. Two years later the man was able to earn his living again. Next I show you some changes in the joints. We can demonstrate here rarefaction of the bones produced by the electric shock. This tissue change occurs not only in the nervous but also in the vascular system. The next shows a case of self-mutilation. The man wound this round his finger seven times and touched an electric conductor, in order to

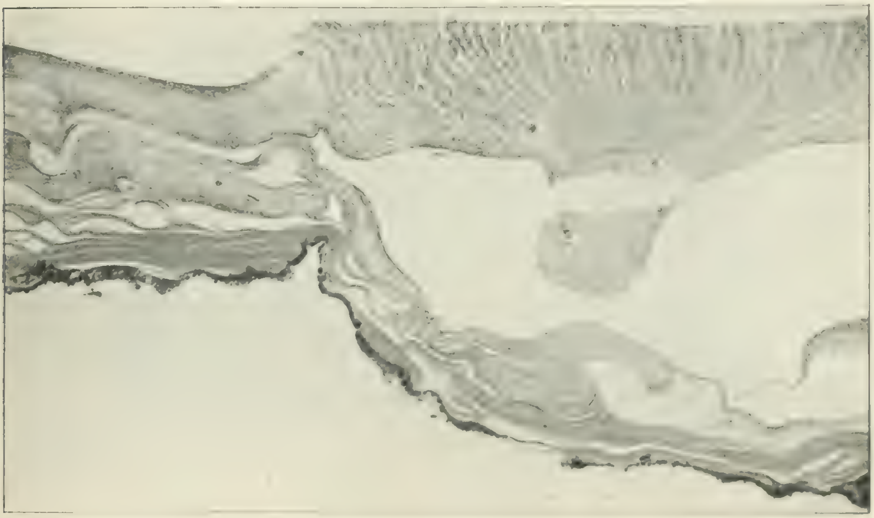


FIG. 2.

Section showing fine metallic particles in the superficial layer of the skin; internal laceration of the skin.

convince the inspector that he had had an electric accident. We could not understand how it happened, and eventually the man admitted that it was self-mutilation. The next shows a painter who was employed to paint a post. On this post there are two horns for lightning conductors. They were hidden by the branches and the leaves of the tree, and the man did not see them. Consequently, when he touched the overhead wire the current passed through his left hand, his body, and the post to the earth, and he was instantly killed. Notice the changes in his skin. In the next case the man was engaged in connexion with a cable, and was to signal to his colleague when he was ready; but his colleague did not wait, and turned on the electricity, which passed through the cable through his knee to the earth. Notice the enormous lesion and loss of tissue which were produced. The muscles are wanting, and so are the epicondyles, and the knee-joint is open (fig. 4). He was brought into our hospital, and the surgeon who saw him urged amputation of the knee. I said, "It is not necessary to do that, because these electric lesions have a very favourable course of healing, and generally heal without fever or suppuration." They are all painless. When I began my studies twelve years ago, we made an amputation of the forearm of a man, because we were afraid of suppuration setting in and subsequent septicæmia. But now, with better knowledge, we do not amputate, because we know how favourable the prognosis is in these cases. It was not possible to save this man, because he was so very badly burned over his body by the electric arc light, and he died four days later. You see the lesions on his back are very severe and there is an enormous loss of tissue. Here is a picture of a wound in the neck which looks like one produced by hanging. I call your attention to the wound above the ear

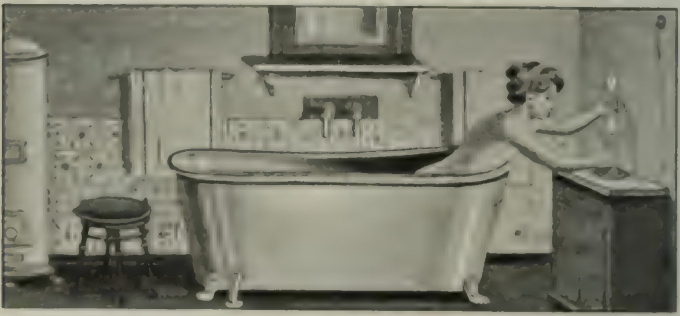


FIG. 3.

Accident caused by handling a badly insulated electric lamp.

When the patient was brought in we looked for the lesion produced by the second pole. I could not find it because the wound was covered with quite undamaged hairs. But later there was a scab which revealed the situation of the second wound. These injuries are not always burns, though generally they are when there is an electric spark which comes into contact with the skin. The differentiation is important from a forensic point of view, because these electric lesions are painless. This fact is of use in cases where workmen bring claims for damages on account of wounds which they say are very painful and keep them awake at night. Electrical wounds are not painful. The case here illustrated was that of a man who was working on an iron gallery in a small room. He touched with his back an electric wire which was insufficiently covered. The current passed from his back to his body, through his body to his feet, and so to the earth, but he did not become unconscious. His clothes were not much damaged, but there were enormous wounds on the body. Notice the changes on his hands where he touched the railings. The body of such a case (a man aged 60) was brought into our Forensic Institute. He was found dead in the factory, and we did not know what had happened. Examination of the hands showed these lesions, and Professor Kolisko said immediately that he was killed by electricity and must have touched an iron railing. Examination of the place showed that this opinion was right. The scar following an electric accident is soft and smooth, and does not shrink.

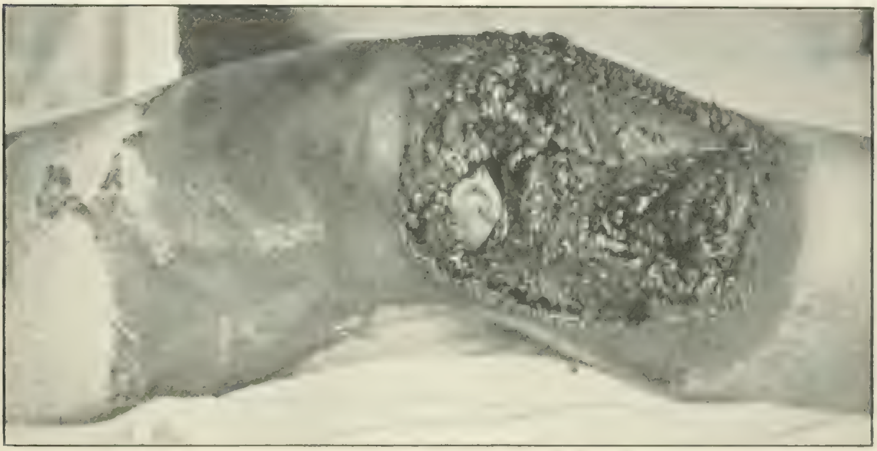


FIG. 4.

Destruction of the knee-joint; metallic impregnation; electrical burns.

This, as you know, is very different from the scar of an ordinary burn. I show you slides illustrating this. This man was employed in measuring the copper vase, which was not covered, and, grasping the copper wire, made a short circuit. The result was that the copper melted. By good luck he was wearing glasses, and this saved his eyes. I show you a photograph of the glasses now; the opaque spots are yellow spots which show that the metal got down so deeply into the glass that it could not be removed. The glass is quite impregnated by it. Here is a case of ophthalmia electrica. The next photograph shows recovery four weeks later.

Now I show you the results of microscopical examination. We have seen that there is degeneration of the nerves, and how it is produced. I have examined the brain and spinal cord from a man who was killed by electricity, and I show you here a slide from the medulla oblongata, showing the boundary between the grey and white substance. On this boundary there is a hæmorrhage. You can see also the ganglion cell. The ganglion cell is destroyed, and the nucleus lies outside of the cell. There is a similar destruction in the brain. The next slide is from a guinea-pig which was subjected to the electric current. Here you see the blood which has penetrated into the cell.

Next I show you a slide to demonstrate the strength of the current. Here is a gaspipe which we obtained from the County Council of Dresden, and here is a waterpipe from the City of Erfürt, sent to our Electro-pathological Museum. See how the pipe is broken, and its surface shows corrosions; this is produced by currents which go into the earth, and that this corrosion can be so produced is very important from the hygienic point of view. In Vienna a waterpipe was damaged by this means, and you can understand that if a gaspipe were made to leak in this way, and the gas were ignited by the electricity, the results might be very alarming. The next slide shows a piece of cable, and you see the steel cone, with holes made in it by the labourer's pickaxe, as a result of which there was short-circuiting, and the cable at once destroyed.

I wish now to speak of death by electricity. What is the mechanism of death by electricity? How is death produced? As there are many who have been engaged on this question, so we have many opinions. The first author who worked at this question was the French physician Marat; that was before he became a Revolutionist. He worked with a charge from a Leyden jar, with insulating gloves, and said that death by electricity was death from irritability of muscles. Some say that death by electricity is primarily produced by stoppage of the heart; others say it is by paralysis of respiration; others that it is a paralysis of the brain.

and so forth. I do not agree with any one of them, but I agree with them all together; all are right. Death occupies only a fraction of time. Even if we had a stop-watch we could not differentiate. For instance, a man is struck by lightning, and falls down. He raises himself, and speaks to his companion. If he speaks he must be able to breathe. Then he falls dead, because his heart has stopped. Dispute on this point is quite useless, for everybody is right. In many cases it is apparent death, it is suspended animation, and many victims might have been saved if this idea had been more prevalent. If the physician cannot feel the pulse he often says the man is dead. But if artificial respiration is done, the victim revives. The first case recorded was that by D'Arsonval, of Paris, when he was called to a victim two hours after the accident. This artificial respiration must be continued for hours. Another point I wish to accentuate is this: if a person is struck by electricity, the rescuers run away for a physician, they run to the telephone, &c., instead of immediately commencing artificial respiration. The instructions for first aid which are in use in Austria, and Germany, and America, are not in accordance with present knowledge.

The following are the instructions for first aid in electrical accidents issued by the Union of Austro-Hungarian Electrical Works, according to the resolutions passed at the Congress of Milan in 1909, and Salzburg in 1912, and recommended by the Austrian Government for use in electricity works in June, 1912:—

FIRST AID IN ELECTRICAL ACCIDENTS.

(1) Remove the victim from contact with the live wire.

(*a*) Cut off the current at once by opening the nearest switch, by removing the safety fuse, by severing the wire, or by short-circuiting the current. Use an insulated tool, a piece of dry wood, a hatchet with a dry handle, or wire nippers with insulated handles.

(*b*) The rescuer must take the greatest care to see that his own hands and feet are insulated. He should stand on a dry board without nails, a dry coat, carpet, or stool; he should protect his hands with the sleeves of his own coat, a dry cloth, or, better still, india-rubber gloves.

(*c*) The rescuer should place an insulating material under the body of the victim, a dry rug, or sack, or a board without nails or other metal. Sometimes it is possible for the sufferer to tear himself free from the wire by leaping into the air, and dragging himself free while the earth contact is broken, or the rescuer may lift the patient from the ground whilst himself standing on an insulator. He should avoid touching the sufferer without proper insulation, and on no account should he touch the bare skin without this precaution. When the sufferer cannot free himself from the contact, each of his fingers should be carefully freed one by one, wrapping each one as it is removed in a dry cloth.

(*d*) If the wire is wound round the victim, it should be removed with well-insulated hands. The live end of the wire must be held fast or tied back to an insulator in order to avoid further accident.

(2) First aid should be undertaken immediately, and the sufferer must not be left alone even for a moment.

(3) The patient should be laid in a horizontal position, the clothing loosened, and the upper part of the body stripped, so as to admit air and light.

(4) The head of the patient must not be allowed to hang down, but should be slightly raised on a pillow formed of a folded coat.

(5) If the patient is unconscious, means of resuscitation should be adopted at once.

(*a*) The mouth, nose, and throat should be examined to see if the air can enter freely. Any mucus, food, or false teeth must be carefully removed.

(*b*) On no account must any liquid—water, wine, or brandy—be given to a patient in an unconscious condition.

(*c*) If there is no movement of breathing, artificial respiration must be commenced without a moment's delay.

(*d*) If there is only one rescuer, he should kneel with the head of the patient between his knees. Taking an elbow of the patient in either hand, he presses them firmly together against the patient's chest; he then separates the arms, drawing them sideways and upwards over the patient's head as if the patient were stretching himself; the elbows are then again brought into contact and pressed against the patient's chest; and these motions of expiration and inspiration are repeated some fifteen times a minute.

(*e*) When there are two helpers, one of these should be sent immediately to fetch a doctor, while the other continues the artificial respiration.

(*f*) With two helpers, one kneels on either side of the patient, each taking one of the patient's arms by the wrist and the middle of the upper arm. The motions are repeated as above, care being taken that both arms are moved in unison. These movements should not be too quick, the upward and the downward motion being repeated from fifteen to twenty times a minute. Any forcible movements or rough pressure on the stomach or liver should be avoided.

(*g*) In case of necessity, artificial respiration should be kept up for two or three hours. Many cases have been saved when the victim has been apparently dead for hours.

(*h*) Where there is only one helper, and he greatly fatigued, he may from time to time make use of another means of artificial respiration. For this he kneels astride the patient, and with a clean handkerchief seizes the patient's tongue with his right hand, the left hand being placed flat on the patient's chest. He now alternately draws the tongue forcibly forwards, and presses with the left hand on the patient's chest, while allowing the tongue to recede. This alternating motion should be repeated twenty times a minute.

(6) Whilst the artificial respiration is being carried on, the second helper should feel and listen for the beat of the heart under the left nipple. If no heart-beat is to be detected, he should immediately proceed to stimulate the surface of the chest over the heart. This may be carried out by rubbing with cold and hot towels alternately, by slapping the surface of the chest with a wet towel, or even striking the neighbourhood of the heart with the closed fist.

(7) Of course, the preceding instructions must be modified if the patient is seriously injured by wounds or broken bones.

(8) The treatment may be varied by stimulation of the soles of the feet, by cold irrigation of the bowel, or by alternately pouring hot and cold water over the chest and abdomen. *In no case should the subsidiary treatment be allowed to interfere with the continuance of artificial respiration.*

(9) During the whole process of restoration the patient should be carefully watched for any return of the natural breathing. The return of consciousness should be encouraged by calling the patient by name, by stimulation of the skin, slapping the chest and face, &c. After the return of consciousness the patient should still be watched, and not left alone. He may have warm drinks—coffee, tea, or a little alcohol—and should remain for some time in a semi-recumbent posture.

(10) The treatment of burns or superficial injuries, which usually follow a very favourable course, should be left to the physician. The only complication which may require first aid is the occurrence of hæmorrhage.

The blood-pressure changes vary according to the tension. Up to 80 volts the blood-pressure rises, but with a higher tension in the same animal the blood-pressure falls, so much so that the heart stops. I have tested in the Physiological Institute of Professor von Tschermak, in

Vienna, not only the pressure in the carotids, but have taken a kymographic tracing of the apex of the heart. You see that when we subject the animal to a voltage of 110 the blood-pressure drops (fig. 5). In a dog we notice the waves are greater, and 80 volts for only a short time give us a fibrillary curve (trémulations fibrillaires). After a few seconds the heart-beat is sometimes better again, especially in a rabbit.

I now wish to call your attention to some experiments I have made, and which I do not myself understand. On subjecting an animal to 110 volts the blood-pressure decreased, and afterwards increased again. When I use that voltage with two poles, one in the mouth and the other in the rectum, and electrolyse the vagus nerve, the blood-pressure decreases at the same rate. But if I subject the same animal to the danger of a fatal current, we see the absence of this decrease (fig. 6). So all we can say is that we have thus produced an immunization against electricity by electricity. And this seems to me very important. Professor von Tschermak and myself, after chloroforming a dog, opened its skull, and then irritated the fore and hind legs; these curves show every movement, and how long the irritation lasted. I subjected the animal to an electric shock of 120 volts for three seconds. What happened? The blood-pressure rose for a short time, but then decreased continuously until we got this abscissa. I could not indicate the breath-

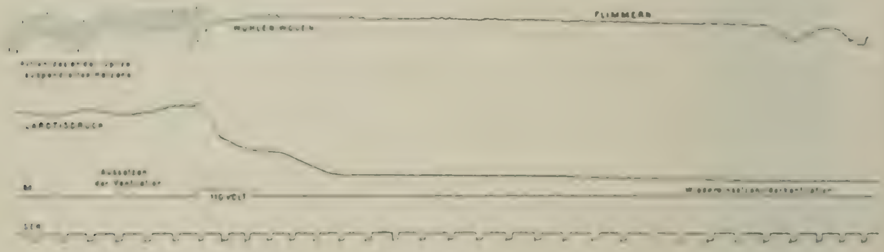


FIG. 5.

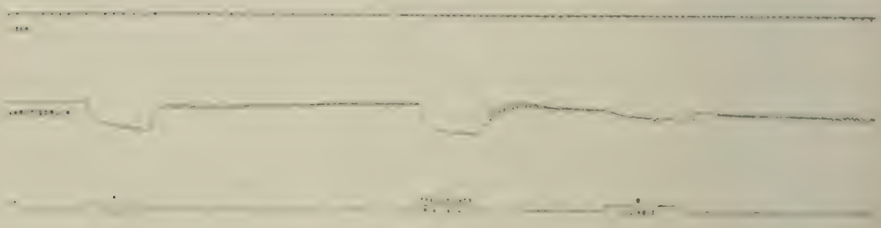


FIG. 6.

ing on the one diagram. The spasm of the legs lasted forty-five seconds. I then irritated with a current of medical strength, and you see the result. The movement ceased about one hundred and five seconds after the shock. What can we learn from this? That after the shock the brain is enormously excited, but after this excitement the irritability subsides, and there is decreased susceptibility to electricity (fig. 7). There is a paralysis of the brain, but that paralysis is not definitive, the irritability of the brain returns. So in the case of victims, everything depends on whether the action of the heart returns. The victim falls down unconscious, but after a time he is able to raise himself up and speak, in spite of the fact that his heart is not beating. But this excitation of the brain cannot last for long, and that is the reason prompt first aid is of such immense importance; it is the critical time the time of payment to death (Verfallsfristanden Tod). Ten years ago this question had not been decided.

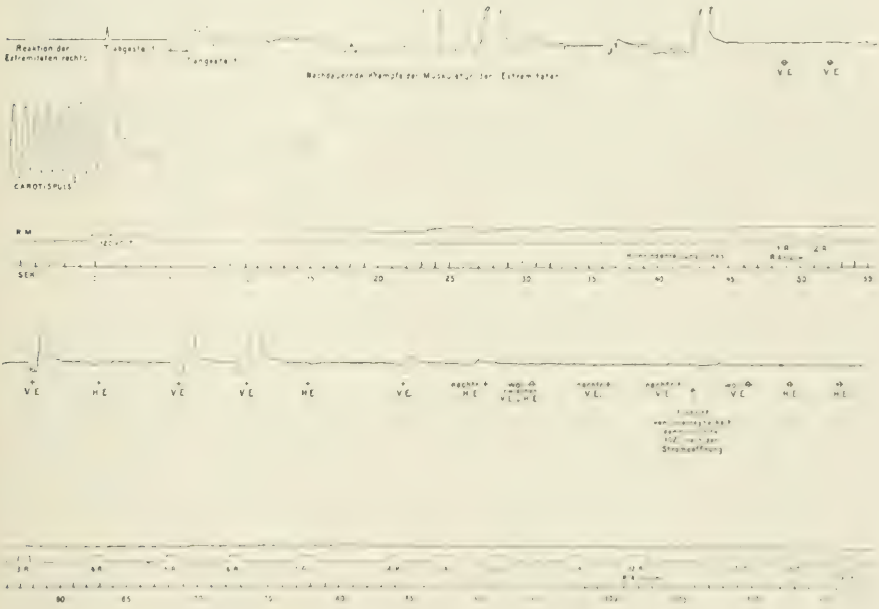


Fig. 7.

I show you some slides of experiments with the electric current on rabbits; you see the degeneration of the sciatic nerve.

When a victim is struck by an electric current you should know how to free him without becoming yourself implicated; a person seeing another drown has regard also for his own life. It is not possible to teach in writing how to free the victim of an electric discharge: it should be practically demonstrated to pupils and electricians.[1] Immediately after freeing the victim you must attend to the patient's breathing. If a fellow-workman lays the victim on his face, his efforts at restoration may injure the liver and cause fatal hæmorrhages. Or he may press out the stomach contents. In one case, after an electrical accident in Vienna, the stomach contents were pressed out into the trachea and inspired into the lungs, so that the man was really drowned by his own food-stuffs. The great point I would insist on all through is that efforts at restoration must be commenced at once, and artificial respiration must be done carefully, but not forcibly.

Dr. Jellinek concluded by demonstrating the way in which accidents happened, and the manner of dealing with them, by means of the cinematograph.

DISCUSSION.

Dr. LEWIS JONES said he was sure he could speak for all present in thanking Dr. Jellinek for his paper. The hour was late, but there were one or two points which he might just mention, and one was that the cases he had seen of people who had very serious non-fatal accident did not appear to suffer from any after-effects of a nervous character. He mentioned the point because from time to time he had read accounts of persons bringing legal actions for compensation and describing themselves as suffering from all sorts of nerve symptoms as the sequel of an electric shock. The second point was that in some cases where death was not caused by the primary effect of the electric shock, a fatal result might follow from the very serious thermal effects produced by the passage of large currents through the limbs. A few years ago there was an accident in which a young man received such large currents through his body that the muscles and blood-vessels of the limbs underwent a heat coagulation necessitating amputation. He survived the accident for nine days, and it seemed strange that though the current in that instance had been so great as to raise the temperature of the limb sufficiently to destroy the living tissues, nevertheless the heart had not been arrested by the electric shocks.

Professor SILVANUS THOMPSON said that a great many things which Dr. Jellinek had mentioned had been of extreme interest to him, and though, happily, he had never seen a man killed by electricity, he had seen various accidents of other kinds, where machinery had been put out of order and destroyed. Some seventeen years ago he took out a life policy for himself in a certain insurance office. When the policy came to him to be duly signed, it was found to contain a clause stating that he (Professor Thompson) would on no consideration step upon a switchboard of an electric lighting station. He promptly scored it out and said he would not sign it, as that was his business. The insurance company in the result omitted it. Many people interested in electricity had to go on switchboards from time to time, and he was surprised that the number of accidents in connexion with them were so few, particularly on high-tension switch-boards, considering the risks which the special workmen had to run in fitting and adjusting things on them. He could only conclude that the immunity they had was due to the circumstance that they were mostly highly skilled men, who were able to appreciate the risks which they ran. And that was not all, for at this lecture he had been learning to appreciate that the Government of this country made useful regulations which certainly would have prevented some of the accidents which had been depicted as occurring in Austria. For instance, the fencing of electrical machinery was carried out in this country in a way which was superior to that in any other which he had visited. We had a Home Office, with a Mining Department, and a Board of Trade, which looked after these things. We were apt to blame our Government Departments for their grandmotherly legislation and their details and their inspectors, but it was undoubtedly because we had these things that we were so immune from accidents. Mr. Ram, who was sitting next to him, had for years been adviser to the Home Office, as Mr. Trotter advised the Board of Trade, on questions of this kind; and there were other inspectors for mining. People should reflect, too, that they owed a great deal to the gentlemen who had been put in those positions, for they had, without creating any antagonism on the part of the industry, succeeded in carrying out good and effective rules for the regulation of the dangerous trades. The very first slide thrown on the screen showed an accident which arose from an electric lamp being attached to a gas fitting, a state of affairs which was impossible in this country if the regulations were obeyed. No insurance office would pass an installation of that kind, unless that fitting was detached from the gas pipe, and hung independently from an insulated support. Again we had to thank grandmotherly Government Departments and insurance offices for the advantages we enjoyed of comparative immunity from accidents due to the presence of unprotected conductors. The comparative insensibility to the result of electric stimulation of those who, like himself, were constantly receiving slight charges of electricity, made one wonder whether those who did so were not specially immune from the greater danger.

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[1] In some schools of Vienna (k. k. Gewerbeförderungsamt und Technolog. Gewerbe-Museum) we teach the pupils by means of pictures and lantern slides (from our Electropathological Museum) how electrical accidents originate and how to avoid them.

Mr. G. SCOTT RAM (Electrical Inspector, Factory Department, Home Office) said he was somewhat surprised to notice, from the pictures exhibited, showing the resuscitation of those apparently dead, that the old Silvester method was still advocated. Recently, in this country, Professor Schäfer's method had been adopted very largely for drowning cases, and was recommended for electric shock. It was now used entirely by the Metropolitan Police. Though he had known of a number of cases of electric shock which had been brought round by the Silvester method, nevertheless the medical men who had given advice to the Home Office and other departments on these matters had pointed out that the Schäfer method had proved so much better in drowning cases, that it must also be better for electricity, and it was now generally recommended. The recommendation was, however, only of recent date, and he had not had actual experience of any case in which the method had been used for electric shock; but that day he was looking at a work recently published by an American, Dr. Lauffer, attached to the Westinghouse Electric Company at Pittsburg, in whose extensive electrical works and stations he had had a wide experience, and he recommended the Schäfer method. Dr. Lauffer gave an example where in one of the works the chief electrician had dealt with six cases of electric shock in which the victim had been rendered insensible, and in every one had been brought round apparently by the Schäfer method. The lecturer did not touch upon the relative dangers of the alternating and the continuous current. It was frequently stated that the continuous current was more dangerous than the alternating; but he did not consider that statement was borne out by the facts. In the last ten years he had had under his notice on premises under the Factory Acts forty-five cases of persons killed by alternating electrical shock at 250 volts or less, as against only three by continuous current, though he believed the alternating current and continuous current were used to about an equal extent. One thing he would like to comment upon, as the lecturer had done so — if he had not he would not have cared to refer to it himself in such an assembly — and that was that if only one person were present, he should not run for the doctor, but, as the lecturer had very rightly insisted, he should commence resuscitation measures without delay. It was also wrong to send the patient, perhaps some distance, to a hospital before he had been brought round, as he might die on the way. In one case which came under his notice a man had a shock at a switch pillar in the street and was rendered insensible. A policeman was passing at the time and saw it was an electric pillar, and ran across the road and told a doctor what had happened. The doctor told the policeman to take the man to the cottage hospital, which was a mile away. It was half an hour before he could be got there, and he was then pronounced to be dead. That man did not really have a chance. Sometimes medical men did not seem to give these cases sufficient time. He had known cases where a medical man had been sent for and pronounced the patient dead, and had gone away within half an hour of the accident. In one well-authenticated case the victim's friends decided to "have another go at him," and succeeded in restoring him after he had been pronounced dead. One sometimes saw results which it was difficult to account for. The effect of the current must depend on what part of the body it traversed. One recent case, unlike any others he knew, was interesting in the fact that the current did not appear to have passed through any vital part of the body. The ground of certain works had become charged by leakage from a 400 volt alternating three-phase circuit. It was found to be so charged, and workmen were cautioned against walking over that patch. One man said he did not care and started to walk over it; he fell down and was killed. The current must have passed up one leg and down the other; and it was difficult to understand how it could have gone to his heart, or how it could have interfered with his breathing. He had notes of other interesting cases, but the hour was too late to deal with them.

The PRESIDENT (Dr. Reginald Morton) said he knew that many felt they would like to continue the discussion, but, as had already been said, the hour was late, and before asking Dr. Jellinek to reply he wished to say how deeply the Royal Society of Medicine was indebted to him for coming all the way from Vienna to address them. He knew it had been done at a considerable sacrifice of his valuable time. The address was a masterly production, and it showed that he had done an enormous amount of very careful and painstaking work, and in a manner possible only to a man whose interest in it had practically amounted to a passion. He hoped that the points which Dr. Jellinek had impressed on the meeting would, from that day, be much more widely disseminated. The knowledge of the medical profession on the question of electrical accidents was deplorably small, but this was chiefly because the ordinary practitioner saw so few cases. He might go through his whole professional career without coming across one instance of the kind under discussion. It should be clearly understood that the first thing to do in a case of electric shock was to apply the ordinary simple method of resuscitation, and if that were properly grasped it would be a great gain. Dr. Jellinek had proved that the critical time was the first few seconds or minutes after the shock, and that was pre-eminently the time to act. These facts should be made known throughout the electrical industry as well as the medical profession. Dr. Jellinek showed one photograph which revealed rarefaction of the ends of the bones in the wrists which he said was due to electric shock. No doubt this was correct, but the question which arose in his mind was as to whether it was a primary result of electric shock or secondary to a previous arthritis. [Dr. JELLINEK: I think it was primary.] It should also be remembered that electric wounds were, for the most part, painless; and when self-mutilation was carried out by this means the patient was almost sure to complain of pain, often saying it produced loss of sleep at night. Though he had been interested in electrical matters for a long time, he had come across only one instance of electrical injury, and that was in a generating station at Taunton, where there was a small 35-kilowatt machine, which the engineer treated with scant respect. One day he made a short circuit, the current passing from little finger to thumb. The wounds were just of the kind Dr. Jellinek had described, quite painless, and healed up in about ten days, with only a little exfoliation. He was sorry Dr. Jellinek had not found time to speak of the psychological effects, for one case had been recorded in which the death of a person occurred when touching a conductor which at the time was quite dead: the victim thought he had touched a live wire and suddenly died. This was an extreme instance of the psychological factor determining the severity of the accident. The film shown belonged to the Austro-Hungarian Government, and he was sure the meeting would wish to express its deepest gratitude to that Government for allowing it to be brought for the edification of the meeting.

Dr. JELLINEK, in reply, said that Dr. Lewis Jones had mentioned the difference between the effects of high- and low-tension currents; he agreed it was sometimes difficult to explain why 100 volts were fatal and yet 1,000 volts could be received without much injury. But it was interesting that the danger-point in electrical voltage had not only an inferior but also a superior limit, and it did not seem possible to explain this from a physical standpoint. Professor Silvanus Thompson mentioned accidents on switch-boards, and expressed wonder that they were not more frequent. He was of the same opinion. Though there were accidents enough in Vienna, there were still more in Germany. But he must, in honour of the Austrian Government, and in honour of electro-technicians in Austria, say that the instructions were so excellent that, if they were observed, no accidents would happen. He wished to emphasize his statement that carelessness, recklessness, and thoughtlessness were the chief causes of accidents. Mr. Ram spoke of artificial respiration by Professor Schafer's method. He knew this method very well, because he once used to employ it; about three months ago he published a paper[1] in the official journal of the medical faculty of his University on the subject of first aid, and specially discussed the Schafer method. The ordinary labourer in Austria did not know which was the stomach and which the liver, especially if the victim were lying on his face. In Pittsburg the Westinghouse practice seemed to be to use force, and there was danger of rupturing viscera. They had had fatal accidents from violently carrying out first aid—one after supposed death by lightning. There were, especially in Germany, machines to induce artificial respiration, and there seemed to be the idea that one must procure a promoter; but one could not have so many promoters that there should be one wherever an accident happened—moreover, precious time was wasted in obtaining the machine. Most of his fatal cases were caused by the continuous current, and, judging by the chemical action, the corrosive electrolytic action, the continuous current seemed to be more dangerous, but this question was undecided. He did not say "Don't send for the doctor," but "If there is only one person present he should try artificial respiration, and if another person comes, send him for the doctor." His experience agreed with that of Dr. Morton that electrical wounds were painless. He was happy to hear the President mention the fact that his Government had sent the film, and he was glad to be allowed to bring it. It was satisfactory to know that the representative of the Austro-Hungarian ambassador was present, and he would be able to acquaint his Government with what had been said.

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[1] "Die Organisation und Durchführung der ersten Hilfe bei elektrischen Unfallen," *Wien. klin. Wochenschr.*, 1912, xxv, p. 962.

Electro-Therapeutical Section.

December 20, 1912.[1]

Dr. REGINALD MORTON, President of the Section, in the Chair.

Paralysis of the Trapezius from Injury of Spinal Accessory Nerve, and Recovery.

By H. LEWIS JONES, M.D.

THE patient was a young woman who had an injury to the spinal accessory nerve resulting in paralysis of the trapezius. The nerve was sutured, and after a time power had returned. Though the patient had been conscious of improvement, the reactions in the trapezius were not satisfactory until this year. The case could now be regarded as quite cured. The operation was done seven years ago, and for a long time she had had electrical treatment, but lately she had lived at a distance.

Dr. Lewis Jones considered that electrical treatment hastened recovery, provided the nerve was not actually divided. Several years ago he had a case in which there was injury to the musculo-spiral nerve, and for financial considerations he dismissed the patient as soon as he began to improve, telling him that continued progress might be expected if he exercised his arm and had regular massage to it; but if he did not improve he was to report himself. He did not make progress, and so electrical treatment was resumed, when progress again began. That case seemed to prove that the application of electricity accelerated recovery.

Dr. FRED. BAILEY (Brighton) said that fifteen months ago he cut through his ulnar nerve, and commenced treatment very soon after the injury. He noticed that if for any reason treatment had to be suspended, progress was retarded, but within three or four days of resuming the electricity progress had recommenced.

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[1] Meeting held at the Middlesex Hospital.

Recurrent Carcinoma of the Breast treated by X-rays.

By E. P. Cumberbatch, M.B.

Dr. Walsham had suggested that this patient should be shown as an instance of beneficial action of X-rays in the treatment of recurrent carcinoma of the breast. The left breast was removed in August, 1902. Five years after that she noticed a small lump on the lower end of the scar, just over and below the situation of the apex beat. In 1907 this lump slowly got larger, but she did not come for treatment until August, 1911. Then she came under the charge of Dr. Pirie, now at Montreal. Dr Cumberbatch saw her for the first time in October, 1911, and noticed that she had a circumscribed lump growing in the skin over the region of the apex beat, the size of half-a-crown. It was raised ¼ in. above the surface, was quite hard, with irregular surface speckled with red points. She had also a number of "melon-seed" bodies scattered under the skin of the left axilla and the front of the chest. He made a careful chart of the growths, and then commenced X-ray treatment. Not until the beginning of last year (1912) did he notice a diminution of the size of the above-mentioned bodies, and by April they had all disappeared. The skin in the region of the larger growth was now faintly hyperaemic, but there was nothing visible there now. Six months ago he saw an enlarged gland above the right clavicle, and small "melon-seed" bodies in the right axilla, and another below the right nipple. Treatment was continued, and in September, 1912, she went under the care of another officer. Since then, one or two fresh nodules had appeared on the left side, but the gland which he had felt above the right clavicle had disappeared. He had given her quite small doses, half to one Sabouraud, unfiltered, once a month. This was one case of a small collection in which the treatment by small doses, infrequently applied, had been effective, but she had weathered the storm much longer than the others—namely, since 1902. One could feel a bony lump over the sternal end of the clavicle, the nature of which he did not know. It came up soon after the operation, and had not increased in size. It had also received X-ray treatment.

DISCUSSION.

Dr. FRANK FOWLER (Bournemouth) said he thought the treatment might have been still more efficient. He gave full pastille doses each time, one over the scar, one in the axilla, and one in the supra-clavicular region. That procedure was always carried out in his hospital even for prophylactic treatment. That full dose was applied to each part once a fortnight. He put the Sabouraud pastille underneath the aluminium.

Dr. FINZI said he gave one and one-third Sabouraud dose of filtered X-rays every fortnight, and that had become easy with the Cyclops tube, as it enabled one to use large currents for a considerable time. The rays were filtered through at least 1 mm. of aluminium.

Mr. GILBERT SCOTT said his experience with nodular recurrent cases was that when these nodules began to multiply rapidly the patient, as a rule, was dead in six months. He did not regard the lump the size of half-a-crown in this case as a superficial nodule. These nodules appeared as a rule down the neck, and on other parts of the body, and it became a hopeless task to follow them round although with apparently any dose of X-rays they disappeared rapidly. Nodules in the liver usually occurred at this stage.

Dr. FRED. BAILEY said he had a patient in whom nodules had been occurring at intervals for three years. They had disappeared under more or less continuous treatment during that period by *hard* X-rays to the whole area, and radium to the nodules as they appeared. Only two months ago he discovered a fresh nodule, but the patient had kept in fair health and recently had taken quite a strenuous holiday in London.

Dr. NORMAN ALDRIDGE considered that the dosage was determined by how much the skin would stand. His practice was to give half a Sabouraud pastille dose, filtered through 0·5 mm. of aluminium once a week, and in many cases, after six or seven sittings there was sufficient erythema to justify one in stopping it. His dosage was really the same as Dr. Fowler's, who gave a whole pastille dose once a fortnight. But he also had had several cases in which he had to suspend the treatment after six or seven sittings owing to the erythema and inflammation; in one case this proceeded to some destruction of the skin. His practice had been to give the largest dose the skin would stand, and in many cases that was not more than half a pastille once a week. He had a patient who had gone on with that for more than two years. In that case there were recurrent nodules in the breast area, but they disappeared after less than twelve doses. Then symptoms pointed to metastasis in the stomach, and later, in the lungs. He followed these up with the rays, and there had been no burning of the skin, nothing more than a browning of it.

Dr. IRONSIDE BRUCE said it had surprised him to hear the statement that the size of the X-ray dose should be determined by what the skin could stand in cases in which the treatment was employed to influence tissues deep in the skin, because by proper filtration large doses could be given without producing any skin reaction. He could show cases which had been exposed through a felt filter twice a week, for three or four months, without any skin change—cases of exophthalmic goitre. He did not think anyone would know, from looking at the skin, that they had been irradiated. To see the effect of the maximum X-ray dose he had given a half-hour exposure, from a tube running at 5 ma. through a filter, consisting of 4 in. thickness of felt, without causing more than a slight reddening of the skin.

The PRESIDENT (Dr. Reginald Morton) said that the case was remarkable in many ways, particularly in regard to the small doses given and the long intervals between. Mr. Cumberbatch used no filters, and theoretically that seemed right for these superficial nodules, for one wanted absorption to take place. One should not, therefore, use a filter to absorb the rays which otherwise would be stopped by the superficial layers of the skin. The other interesting point was the dosage, and how long it could be given without causing burning of the skin. He had used metallic screens himself in the past, but had abandoned them, because when using them one was always being troubled with dermatitis when least expected, and that interrupted the treatment. His results had been better since he employed non-metallic screens. He now scarcely knew what it was to cause even hyperæmia. The X-ray treatment of malignant disease was a very important subject, and it was a pity the Section had not set aside a whole evening to its debate. There was need for much more accurate knowledge. The tubes were a variable quantity, and neither these nor filtration were fully understood. Nor did one know exactly the quality of ray which was being absorbed into the tissues, and until a standardized system was established, the X-ray therapeutist would be groping in the dark. For screening, Dr. Ironside Bruce used thick layers of felt. He (Dr. Morton) had tried them, and they were satisfactory, but he liked a thinner material, and so he used lint which had been saturated with a solution of tungstate of soda and dried. It must contain a metal of high atomic weight. He used from one to three, four, or six layers of this, according to the degree of screening required. It was also very cheap, and separate pieces could be used for each patient. If those who used metallic screens and had dermatitis in their cases would use non-metallic ones, that trouble would disappear. One speaker said he put the pastille dose close to the metallic screen but underneath it. He (Dr. Morton) thought that was wrong, as there was radiation from the metal which affected the pastille.

The Treatment of Exophthalmic Goitre.

By W. N. Kingsbury.

The underlying pathology of exophthalmic goitre is not even yet fully determined. It has been ascribed to a central lesion in the medulla oblongata; while, on the other hand, it has been regarded as a pure neurosis. Moebius and Greenfield have stated that it is primarily a disease of the thyroid, and this seems to be the view which Kocher also takes. Whatever the actual cause of the disease may be, the fact remains that, in practically every case, at some time or other, some enlargement of the thyroid gland can be made out.

The histology of the normal thyroid may very briefly be epitomized as follows: The gland is covered with a capsule of dense areolar tissue which sends in fibrous trabeculæ. These trabeculæ enclose the vesicles. The vesicles are round or oblong irregular sacs, and consist of a wall of thin, delicate, hyaline membrane, apparently non-cellular, lined by a single layer of short, cylindrical or cubical cells. The vesicles are filled with transparent colloid material. The active principle is probably that substance separated by Baumann and known as thyro-iodine. It is present in the colloid substance in combination with protein matter. Asher and Flack say they have proved that there exists an internal secretion in the thyroid which may be activated by irritation of nerves going to vessels as well as to epithelium. Hugo Wiener has found that, after the excision of the inferior cervical sympathetic ganglion the thyreoglobulin of the thyroid is greatly diminished, as is also the total quantity of iodine. This is not the case after excision of the superior cervical ganglion, and he therefore concludes that the secreting fibres ascend probably from the fifth and sixth cervical nerves.

The changes in the thyroid gland in Graves's disease, as shown by Greenfield, are those of an organ in active evolution. Increased proliferation of the cells lining the vesicles occurs. The cells become cylindrical instead of being cubical. The vesicles themselves become branched or stellate, and their contents lose their colloid nature and become mucous and granular. These changes, according to Edmunds, resemble those which occur in compensatory hypertrophy after partial excision of the gland.

Thyroid extract given in excess produces the following symptoms: Tachycardia of the physiological type of Lewis, headache, tremor, sweating, prostration, loss of weight, and increased nitrogenous output due to increased protein katabolism. Edmunds has observed exophthalmos produced in monkeys and dogs as the result of excessive administration of thyroid substance. Béclère has reported a case in which this symptom developed in man after overdosing with thyroid extract. When we consider that practically all the symptoms of Graves's disease which cause the discomfort and disability of which the patient complains—those symptoms which prevent her from following her ordinary occupation and which, if not controlled, may lead to yet more serious results—can be produced in a normal individual or in a myxœdematous patient, by the administration of excessive doses of thyroid extract, I think we come to a logical conclusion if we consider that these symptoms are due to the excessive secretion and to the pouring into the blood of larger amounts than normal of the hormone elaborated by the thyroid. Kocher, in a paper read in the latter part of 1910 before the Surgical Section of the British Medical Association,[1] adduces strong evidence to show that the above conclusion is probably a correct one. He quotes from the work done by Bruns, Baumgarten, and de Lignevis to show that Graves's disease can be produced in nervous people with goitre by the administration of iodine. This symptom-complex, artificially produced, he terms "Iodine Basedow." It must, however, be remembered that closely associated with the thyroid are the parathyroid glands. These may be involved in Graves's disease, and it is difficult to say exactly what the relation is. It is possible, however, that they are responsible for some, at any rate, of the nervous symptoms. Be this as it may, they participate with the thyroid in the action of the X-rays when the patient is so treated.

The symptoms of Graves's disease do not always make their appearance in the same order, nor do they subside in the same way when the disease is being cured. As a rule, the first symptom to appear is the tachycardia, which at first always, and even after a prolonged time in most cases, is of the physiological type. This is generally followed by exophthalmos, by the obvious enlargement of the thyroid and tremor. In established cases Kocher reports a definite blood change. There is a diminution in the number of polymorphonuclear cells from a normal 5,000 to 1,000 per cubic millimetre, while the small mononuclear cells show a relative increase from 20 per cent. in normal blood to 50 per cent. This change is not absolute, for in undoubted cases of Graves's disease it may not be present, but these cases are exceptional. Probably, during the process of cure, the cardiac symptoms are the first to subside. The exophthalmos then improves, and the thyroid may regain its normal size, but this sequence may be varied. Thus, then, if the symptoms of Graves's disease are—and, I think, we are logical in assuming that they are—due to excessive discharge of thyroid hormone, it will follow that if it is possible to diminish the output of this substance we shall considerably relieve our patient. Until comparatively recently the treatment of exophthalmic goitre was purely medicinal.

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[1] *Brit. Med. Journ.*, 1910, ii, pp. 931-35.

Electrical treatment, in the form of galvanism to the thyroid, and to the cervical sympathetic, was tried with, in some cases, favourable results. Let us consider the results obtained by means of medical treatment. Medicinal measures are notoriously uncertain and frankly disappointing. The drugs advocated are numerous. They range from digitalis to rodagen and thyroidectin. Now where we find a multiplicity of drugs recommended as being worthy of trial in any disease we may be fairly certain that the good results ensuing after the exhibition of any or many of them are not particularly striking. Hector Mackenzie states that, as the result of treatment of patients in hospital, 35 per cent. were cured, or were almost cured, and not more than 25 per cent. ended fatally.

A later stage in the treatment of Graves's disease was reached when this complaint came within the domain of the surgeon. Until a short time ago, when the physician despaired of curing his patient, or when the patient got tired of being treated with drugs, the aid of the surgeon was invoked. The results of surgery are good, but it is not a very uncommon occurrence for a patient who had previously shown no signs of Graves's disease to develop exophthalmic goitre after an operation. This sudden onset may also occur after a severe shock, or after great excitement. Kocher explains this phenomenon by the theory that an increased metabolism of the nervous system, such as might arise from sudden or prolonged nervous exhaustion, produces toxic substances which, when they reach the thyroid, stimulate it to increased activity. Again, surgical interference is inseparable from the attendant worry and anxiety with which most patients, and those suffering from Graves's disease in particular, look forward to an operation. The risk from the anæsthetic—a risk emphasized by the fact that the thymus is very often enlarged in these cases, when a condition approaching status lymphaticus obtains—and the shock after the operation, are not to be looked upon lightly. Kocher, in his first 170 cases of operations on exophthalmic goitre, had a mortality of 5 per cent. This has certainly come down in his more recent cases, and the mortality of his total number, 469 up to end of 1910, was 3·4 per cent. Mackenzie states that of thirteen cases operated on at St. Thomas's Hospital five improved, three were left *in statu quo*, and five ended fatally. Hale White gives four deaths out of eleven operations. Rehn says that in severe cases the mortality is as high as 22 per cent. McWilliam collected 1,055 cases operated upon by Kocher, Crile, Mayo, and others, with a mortality of 4 per cent.

Despite the fact that the results of surgery are good, are we justified, in the face of these statistics, in recommending our patients either to submit to operation or to undergo medical treatment if we can offer them any other line of treatment which will give results anything like comparable with those of surgery?

The operation usually performed is, I believe, one either of partial thyroidectomy or of ligature of one or more of the thyroid arteries, usually the former method. Both of these operations have, as their basis, the object of diminishing the output of the internal secretion. We find that, in old cases of Graves's disease which have got well, the thyroid shows an increased formation of fibrous tissue with a certain amount of cystic change. Can we produce, more quickly than Nature, without the disadvantages of surgical procedure, a similar result? As far as I can make out this is exactly the condition produced in the thyroid after it has been X-rayed. And surely the treatment of an enlarged thyroid with X-rays is simple enough, without the worry and anxiety inseparable from the anticipation of the operation and without the risk of anæsthetic. Moreover, the action of the X-rays does not stop at this imitation of Nature in the production of fibrous tissue. Its inhibitive action on cell activity is one of its best known results, as evidenced by its action on the testicles, hair-follicles, &c. In this way, then, can the hyperactivity of the gland be controlled.

Rowntree, in his work on the "Histology of X-ray Dermatitis,"[1] has shown an increase in the nuclei of the fixed connective tissue cells in the scalp, subjected to several exposures. In a portion of skin from a finger, amputated for X-ray carcinoma, he states that there was marked increase in the thickness of the deeper-lying vessels, while the corium was represented by a cellular connective tissue in which all the normal structures had disappeared. There were no masses of plasma cells or leucocytic infiltration. The connective tissue approached ordinary scar tissue in general appearance. He suggests that his specimens show a progressive series of changes in the two types of tissue present, epithelium and connective tissue. The changes in the epithelium, he states, are of a retrogressive nature, while the changes in the connective tissue are formative in character. This may give some clue to the changes in the thyroid after irradiation, and may explain the diminution of secretion by the direct action on the cells lining the vesicles, helped by the contraction of freshly formed connective tissue. The changes observed in the deeper blood-vessels may obtain in the thyroid and further contribute to the causes of the lessened formation of the hormone by diminishing the blood supply. This change may conceivably be due to secondary radiations set up in the vessels themselves as the result of impact of the γ radiations. In most cases, after being treated with X-rays, the thyroid, after a time, becomes definitely smaller and occasionally it reverts to its normal size. In other cases it remains large after all the other symptoms have cleared up.

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[1] *Arch. Middlesex Hosp.*, 1909, xv, pp. 192-99.

Sir Alfred Pearce Gould recently operated on a case of Graves's disease which had been treated with X-rays and practically cured. He operated because the patient complained of dyspnœa after she had been so treated. He found that the thyroid generally was harder than normal and that the isthmus was practically converted into a fibrous band which stretched across the trachea, and by contracting had pressed upon it and thus caused the dyspnœa complained of by the patient.

As regards the method of treatment, all our patients, with one exception, have been able to come down to the Department to be treated. We have not, as yet, fully determined whether full pastille doses given once in three weeks, or half pastille doses i.e., in point of time given once or twice a week, afford the better results. Our series of cases is not yet long enough to enable us to give preference to either, and, moreover, since the pastille dose was given in all our first cases, those which have been having half pastille doses are, for the most part, at present in the process of being treated. The patient is X-rayed, first one side of the neck, and at the next sitting on the other side, and so on alternately. The anode is placed 7 to 8 in. away from the skin: 0·3 to 0·7 ma. of current are going through the secondary. A fairly hard tube is used, one whose equivalent spark-gap is not less than 5 in. No filter beyond the air has been interposed between the skin and the tube in this series of cases. An erythema is occasionally produced, but the patient is carefully watched, and no further untoward effects have resulted.

During the past twelve months we have treated seventeen cases of exophthalmic goitre in the Electrical Department, some as in-patients and some as out-patients. Of these seventeen patients two discontinued treatment before there was any probability of finding any improvement. Of the remainder eight were treated as in-patients. Three of these were practically cured before they left the hospital, and the others continued treatment after their discharge. One of these has since stopped treatment and is practically well. One discontinued after two pastille doses as out-patient, and was much relieved. The remainder are still under treatment, and are all improving.

Thus, then, of fifteen cases, eight were treated as in-patients in the first instance, and some continued treatment as out-patients. Of these, four were cured, one was much relieved, and three are still under treatment and are all improving. Seven were treated as out-patients, of whom one improved, but relapsed and was operated on; one had four pastille doses, and then discontinued (was much better); five are being treated now, and with one exception are improving.

To sum up, of fifteen cases, four are cured, two much relieved, eight still under treatment (one doing well, one not improving very much), one improved, but relapsed and was operated upon.

Although these results are not quite so strikingly good as those of Dr. Stoney yet they are better than the results of medicinal treatment, and practically as good as those achieved by operation, without the disadvantages of the latter procedure. Dr. Stoney[1] reports out of forty-one cases: fourteen completely cured; twenty-two cases derived great benefit; four were unsatisfactory; one did not do well, was operated upon, and died; thus giving a percentage of 87·8 who resumed their ordinary method of living. Many of our patients have only come for treatment during the past three months, and these have not yet had a sufficient number of exposures for the desired effect. It seems that the best results are obtained when the patient is at rest in bed, free from the worry of home, and is well fed and looked after.

Perhaps the most striking feature about these cases, in-patients and out-patients, is that after only a very few exposures they practically all

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[1] *Brit. Med. Journ.*, 1912, ii, p. 476.

Initials of patient	In-patient or out-patient	Dose	Pulse before	Pulse after	Neck	Eye signs	Remarks
		Pastilles					
M. G.	In-patient	4	132	82	13½ to 13¾ in.	Unchanged	Says she feels better; discharged as much improved
M. P.	Out-patient	11	116	88	No note	No note	Made slight improvement and felt better; relapsed and was operated upon
C. R.	In-patient	8	102	100	17¾ to 16½ in.	Exophthalmos much less	Tremor disappeared; thyroid was smaller and firmer; was on police force and went back to work; is the only man in the force, as far as I can find, who has returned to duty after exophthalmic goitre
E. A.	In-patient	7	144	108	No note of exact measurement; said to be good deal smaller	Exophthalmos disappeared	Tremor much less, and gland smaller; was the only patient too ill to come down for treatment; was treated in ward and did very well; was seen a few days ago, is doing her usual work and is practically cured
A. L.	In-patient	6½	102	92	No note	No note	Tremor less; had 2½ pastilles as out-patient, and then stopped treatment; was much better
E. B.	In-patient	11	158	92	Neck smaller	Exophthalmos less	Less pulsation in thyroid; was sent out after being nearly three months an in-patient; could do nothing when she first went home, but can now do a little house work; still being treated
S. D.	In-patient	5	168	126	13¼ to 12¾ in.	*In statu quo*	Tremor unaltered, feels much better; being treated as an out-patient
G. T.	In-patient	2½	—	—	—	—	Has not since been treated
A. T.	In-patient	4	132	92	No note	No note	Had 2 pastilles while an in-patient, and has since been treated as an out-patient; much improved
K. S.	In-patient	10	102	88	13 to 12¾ in.	*In statu quo*	Tremor less; had 2 pastilles, and has since been treated as an out-patient; feels better, doing ordinary work
C. T.	Out-patient	5	124	100	No note	Exophthalmos less	Tremor less, feels better
E. S.	Out-patient	7½	120	110	12¾ to 12¼ in.	Exophthalmos less	Tremor slightly better, feels better, palpitation better, and sleeps fairly well; lost 3 lb. since being under treatment, but has had a good deal of worry
M. M.	Out-patient	3	106	100	Neck smaller	No eye signs	Tremor persists; patient is improving
L. J.	Out-patient	9½	164	132	No note	Not marked	Feels better, but there is more tachycardia and she has lost 4 lb.
S. P.	Out-patient	3	—	—	—	—	Of this patient I have no notes
E. S.	Out-patient	4	—	—	—	—	Not seen since; at last appearance there is a note to the effect that she is much better
W. A.	Out-patient	10	146	130	14 to 13½ in.	Exophthalmos less	Tremors less; feels better and is now working

Of these, 13 show a definite diminution in pulse-rate; 7 show some diminution in the neck measurement around the biggest circumference; in 2 more it is stated to be less; 6 show that the exophthalmos has become less, and in 1 it disappeared.

volunteer the statement that they "feel better." This may possibly be a psychic phenomenon, but, in view of the fact that they make progressive improvement, and one or more of their symptoms have diminished, I think it is more probably a matter of actual fact than due to suggestion.

In future we propose to have a blood count—i.e., a differential count—of the leucocytes made both before, and at different times during, treatment, and observations are being made on the excretion of creatinine and creatin as affording some idea of the endogenous nitrogenous metabolism. As I have already pointed out, in most cases of exophthalmic goitre the thymus is enlarged. In future cases we propose to attempt to X-ray this gland as well as the thyroid.

Quite recently Regaud and Cremieu have done experimental work on kittens, and have produced a progressive reduction of the thymus after a moderate amount of irradiation. Larger and successive doses reduce the thymus to such a degree that no sign of regeneration has been seen after two months. From this experimental work they have gone on to treat the thymic hypertrophy of infants and the size is said to be notably diminished after the second day.

The treatment of Graves's disease by means of X-rays is not a new one, and it has been carried out by you for some time past. I have here recorded nothing fresh, but I have attempted first to trace out the relationship existing between the thyroid in health, in Graves's disease, and again in Graves's disease after treatment with X-rays; and, secondly, to compare the medical and surgical treatment with treatment by irradiation, and to place before you, as far as I can, the results of X-ray treatment in this Department during the past twelve months.

Of the many insufficiencies and shortcomings of this paper I am well aware: for them I alone am responsible. For the rest I wish to express my thanks and gratitude to Mr. Lyster for his help and advice both in the treatment of the patients and in preparing this paper, and to Dr. J. Strickland Goodall for his help regarding the histology of the normal thyroid.

Electro-Therapeutical Section.

January 17, 1913.

Dr. REGINALD MORTON, President of the Section, in the Chair.

The Use of Condenser Discharges in Electrical Testing.

By H. LEWIS JONES, M.D.

THE subject of my discourse is the use of condenser discharges for the testing of nerve and muscle, and I hope to show that the condenser method possesses advantages which are destined to render obsolete the present method of testing by induction coil and continuous current discharges. That method has been in vogue for more than forty years, for I find that in 1868 the phrase "reaction of degeneration" was used by Erb, to whom we owe the method of testing which we have relied upon up to the present time.

The advantages which the condenser method offers are the following: Firstly, it is more rapid; secondly, it is more precise; thirdly, it gives more information; and last, but not least, it is far less painful to the person tested.

I need not waste time in enumerating the disadvantages of electrical testing by the old method. The defects of induction coil currents, the doubts as to whether the results given by different coils are strictly comparable one with another, the uncertain meaning of the loss of response to coil currents and of the survival of the response to continuous currents, are sufficient examples of the disadvantages to which I refer, and with which you are all familiar.

The principle upon which the testing of muscle by condenser discharges is based is quite simple. A condenser of known capacity is charged, from the main or from a battery, to a constant voltage, and is then discharged through the muscle. The electrodes used are those we are familiar with, and they are applied to the motor points in the usual manner, or to the distal tendon when the longitudinal reaction is being investigated. A contraction is produced when the capacity of the condenser is sufficiently great and the production or non-production of a contraction is all that is looked for. There is no need to use one's judgment to help one to decide whether the contraction produced should be called sluggish or not sluggish, and this makes for simplicity.

The character of the wave of discharge of a condenser is well defined; it is determined by the resistance of the circuit through which the discharge takes place, and by the capacity of the condenser. Its height, but not its duration, is determined by the potential to which the condenser is charged. When the charging potential is constant, and the resistance of the circuit is constant, the waves of discharge become longer as the capacity used becomes greater. Fig. 1 shows the discharge waves of four condensers whose capacities are as 1, 2, 3, 4, as they occur under the conditions of electrical testing of nerve and muscle. One can see that they differ only in their durations. This being so, we at once can recognize the possibility of using condensers for applying to a muscle a series of discharges alike in other respects but with durations which may range from the shortest possible to the longest, if only we can provide ourselves with a series of condensers having a sufficiently wide range of capacities; and if we keep our charging voltage constant, and the resistance of the patient remains constant, the discharges will be identical discharges in all other respects except that of length or duration.

It is easy to keep the charging voltage constant, and also easy to keep the resistance of the patient constant, because for these brief discharges there is no polarization, and the resistance of the body under the conditions of the case is singularly constant, being less than 1,000 ohms in the ordinary procedure of testing if the electrodes are kept in good contact and at uniform pressure. The question of the resistance of the body to momentary currents is a very interesting one, which has been well dealt with by Dubois, of Berne, and his conclusions, from which my figures are taken, may be accepted as being quite satisfactory. There is not now time to refer further to the investigations of Dubois on the resistance of the body to currents of short duration, but they may be found in a paper in the *Annales d'Electrobiologie* for March, 1899.

If we apply to a healthy muscle a series of discharges, using condensers of increasing capacity, the contractions all appear very much alike, except that the larger capacities produce stronger muscular contractions, but if we do the same to muscles which are more or less damaged we find that they do not respond to the smallest capacities, but that they still respond to the larger capacities and the process of testing with condensers therefore consists of ascertaining the magnitude of the least capacity which is required to produce a visible contraction in a given muscle. When a charging potential of 100 volts is used, the normal, or almost normal, muscles react with capacities between 0·01 and 0·08 micro-farad (hundredths of a micro-farad), the muscles with partial reaction of degeneration react between 0·1 and 0·8 (tenths of a micro-farad), and the muscles with complete and profound reaction of degeneration may require 1, 2, 3 micro-farads, or even more, before they respond.

Translated into length of waves, this means that a muscle responding first with a capacity of 1 micro-farad requires a wave one hundred times as long as that necessary to excite a normal muscle, which reacts to 0·01 of a micro-farad. By using a battery of condensers ranging from 0·01 to 2 micro-farads with ten intermediate steps of capacity, we are placed in the possession of a range of twelve different wave lengths, and soon discover, on practical trial, that muscles occur which may require any one of these different lengths of wave for the production of their first visible contraction. We thus acquire a method which allows of the ready recognition of twelve different degrees of muscular excitability, instead of the three grades of "normal," partial R.D. (reaction of degeneration) and complete R.D., with which we have hitherto been satisfied. The values which I have taken for the twelve capacities are as follows :—

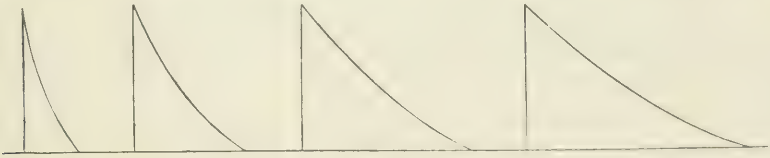

FIG. 1.

Discharge curves of condensers when their capacities are in the ratios of 1, 2, 3, 4, with constant voltage and constant resistance.

No.	Capacity	Discharge duration
1	0·016 micro-farad	[illegible] second
2	0·025 ,,	[illegible] ,,
3	0·05 ,,	[illegible] ,,
4	0·062 ,,	[illegible] ,,
5	0·08 ,,	[illegible] ,,
6	0·125 ,,	[illegible] ,,
7	0·25 ,,	[illegible] ,,
8	0·33 ,,	[illegible] ,,
9	0·5 ,,	[illegible] ,,
10	0·66 ,,	[illegible] ,,
11	1·0 ,,	[illegible] ,,
12	2·0 ,,	[illegible] ,,

These durations of discharge are approximate only, but serve to indicate the general order of the values. If these durations are compared with the wave lengths of induction coil discharges, one is struck by the fact that the small capacities, to which normal muscles readily respond, give waves which are much shorter in their duration than those of medical coils. The waves of most medical coils last about $\frac{1}{400}$ second, and even if the effective part of them is only half of this, say $\frac{1}{800}$ second, that duration is still too great to reveal the slighter degrees of deviation from the normal, and as is doubtless apparent to all those who have thought about the subject, we reach the conclusion that muscles have been reported as normal during all these years of electrical testing, although they have, in many cases, been by no means normal, the reason being that we have been unable to appreciate their real condition, much less to express their abnormality in any numerical way.

The electrical testing we have been using, which I now propose to call the old way of testing, has been based upon the interrogation of the muscular contractility by applying to it the short waves of the induction coil and the long waves of the closure of the continuous current. We have not known the wave length of either, or at least we have made no attempt to control it, and our short waves have not been short enough, while our long waves have been too long.

Minimum capacity, micro-farads		Minimum wave duration seconds
0·02 to 0·05	Thigh muscles	$\frac{1}{20000}$ to $\frac{1}{8000}$
0·08	Interossei	$\frac{1}{4800}$
0·1	Gastrocnemius Soleus	$\frac{1}{3200}$
0·25	Peronei	$\frac{1}{1600}$
1·0	Tibialis posticus Flexor communis digitorum	$\frac{1}{700}$
2·0	Tibialis anticus Extensor longus digitorum Extensor proprius hallucis	$\frac{1}{200}$

This is a report of a condenser test of the leg and foot muscles of a patient with partial paralysis of one leg, due, probably, to a lesion of the grey matter of the lumbar enlargement. The old test showed complete R.D. in the anterior tibial group, poor normal responses in the peronei and the intrinsics of the foot, and partial R.D. in the calf muscles. It can be seen that the condenser test gives a much fuller account of the condition of his muscles, and one which can be drawn up in the form of a report expressed in simple numbers. The figures are given in fractions of a micro-farad and are also translated into figures of time in fractions of a second. Dr. Cumberbatch, who has helped me in my work with condenser testing, has promised to give other examples, at the end of my paper, of results obtained by him at St. Bartholomew's Hospital, and Dr. R. S. Woods, of the Electrical Department of the London Hospital, has also given me the most valuable help and, I hope, will also contribute to our collection of facts directly.

It would be easy to devote a considerable time to a discussion in detail of the various points of interest which one can see to be developing out of the study of the testing of muscles by condenser discharges, but I do not wish to encumber my subject, and on this occasion it is better, I think, that we should confine our attention to the most practical and fundamental points.

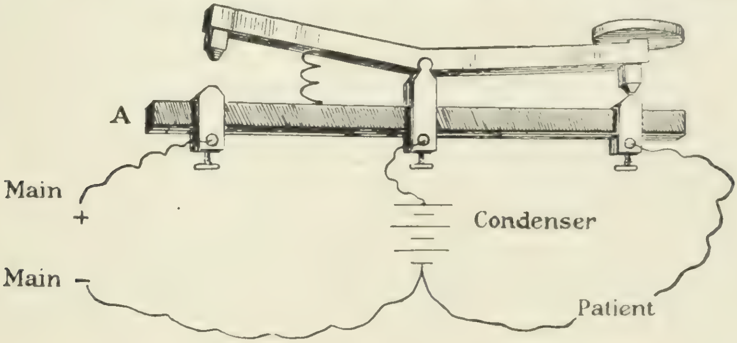

FIG. 2.

Plan of Morse key for charging and discharging a condenser.

I have here an apparatus for testing by the new method. It consists of a box much like a battery box, with twelve studs, and containing twelve condensers of the capacities already mentioned, with a moving arm for bringing any one of them into action. The condensers are charged from the source and discharged through the patient by a Morse key, an instrument which makes two alternative contacts in turn, the one for the charge and the other for the discharge. The Morse key may be adapted to a testing handle or used as a metronome, or converted into a special revolving commutator, and I have here examples of each. My own preference is for the metronome form, with wires dipping into mercury cups. When the contacts are kept clean it makes and breaks the circuits very well, but care must be observed in its use, for the patient may get a severe shock if the dipping-in wires are so long as to connect both the end cups at one time, for then they form a bridge which can convey the full current of the charging source to the patient. This danger can be avoided by the observance of the rule of always making trial of the current on one's self before touching the patient.

The use of the metronome in all kinds of electrical testing is to be recommended. I have so used it for several years, and consider it more handy than an electrode fitted with a closing key on the handle; and the tick-tack of the metronome is useful as it gives an audible signal to the operator which tells him when the muscular contraction should appear, and also by the rhythmic sequence of the impulses one is enabled to distinguish muscular movements due to the testing current from any other accidental voluntary movements of the patient.

I should now like to explain my reasons for choosing the particular values of the condensers which make up the testing box. They are arbitrarily chosen, and it is possible that some day other values may be preferred, but in an investigation of this kind one has to begin by feeling one's way, for there are not many indications in the literature to guide one in the choice of the best values.

Mr. Leslie Miller has given me great help in the manufacture of the boxes, of which I have used several with different arrangements of condensers, and the figures placed before you represent my present views as to what constitutes a suitable set. In the first place, I think that a range of twelve capacities is enough to begin with, and the chief question has been how best to distribute the values in a set of twelve, beginning with about 0·01 and ending with 2 micro-farads. So far most of the interesting differences which I have met with in the muscular responses come between 0·05 and 1 micro-farad.

In view of the difficulties of obtaining small capacities which are cheap and reliable, I have adopted a special plan of construction—namely, that of arranging my capacities in series. There is a formula for the capacity of a number of capacities in series which is not difficult to utilize. It is this:—

$$C = \frac{1}{\frac{1}{C_1} + \frac{1}{C_2} + \frac{1}{C_3} + \frac{1}{C_4}\ \&c.}$$

C being the capacity of the combined series, and C_1, C_2, C_3, &c., being the capacities of the components. Thus the capacity of two condensers in series, when each is of 2 micro-farads, is 1 micro-farad, and my box is made up of the following units—viz., four of 2 micro-farads, two of

1 micro-farad, four of $\frac{1}{4}$ micro-farad, and two of $\frac{1}{20}$ micro-farad. These, in series, give the fractions contained in the box. Except for the smallest they are commercial sizes, they are uniform in capacity, neatly made, free from leakage, and in the series arrangement their individual errors tend to neutralize out. This is a valuable point. I think it so important that different investigators should have identical instruments that I favour this special method of construction. The manufacture of condensers, or rather the standardizing of them, is very difficult for such small sizes as 0·01 or 0·02 micro-farad, and these sizes are not made commercially as are the larger ones of $\frac{1}{4}$ micro-farad and upwards. In my arrangement the smallest size used is 0·05 micro-farad, which can be made reasonably easily. The voltage I have chosen is 100 volts, but other voltages may be used, and if other voltages are used the results obtained can be made comparable by expressing them all in durations of time. I hope soon to prepare a table with which we can compare 100 volts with 120, 200, and 240 volts.

With 200 volts instead of 100 the only change necessary in the construction of the testing-box would be the provision of one more condenser at the smallest end. On 240 volts a capacity of 0·005 gives the threshold of contraction, as I am informed by Professor Cluzet, who tells me he uses 240 volts in his condenser testing.

There are certain points to be considered in the special case of muscles showing a profound reaction of degeneration. Cluzet touches on this point, and tells us that there are some cases of this kind in which no response is produced even by the largest capacities, although a response can be excited by the closure of a continuous current.

I have also found this to be the case and have tried to get over the difficulty, the essence of which is as follows: In extreme R.D. the excitability of the muscle has fallen so much that the condenser discharge even of 2 micro-farads ($\frac{1}{200}$ second) is still too short to excite a contraction. With larger condensers one could get longer waves, but with 2 micro-farads at 100 volts the discharges are already disagreeably strong, and provoke much movement in neighbouring healthy muscles, which tends to obscure the clearness of the test. I therefore decided to seek a means of prolonging the duration of the discharge from the largest-sized condenser, without increasing the shock to the surrounding healthy muscles, and the best way of doing this is by the introduction of a resistance in series with the patient. A very good way is as follows: Raise the charging voltage from 100 volts to 200, which is easy on most electric lighting systems. Introduce into the patient's circuit a resistance of several thousand ohms and proceed as before. The initial shock is reduced by the added resistance, and the duration of the discharge is extended. Thus with 100 volts and 2 micro-farads, and the patient's resistance taken at 1,000 ohms, the duration will be $\frac{1}{200}$ second, with 1,000 ohms added it will be $\frac{1}{100}$ second, and with 4,000 ohms added the duration will be five times as long as at first, or $\frac{1}{40}$ second, but the initial value of the discharge will be proportionately reduced, because the charge in the condenser is obliged to escape more slowly. The effect upon healthy muscles will be much reduced, and the effect upon the R.D. muscles will be reduced to a less degree. By raising the voltage to 200 we keep our long waves and double their initial value, and in general will succeed by this way in provoking the looked-for contraction, even in very degenerate muscles.

This procedure is quite easy in practice, and Dr. Cumberbatch has been testing its value upon some cases, and has some facts bearing on the matter which I hope he will contribute to the discussion directly. In some such way as this we may hope to overcome the difficulty of the non-response of very degenerate muscles to condenser discharges.

And now, gentlemen, I want you to understand that what I have been telling you to-night is not in any way my discovery. All I have done is to put before you, I hope in a simple and clear manner, the present position of condenser testing. The position is not one which has been rapidly attained, and many workers have contributed to its attainment. Only to mention a few, I would at least name to you Boudet de Pâris (Paris, 1888), Hoorweg (Utrecht, 1899), Dubois (Berne), Zanietowski (Prague), Doumer (Lille), and Cluzet (Lyons).

Much of the early work with condensers dealt chiefly with the physiological and the mathematical aspect of the problem, and the papers, involving, as they do in some instances, a knowledge of differential equations, logarithms, and trigonometry, and dealing much with the search for a mathematical formula to express the co-efficient of excitability of nerve and muscle, have perhaps tended to discourage clinical workers from taking up the subject for practical use. It was a paper by Professor Cluzet, in the *Paris Médical* of April, 1912, which awakened in me a lively interest in the possibilities of practical condenser testing. In that paper a form of testing apparatus for clinical use is figured, the general effect of condensers of different capacities upon damaged muscles is stated, and the essential principles of the method of practical clinical testing are described. In what I have given you to-day I have tried to expand that subject, to amplify some of the details, to select definite values for the condensers used, and to contribute some results of my own experience, in the hope of calling the attention of workers in this country to an important new development.

There is much work to be done in the way of testing muscles by both methods, the old and the new, in order to compare them together. This has hardly been begun yet, but I have already seen enough to convince me that the use of condensers in testing will yield much new knowledge, valuable both for diagnosis and prognosis, and will entirely supersede the old method of electrical testing. It will also probably bring out some new points of diagnostic value which we do not as yet suspect, and quite possibly will show us some discrepancies from what we now regard as the settled order of muscular excitability in disease. It remains for us in this country to advance still further the progress which has already been made.

Other ways of using condensers for testing have been proposed, as, for instance, by varying the voltage, instead of the capacity; or by using two condensers, one large and one small, and observing the effect of introducing resistances. But to my mind none of these are so simple as the method I have laid before you of varying the capacity in a regular series, while maintaining everything else uniform.

Those who are interested in pursuing these methods further may consult the numerous papers of Zanietowski, and should also see a paper by Doumer in the *Annales d'Electrobiologie* for April, 1911.

RESULTS OBTAINED BY TESTING BY MEANS OF CONDENSER DISCHARGES, AND THEIR COMPARISON WITH THOSE OBTAINED BY THE OLDER METHOD.

Dr. E. P. CUMBERBATCH demonstrated on the epidiascope charts of testings of patients. The first table showed the results obtained from a test made both by means of condenser discharges and by currents from coil and cells. The test was made upon certain muscles of a patient suffering probably from progressive muscular atrophy. Some of the muscles, when tested by the older method, gave normal reactions; others gave a partial R.D.; others a complete R.D. But when the same muscles were tested by discharges from condensers of varying capacity, it was possible to place a number against each muscle, as seen in the table, representing the capacity of the smallest condenser to the discharge of which the muscle would contract. The muscles contracting to the larger capacity condensers gave a complete R.D. reaction; those contracting to the smaller capacity condensers gave a normal reaction. But it was on the remaining muscles that the condenser method gave the most helpful information. These muscles responded to coil currents, but with currents from cells it was often a matter of personal judgment whether the response was sluggish or not. These muscles responded to discharges from different condensers which were all of medium capacity.

TABLE I.

	Condenser charged to 100 volts		Reaction to coil and cells
Hand	Intrinsics (median nerve)	2·00	Complete R.D.
	Intrinsics (ulnar nerve)	3·00	
Forearm	Flexor carpi ulnaris	0·05	Probably partial R.D.
	Flexor carpi radialis	0·07	
	Flexor sublimis digitorum	0·50	
	Extensor carpi ulnaris	0·10	
	Remaining extensors	0·25	
Arm	Triceps	0·10	Partial R.D.
	Biceps	0·50	
Face	Facial muscles	0·02	Normal
Foot	Interossei	0·07	Normal
	Extensor brevis digitorum	0·07	
Leg	Extensor communis digitorum	0·25	Partial R.D.
	Tibialis anticus	0·50	
	Peronei	0·70	
	Calf muscles	0·25	
	Tibialis posticus	0·25	
Thigh	Quadriceps	0·25	Probably partial R.D.
	Adductors	0·10	Partial R.D.
	Hamstrings	0·50	

The second table shows the result obtained from a test made upon some of the leg muscles of a patient who was suffering from the results of anterior poliomyelitis. In the first part of the table is recorded the results obtained when testing by the older method. When tested by condensers charged to 100 volts (as in the preceding case) it was found that none of the muscles of the left leg would respond even when the largest capacity condenser was used. Evidently these muscles were so torpid as to require a discharge still slower than that which the largest capacity condenser could give. By introducing resistances, successively increasing in amount, into the discharging circuit, and charging the condensers to 200 volts instead of 100 volts, it was found that some of these torpid muscles would respond until, with 5,000 ohms in the circuit, all responded.

TABLE II.

	Reactions to coil and cells	
	Right side	Left side
Tibialis anticus	Weak normal	Complete R.D.
Extensor longus digitorum	Weak normal	Complete R.D.
Extensor longus hallucis	Weak normal	Complete R.D.
Peronei	Weak normal	Complete R.D.
Calf muscles	Complete R.D.	Complete R.D.

	100 volts, no extra resistance		200 volts, 1,000 ohms in circuit		200 volts, 2,000 ohms in circuit	
	Right	Left	Right	Left	Right	Left
Tibialis anticus	0·02	No contraction obtained even with the largest capacity condenser	0·01	? 2·00	0·01	2·00
Extensor longus digitorum	0·05		0·02	No contraction	Not tested	No contraction
Extensor longus hallucis	0·02		0·01	1·00	Not tested	1·00
Peronei	0·02		0·01	No contraction	Not tested	No contraction
Calf muscles	0·25		0·07	No contraction	Not tested	No contraction

	200 volts, 3.000 ohms in circuit		200 volts, 4,000 ohms in circuit	200 volts, 5,000 ohms in circuit	
	Right	Left		Right	Left
Tibialis anticus	0·02	? 1·00	Results similar to those shown in preceding column	0·02	2·00
Extensor longus digitorum	Not tested	2·00		0·05	2·00
Extensor longus hallucis	Not tested	0·50		0·05	0·50
Peronei	Not tested	2·00		0·02	2·00
Calf muscles	Not tested	No contraction		0·10 (very slight)	3·00

DISCUSSION.

Dr. R. S. WOODS (London Hospital) said that Dr. Lewis Jones had given him the opportunity of using the condenser method in the case of some of his (Dr. Woods's) patients. He had used the method of condenser testing only with Dr. Lewis Jones, but from what he had seen of it he realized that it would revolutionize muscle testing. It seemed that it not only made more definite the line between normal and abnormal muscle, but enabled one to divide up the muscles which formerly were classed as abnormal into a graded series, from those verging on to the normal to those in which, with even large condenser discharges, no response at all was obtained.

Dr. Feld. Bailey (Brighton) said that, seventeen months ago, he severed his own left ulnar nerve. It was sutured at once and healing was satisfactory; since the operation recovery had taken place in the usual ordinary sequence, faradic response returned in the intrinsic muscles of the hand eleven months after operation. Four months later, fifteen months after suture, there was but slight difference to be discovered, as to faradic response, in the left hand intrinsic muscles as compared with the right. However, when tested by Dr. Lewis Jones with his condenser apparatus, while the sound hand intrinsics responded to the smallest condenser discharge (i.e., with the switch on No. 1 stud, about 0·01 micro-farad), the affected hand intrinsics did not respond lower than No. 6 stud (? about 0·1 micro-farad). One month later the same muscles responded with the switch on No. 4, showing a definite improvement in one month which could not otherwise have been demonstrated. He obtained a similar apparatus from Mr. Miller and had satisfied himself that the method had a considerable future before it, both because it showed these delicate differences in muscle response and also because it was painless.

The President (Dr. Reginald Morton) said it afforded him great pleasure to offer Dr. Lewis Jones his congratulations on the work he had done in connexion with this important subject. The question of testing muscles by means of condenser discharges had been "in the air" for a long time; he believed he heard of it as soon as he took up electrical testing. But it was then so hedged about with logarithms and other difficulties that it was apt to frighten a non-mathematical person. Until he had a conversation on the matter with Dr. Lewis Jones recently, he had only a vague idea as to how it could be used. The greatest credit was due to Dr. Lewis Jones for the simple and practical way in which he had developed this matter. It was thus now a practical method, quite simple and comprehensible to clinicians. He foresaw that the time was at hand when one's ideas as to degeneration of muscle must be readjusted. They had been dissatisfied in the past with their three stages—normal, partial R.D., complete R.D.—and instead of this coarse division it would now be possible to separate out muscle responses more exactly, and perhaps even to number them 1, 2, 3, &c. He had often felt dissatisfied when sending in reports with the mere statement that certain muscles showed more or less R.D. It seemed to him crude, and detracted much from the pleasure and interest of electrical testing. What had specially appealed to him was the author's method of lengthening the wave. In conversation, Dr. Lewis Jones had just given him a rough but simple method of keeping in one's mind what constituted a normal and a partial degeneration. It was that one could call response to hundreds of a micro-farad normal; to tenths of a micro-farad partial, and to micro-farads only, complete degeneration. That was a convenient guide to bear in mind. He proposed that the Section accord its hearty thanks to Dr. Lewis Jones.

Dr. Lewis Jones, in reply, thanked the members of the Section for their appreciation of his efforts to explain the subject of condenser testing. He was sure it would be found worthy of attention. A great value of the method was the abolition of much of the pain usually associated with electrical testing, and which was distressing to operator as well as patient. At the hospital there were so many children the subjects of infantile paralysis in whom it was a great pleasure to see smiles substituted for tears during the process of testing. When the capacities required reached 1 or 2 micro-farads there was some stinging sensation, but most of the testing called for did not hurt. Dr. Bailey had reminded the meeting of his own case, and he was the first patient upon whom he had had the opportunity of testing the method after he got his first condenser set. Dr. Bailey came and said he had recovered normal reactions in his muscles, but the condenser test showed a considerable deviation from a true normal. A month later the condenser enabled him to recognize an improvement in this respect, which was not recognizable with induction currents. Happily, the whole question of condenser testing was so simple that it had not needed a long paper to explain it. He hoped that in a few months' time individual members of the Section would come forward with reports of cases which they had tested, both with the old and the new methods, and so contribute to a more detailed knowledge.

Secondary X-rays from Animal Tissues.[1]

By S. Russ, D.Sc.

Evidence is gradually accumulating to show that the biological effects associated with different types of radiation (those from radium and X-rays) run parallel with the ionizing power of the rays. The very easily absorbed rays, efficient from the ionization aspect, are responsible for more marked biological effects than are those of a penetrating character.

When a primary beam of X-rays is incident upon a substance, it is found that the substance emits in all directions a secondary X-radiation. This radiation may be identical in character with the primary beam, in which case it is known as scattered primary radiation; it may, however, have properties peculiar to the constituent elements of the substance. Barkla has shown that all elements of higher atomic weight than forty emit a "characteristic homogeneous radiation" when stimulated by a suitable primary radiation; the lower the atomic weight, the softer is this characteristic radiation. Whiddington has produced evidence that aluminium (at. wt. 27) emits a characteristic radiation and, as anticipated from Barkla's work, it is of a very soft type.

The tissues of the body consisting for the most part of elements of low atomic weight, it is not to be anticipated that the passage of a beam of X-rays through the tissues would excite any easily recognized characteristic homogeneous rays in them; consequently, if any differences in the secondary radiation from various tissues are detectable, these differences may, in all probability, be attributed to their variable mineral content.

Some preliminary measurements have been made of the secondary rays emitted by various tissues (of the sheep) when traversed by a primary beam of X-rays issuing from an ordinary focus bulb under a spark gap of 9 cm.: this corresponds to a radiation of medium hardness. The method by which this has been done may be seen from fig. 1. A primary beam from the anode strikes a specimen of tissue (T), placed in a thin mica dish 1·5 cm. deep, fixed 10 cm. vertically below a gold-leaf electroscope (E). A portion of the secondary rays emitted by the tissue emerges through a hole in a lead screen, which may be covered over with sheets of aluminium (A) when required. These rays enter the electroscope, ionize the air in it and cause the gradual fall of the gold leaf, the rate of motion of which is a measure of the extent of the ionization. The electroscope was, with the exception of a light framework, made of tissue-paper, the object being to avoid the production of any tertiary rays by the secondary rays which are under observation. The ionization having been measured, sheets of aluminium were placed over the aperture and the gradual reduction in the ionization measured. In this way the penetrating power of the secondary radiation was found.

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[1] Part of this work was done during the tenure of a Beit Memorial Fellowship (communicated at the meeting of the Section on December 20, 1912).

In fig. 2 will be seen the curves of absorption for water, blood, and bone. The two former are, under these particular experimental conditions, indistinguishable in character and, as will be seen later, indicate that the primary beam has been scattered by these substances without any alteration in its character. The sudden fall in the ionization caused by the rays emitted from powdered bone is probably due to the calcium contained in it. The characteristic homogeneous rays from calcium are, according to Barkla, very soft and would be more than half absorbed by the 10 cm. of air separating the surface of the material from the base of the electroscope. Except for this soft constituent, the secondary radiation is found to be scattered primary radiation.

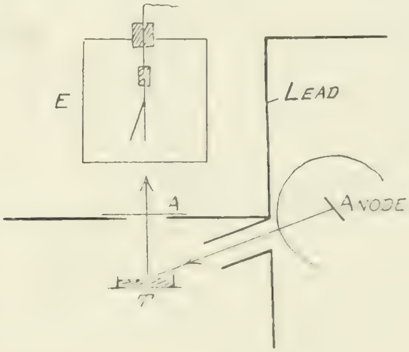

FIG. 1.

On the same diagram are shown two examples of the characteristic homogeneous rays excited in metals by the same primary beam. It is seen that this type of radiation from lead and bismuth is very easily absorbed. These selections have been made as being of special clinical interest. Lead is occasionally used for screening purposes, and the possibility of danger in this procedure is apparent when it is remembered that the production of this soft radiation continues through the different layers of a screen, and the emergent portion may be of considerable intensity. Such secondary rays are also excited by the gamma-rays of radium. The introduction of bismuth into the body for radiographic purposes has another aspect of interest, in that it is a localized source of a very soft radiation upon the passage of the primary beam.

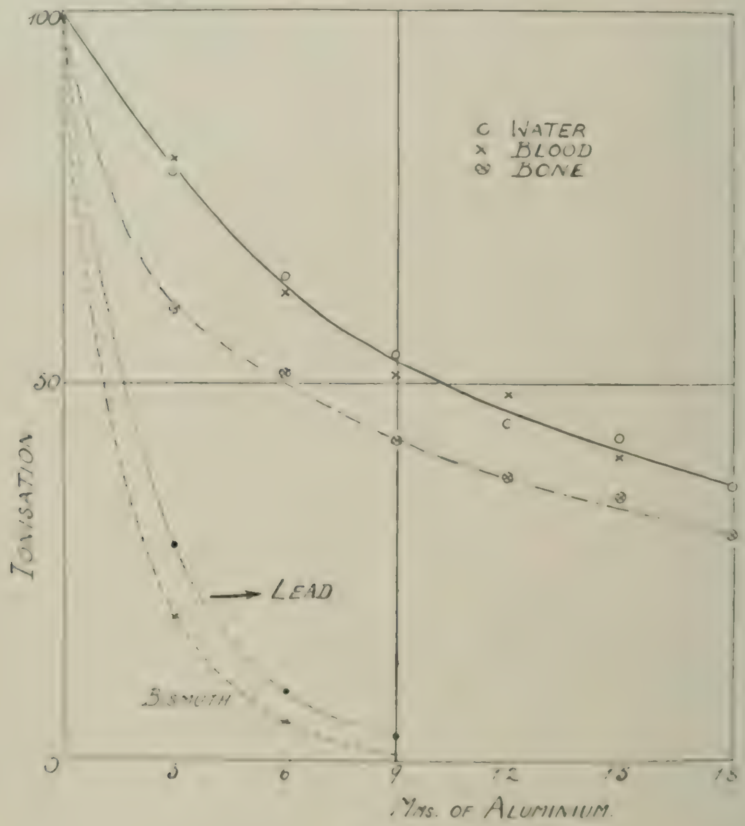

Fig. 2.

In fig. 3 the full line curves I, II and III represent the curves of absorption by aluminium of the secondary rays from spleen under various experimental conditions. Curve I is for natural spleen, and it will be seen by comparison with fig. 2 that the secondary radiation does not differ markedly from that of water and blood. This similarity probably being due to the large amount of water in the tissue normally, the specimen was treated with acetone, which removes the water and fat; the extract was dried and powdered, and its secondary rays again measured, with the result shown in curve II. There are now indications of a soft constituent among the purely scattered rays, the presence of which was previously difficult to be sure of, owing to the preponderance of the scattered rays from the water. Upon incineration of the spleen powder this soft radiation was now found to constitute the bulk of the measured secondary radiation, *vide* curve III, the remainder probably being due to scattered rays from the carbon. The very soft secondary rays are probably attributable to the iron present in the spleen, the presence of which in the incinerated material was detected without difficulty by the ordinary chemical tests. Owing to lack of

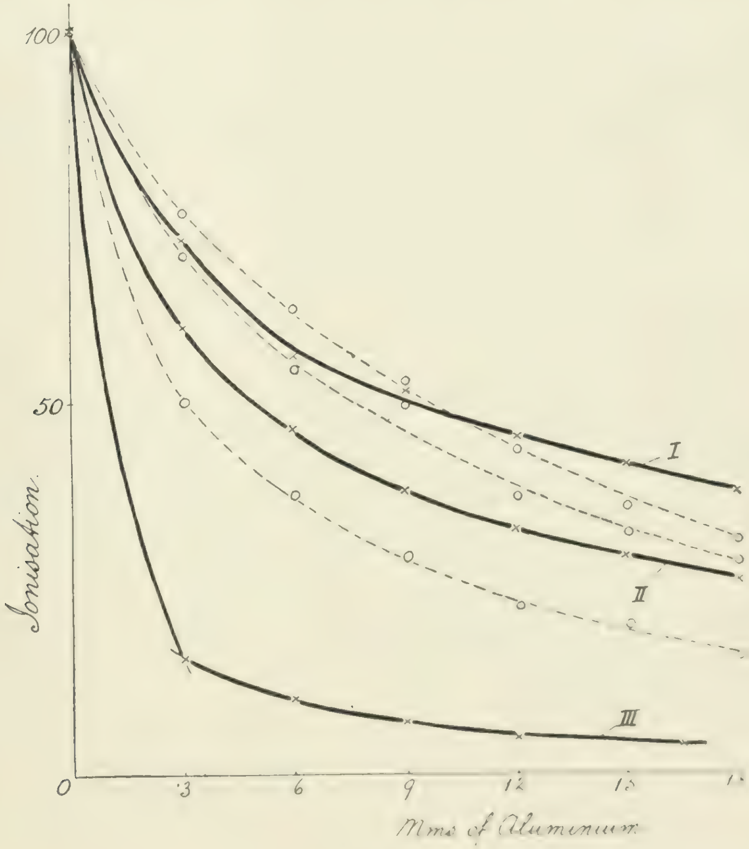

FIG. 3.

observations for very small thicknesses of aluminium, it is not possible to give the co-efficient of absorption for these soft rays. In the same figure the dotted curves represent the secondary radiation from kidney, when measured under the conditions detailed for spleen. It will be seen that the changes in the character of the measured radiation are less marked than in the previous case.

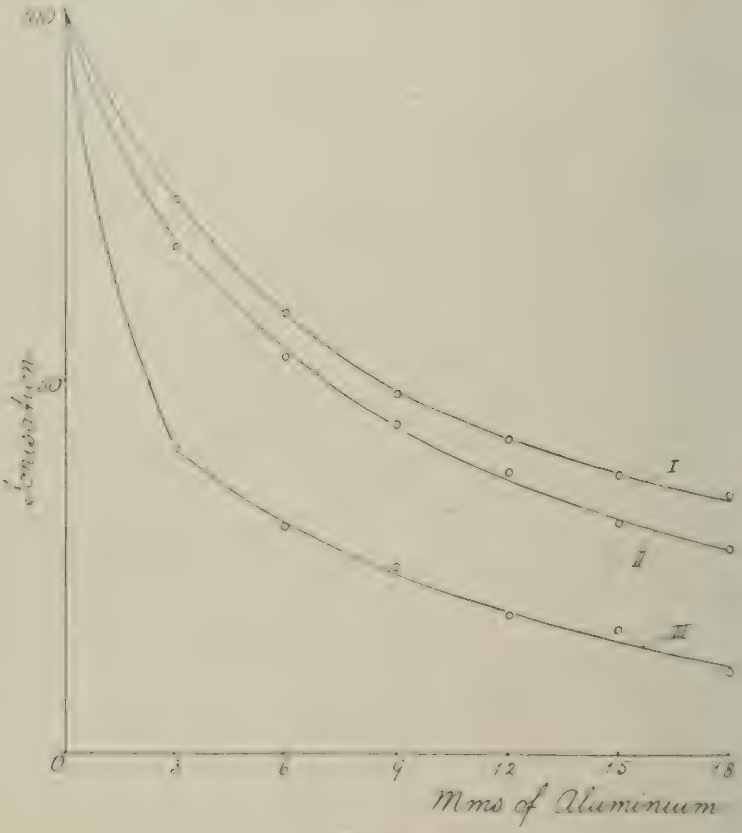

FIG. 4.

In fig. 4 the curves I, II and III represent the results obtained by a similar procedure with normal liver (I), then dried (II) and incinerated (III). The changes are similar to those obtained with spleen, but they are not so marked, although rather more significant than in the case for kidney. When measurements on these lines are made of the radiation from blood, the soft radiation, common to the above tissues, is also apparent.

All these observations have been made with a bulb running at a spark-gap very near to 9 cm. From some previous measurements[1] it has been shown that when the soft constituents of the beam of X-rays issuing from a bulb running at 8·5 cm. spark-gap were cut out by 0·56 mm. of aluminium, the bulk of the remaining radiation had a mean co-efficient of absorption, λ 5·6 cm.$^{-1}$ From other data also obtained for spark-gaps of 4·5 and 19 cm. it is found, by interpolation, that at 9 cm. this constant would have the value 5 cm.$^{-1}$ very nearly. When the secondary rays from the substances (with their normally contained water) under examination are examined *in the same manner*, it is found that the co-efficient λ has the following values:—

Secondary radiation from	Value of λ in cm.$^{-1}$
Water—Blood	4·8
Bone	4·4
Liver	4·6
Kidney	5·6
Spleen	3·6

With the exception of spleen these values do not vary more than about 10 per cent. from the value 5, which indicates that the secondary rays measured were practically identical in character with the primary radiation falling upon the tissues. The low value (3·6) for the spleen suggests that apart from the very soft radiation due to the iron, the secondary rays are appreciably more penetrating than for the other cases. This may be due to some selective absorption of the softer constituents of the primary beam by the spleen. This selective action, which has been shown by Barkla and Sadler[2] to be a general one, would have the effect of hardening the original primary beam, the indications of which would be a diminished value of λ. This view is put forward in lieu of suggesting the presence of some substance normally present in the spleen which emits a hard type of secondary rays.

Of the various tissues examined, bone and spleen give the most marked evidence of secondary radiation other than scattered primary rays. I am informed that the irradiation of the spleen in cases where it is enlarged may be followed by a reduction in its size. To what extent or in what manner the particular secondary rays in question are effective in this process of reduction it is not yet possible to estimate.

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[1] *Journ. of Röntgen Soc.*, 1912, viii. No. 31.

[2] *Philosoph. Mag.*, May, 1909.

but it seems not unlikely that there is some association between this clinical fact and the especially favourable conditions for its irradiation that this organ exhibits.

At the meeting on December 20, 1912, a demonstration was given by the author of the secondary radiation from various substances, and in connexion with the question of the rays given off from metals he supported Dr. R. Morton, who had advocated the abandonment of metal screens in clinical practice. Every piece of metal near a patient being irradiated is potentially an X-ray bulb, and usually a very soft one. The conclusion seems justified that the ideal screen is one composed exclusively of the light elements; compressed paper fulfils this condition.

Electro-Therapeutical Section.

February 21, 1913.

Dr. Reginald Morton, President of the Section, in the Chair.

A Discussion on the Use of X-rays in the Diagnosis of Pulmonary Tuberculosis.

Opened by Sir Richard Douglas Powell, Bt., K.C.V.O., M.D.

There has been no greater gain to science in recent times than the discovery of the Röntgen rays, and the promptness with which that discovery has been utilized in the interests of medical science, the zeal, ability and enterprise, not unattended with personal danger and even sacrifice of life and limb, with which "truth in the inward parts" has been pursued will form a bright record amongst the many great achievements of modern medicine. I do not, however, come here to extol or to question the value of X-ray work, but having been asked to introduce this discussion from the point of view of the general clinician I could not do so without expressing my appreciation, as one who has taken no part whatever in the work, of the many valuable contributions it has made to improved accuracy in diagnosis and precision in treatment. Perhaps I may usefully at once state the position that X-ray examination seems to me to hold with regard to the diagnosis of pulmonary tuberculosis:—

(1) Its object is to concentrate in one view the physical conditions present in the lungs and thus to replace, or correct, or confirm the findings of physical examination by percussion and auscultation. It is contended that the X-ray view is more exact and comprehensive than can be given by any other physical examination.

(*a*) There is, I think, some truth in this, especially from a screen view with perfect instruments and skill, where the lungs are observed in action and the penetrability to air of their several parts can be examined.

It is not impossible that a cinematograph film might be prepared from a succession of flash X-ray photographs which would demonstrate to a class a living picture of the thoracic organs.

(*b*) Much importance is attached to the position and relative fixity of the diaphragm on the affected side as shown by the screen even in early tuberculosis. I have for many years been in the habit of testing this mobility of diaphragm by maintained percussion at the extreme limits during inspiration and expiration, and do not, as a rule, find it altered in very early stages except where there has been pleurisy or the lower lobe is involved.

(*c*) Impaired movement at the apex can perhaps be better observed by ordinary inspection.

(*d*) Auscultatory examination appears to me to have an advantage over photography by the estimation of crepitant sounds and the character of râles, or their absence, as evidence of activity.

Is an earlier stage of tuberculosis to be recognized by the radiographic method than by auscultation? In this, as in the similar claim for bacterial examination of sputum, it is a question of give and take, the one method is or should be supplemental to and confirmatory of the other.

(2) It is maintained by Dr. Jordan (who has, I believe, given us the latest word on this subject) that he has demonstrated by X-ray examination that:—

(*a*) All persons—even those in apparent health—have peribronchial tuberculous glands and streaks of fibrous thickening radiating from them.

(*b*) In 118 out of 150 cases of undoubted pulmonary tuberculosis which he examined consecutively, the disease had started in the neighbourhood of the peribronchial glands, and secondary peribronchial infection followed, spreading to the periphery. In fifty-nine of these cases the tuberculous lesion was limited to the peribronchial (mediastinal) region and its near neighbourhood.

(*c*) In only thirty-two cases could the lesion be said to be restricted to the apices.

I should like to know how far these views are endorsed by other radiographic experts. They are not in accord with my experience, but I have some difficulty in contesting them, because Dr. Jordan states (February, 1912) "At the present day the X-ray appearances are definite and exact; the X-ray findings are facts," which leaves little to be said.

But I will state my difficulties. And first I will quote from

Dr. Jordan's most recent paper,[1] a reprint of which he has kindly sent me, my attention having originally been attracted to his paper in the *Practitioner* for February, 1912. "The glands and other structures at the hilus have to deal with a continuous tuberculous invasion throughout life. Many are able to cope with the invasion, but in the case of some the tissues are not able to offer effective resistance, or the tissues are receiving a greater number of tubercle bacilli than they can destroy; these become the subjects of pulmonary tuberculosis. In every case the invasion takes place by way of the hilus. In my former paper I showed that in at least 40 per cent. of cases of phthisis the disease commences as definite peribronchial mottling. From the hilus the disease spreads in all directions, but most rapidly along the ascending and descending branch of the main bronchus. Sooner or later the disease reaches the apex (by way of the ascending bronchiole); it then extends rapidly at the apex, so that very soon the apex appears, clinically, to be the chief seat of the invasion."

The recognition of disease in health is an interesting rather than a useful achievement, but there are other obstructions and infections in the bronchial glands besides tubercle. One cannot feel surprised at the rarity with which the bronchial glands are found to be quite normal. It must be borne in mind that they are the dustbins of the broncho-pulmonary tracts. They are also the lethal chambers of tubercle and other organisms. But to maintain that they are always or generally tuberculous is, I believe, quite beyond the truth.

(*a*) Are X-ray findings facts? I have always regarded them as shadows which have to be rightly interpreted.

(*b*) Is there any post-mortem evidence in favour of the view that tuberculosis generally, or at all frequently, or even ever, commences in the bronchial glands[2] and extends therefrom to distant centres in the lung? I am aware that children, and perhaps adults, may get their bronchial glands very early affected, although rarely primarily, more often secondarily to bronchial attack, and that tuberculous infection not uncommonly supervenes. A general pulmonary tuberculosis may result through conveyance of tubercle bacilli from the glands to the blood-stream, or the tuberculosis may have an independent infective origin. But that infection should thus locally spread against the lymph-currents from the glands into the lung is new to me.

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[1] "On Peribronchial Phthisis," *Brit. Med. Journ.*, 1912, ii, p. 481.

[2] This is not quite the contention of Dr. Jordan, as I ascertained in conversation with him afterwards, he believing that the first portions of lung involved are peribronchial portions near the hilus which lead to the gland involvement and extend also peripherally. The quotation I have read does not seem to me to carry this meaning, and Dr. Murray Leslie and others would, I believe, maintain the primary gland origin.

(*c*) I cannot say that I am convinced by Dr. Jordan's photographs, which are very beautiful impressions: it is the interpretation with which I am at issue.

(3) X-ray impressions are indelible—like all instrumental records—and one is inclined to say, "There the thing is, and it is no use arguing about it. The machine just records and cannot make a mistake." But a very little experience of instrumental work, cardiographic, sphygmographic, or radiographic, makes us familiar with the fact that the man behind the machine—be he never so skilful—may make mistakes, and still more readily may make misinterpretations; and instruments, therefore, must be kept as our servants, not our masters. "Things are not (always) what they seem."

With the beautiful and costly instalments now available at our great hospitals and the great experience and profound skill of the operators, mechanism-mistakes will become more rare, and if due caution be taken to collate all the other evidences relating to each individual case the physical signs depicted will be rightly construed.

In some few cases of early differential diagnosis in regard to pulmonary tuberculosis, I believe X-ray examination to be valuable. In many other conditions in the course of tuberculosis its aid may be sought with advantage.

(*a*) On the first point I have in mind two cases in which the aid of X-ray examination would have been useful in recognizing foreign bodies simulating tuberculosis, and I have a photograph kindly taken for me by Mr. Lyster of an interesting case of gold-miner's fibrosis and (?) tuberculosis, which I will show to-night.

(*b*) X-rays are of much value, taken at certain intervals, in recording advance or retrogression of lesions under treatment, and especially institutional treatment; great caution, however, is necessary in distinguishing fibrosis radiating from improving lesions from advance of disease. They are also of value in defining the outlines of empyemata, of large cavities in which operative treatment might be suggested, perhaps also in detecting pulmonary aneurysms within cavities, in the recognition of pneumothorax, and as a guide in the inducement of pneumothorax in treatment.

(*c*) Some deep lesions can be detected by X-rays which are obscure to auscultation.

These conditions are quite recognizable by physical examination, but can be better defined and recorded by photography. It must be finally borne in mind that for the present at least, and for a long time to come, X-ray examinations—which are reliable—are the privilege of hospital patients, and the wealthy or those who are within touch of central institutions. They are luxuries to the people, but form an essential part of the scientific equipment of schools of medicine.

THE X-RAY EVIDENCES OF EARLY PULMONARY TUBERCULOSIS IN YOUNG CHILDREN.

By W. J. S. Bythell, M.D.

I propose to give you this evening a condensed account of a series of X-ray observations, including between 300 and 400 cases, which I have carried out during the last eighteen months with a view to determining the value of the rays in the early diagnosis of pulmonary tuberculosis in young children. The majority of these examinations have been made in the X-ray Department of the Manchester Children's Hospital in conjunction with my colleague, Dr. C. Paget Lapage.

The two points to which I have paid special attention are (1) the comparative reliability of the radiographic and clinical methods of early diagnosis, and (2) the seat of origin of pulmonary tuberculosis in young children. With these objects in view, Dr. Lapage has picked out for me children aged from 4 to 12 (mostly from 6 to 10), all of whom have come from the poorer quarters of Manchester and Salford. In something like 45 per cent. of these cases the physical signs were either absent altogether or indefinite, the suspicion or probability of pulmonary tuberculosis being suggested by the presence of cough, or of general symptoms, such as anæmia, debility, loss of weight, &c. In about 28 per cent. definite physical signs of early disease were noted prior to the X-ray examination, while the remaining 27 per cent. were selected as having in all probability as healthy chests as could be found among children of of the poorer classes of a big town like Manchester.

Dr. Lapage was present at the X-ray examinations of all his cases, but did not show me his clinical notes until the X-ray diagnosis had been arrived at. We were able in this way to compare the results obtained independently by the two methods, and to form some idea of their relative value. As regards the *methods of examination* employed, nearly all the examinations were made by the fluorescent screen, plates being used only when required to clear up a doubtful diagnosis. The most important information in these cases is certainly derived from the fluorescent screen, as one is able to study the movements of the ribs and diaphragm, and the lighting up of the lungs, and also to turn the patient into various positions in order to see clearly the roots of each lung in turn, the posterior mediastinum and so on.

Now there are four chief points to which attention has been directed before attempting to make a diagnosis:—

(1) *The Movements of the Ribs and Diaphragm.*—These movements should normally be free, not jerky in any way, and they should be equal, or nearly so, on the two sides. In the majority of cases of pulmonary tuberculosis, even in the early stages, there is definite limitation of these movements, especially of the diaphragm of one or both sides, a condition usually known as "Williams's sign," after the observer who first drew attention to its diagnostic value. I agree with the importance generally attached to it to this extent, that definite limitation or absence of the diaphragmatic movements, either of one or both sides, must be regarded with grave suspicion, but I am perfectly certain from my own experience that absence of such limitation by no means indicates immunity from pulmonary tuberculosis, or even that the disease is limited to the roots of the lungs.

(2) The *lungs* should be free of shadows, or mottling, due to consolidation. Their translucency to the rays should also increase equally throughout during deep inspiration—or, as we describe it, they should light up in a normal manner as they become distended with air.

(3) A very careful examination must be made of the *roots* of both lungs. The crux of the whole matter lies in the answer to the query, "What is the normal appearance of the roots?" and I must confess that I am not quite satisfied yet upon this point. There are undoubtedly always some shadows at the roots of the lungs in the most perfectly healthy children, produced by the connective tissue, vessels and bronchi. These structures give rise to tree-like or branching shadows, radiating outwards from the roots, which in normal lungs are extremely faint and sketchy. The probability is that every attack of bronchitis adds a little to the density of these markings, and I have found a very definite increase in chronic bronchitis; but, in the absence of mottling due to enlarged bronchial glands or pulmonary consolidation, such increase does not, in my opinion, justify a diagnosis of pulmonary tuberculosis. I am aware that Jordan regards any increase of these streaks as a proof of peri-

bronchial infiltration of the lungs. I shall have occasion, however, to refer to his views again at the conclusion of my paper. In the next place, the examination of the roots may reveal other shadows caused by the presence of either pulmonary consolidation or enlarged (that is to say, tubercular) glands at the hilus of one or both lungs. These shadows are in most cases readily seen when present, and are quite unlike the streaky markings which I have described as normally visible. When they are recognized beyond any possibility of doubt, a diagnosis of tubercular infection of the bronchial glands may be safely made, and the only other question to determine is whether the lesion is recent and active, or old and healed. An old tubercular gland which has undergone calcification is unmistakable owing to the density of its shadow and the sharply defined character of its outline; a recent infection of the glands, on the contrary, produces blurred or "woolly-looking" shadows, and the more woolly-looking they are the more recent and active is the lesion. The same distinction also applies to the interpretation of pulmonary shadows due to consolidation, when the infection has extended beyond the lymphatic glands into the lung tissue itself.

(4) Lastly, the child has to be turned into the semilateral position so as to separate the shadow of the vertebral column from that of the heart and aorta, in order that we may obtain a clear view of the posterior mediastinum. Normally this should appear as a translucent space, and any obstruction to the rays will indicate the presence of enlarged mediastinal glands. My experience, however, has led me to the conclusion that enlargement of these glands is much less frequently observed than of the bronchial.

Such is the nature of the evidence which an X-ray examination places at our disposal in our attempt to arrive at an early diagnosis of pulmonary tuberculosis, and we have now to discuss the value of this evidence and the difficulties in the way of interpreting what we see upon the screen or plate.

I have already alluded to the difficulty of defining the appearance of a normal lung root, and I might raise the further question whether there *is* such a thing as a perfectly normal thorax, except in the very youngest infants. Such a chest would contain nothing in the way of lung shadows except the faint branching streaks at the roots representing the connective tissue, vessels and bronchi. Most of the children I have examined have been between the ages of 6 and 10, and even in children so young as this one rarely finds a perfectly normal thorax. Nearly all the cases have shown either an increase of the streaks which I have attributed to chronic bronchial troubles, or they show in addition evidence of having at some time or other suffered from an intra-thoracic tubercular infection.

Dr. Lapage has kindly tabulated for me 150 of the cases which I have examined with him, and the results we have obtained work out as follows:

Clinical examination		X-ray examination		
		Positive	Suspicious	Negative
Positive	43	35	5	3
Suspicious	67	38	16	13
Negative	40	9	8	23

Thus, while out of the cases diagnosed as positive upon clinical grounds 94 per cent. were diagnosed as positive or suspicious upon X-ray examination, out of the forty cases picked out by Dr. Lapage as free from any suspicion of pulmonary tuberculosis no less than 22 per cent. were shown by the rays to be definitely tubercular, and 20 per cent. presented appearances which were decidedly suspicious. In these cases there was not only an entire absence of physical signs, but they were carefully picked so as to exclude any in which the history raised the least suggestion of chest trouble of any description; and yet, as you see, the X-ray examination showed that in children of this age and class only about 55 per cent. could be definitely stated to be free from pulmonary tuberculosis. I may add that the other cases I have examined, amounting to a little over 200, agree very closely with the series tabulated here.

I think we shall all agree that unless we were to live in glass cages, supplied with sterilized food and air, every one of us must be called upon to deal with tubercular infections at various times during life, while town-dwellers (and especially children of the poorer classes who live under insanitary conditions) probably receive big doses of tubercle bacilli at frequent intervals. Therefore we may take it that the vast majority of persons whom we must define as perfectly healthy contain certain scars received in the successful battles that they have fought against the invading bacilli, most commonly in the form of enlarged, or possibly calcareous, glands which will be seen upon the fluorescent screen as definite shadows at the roots of the lungs. The children I have examined have all, as I have said, come from the poorer quarters of Manchester and Salford, and my experience is that among this class of children the great majority after the age of 6 or 8 have one, two, or more shadows at the hilus of each lung. The minority, in whom there is no such evidence of enlarged bronchial glands, commonly present some increase of the normal streaky appearance at the roots, the result, I presume, of bronchial troubles. We may assume, then, if my reading of these cases is correct, that a very large proportion of children at this age (or at any rate among the poorer classes of our large towns) possess enlarged bronchial glands or caseous nodules at the roots which contain active tubercle bacilli. It does not, however, follow from this that they are necessarily the subjects of active tubercular disease, and still less that they are suffering from any form of pulmonary disease. The focus is in most cases a closed one, and the usual course of events is that in time the bacilli die out, leaving a more or less completely calcified gland or nodule which will remain visible upon the screen as a dark and well-defined shadow at the root of the lung. Nevertheless, so long as the glands contain living bacilli they are a source of danger to the child, and may at any time lead to infection of the adjacent lung tissue.

To sum up, the three chief points of practical interest that, as a radiologist, I wish to bring before you and to emphasize this evening are these :—

(1) That in a very large proportion of children, and among the poorer town-dwellers in probably the actual majority, the bronchial glands are enlarged and contain active tubercle bacilli which may under certain circumstances give rise to pulmonary phthisis.

(2) That when the glands are in this condition, or when the infection is commencing to extend into the lungs, the lesions should be readily diagnosed by means of the fluorescent screen, whereas the most careful clinical examination will probably lead to indefinite, if not actually negative results. That is to say, there may be an entire absence of physical signs, and the general health may be such as to merely raise a slight suspicion of some tubercular infection, without any localizing symptoms.

(3) My experience has been that, in children at all events, pulmonary tuberculosis *invariably* starts at the roots of the lungs, as in the slide you see upon the screen.

Out of the 300 or 400 children which I have examined systematically for this purpose I have never seen a single case in which the disease had not obviously originated at the hilus of the lung, and I have yet to meet one in which the sole or even the primary lesion has been apical. I do not mean that when the apex of one of the lobes has been involved there is necessarily a visible track of diseased tissue all the way from the root to the apex, but that when there is a lesion at the apex there is in every case a far more advanced lesion at the root of the lung which has escaped detection by clinical methods of examination simply because it has probably given rise to no physical signs by which its presence could be determined. Dr. Jordan, I believe, goes further than this, and says that most, if not all, cases of pulmonary tuberculosis commence at the roots of the lungs, that is to say, in adults as well as children. According to his view, the disease always spreads from the hilus in the form of a peribronchial infiltration until it reaches a region (such as the apex of one of the lobes) where the conditions are more favourable to rapid growth; he therefore argues that what I have described as an increase of the normal streaky appearance visible at the roots of all lungs is a definite indication of tubercular infiltration, and he would condemn all cases in which this is recognized as the subject of tubercular infection. Personally, as I have said, I am convinced that the streaky shadows may be increased, in some cases at all events, by simple bronchitis or bronchiectasis, and I regard definite mottling and nodules at the roots of the lungs as the only reliable evidence of tuberculosis. When therefore I first read his papers I felt inclined to disagree strongly with him upon this point, but latterly it has occurred to me that possibly the disagreement is really due to a misunderstanding. I can best explain what I mean by the help of this somewhat crude diagram, and I shall be very pleased if Dr. Jordan will give us his views upon the subject during the evening. If, when he speaks of peribronchial infiltration, he means mottling following the course of the bronchi—i.e., a series of mottled shadows giving a general impression of streakiness radiating from the root of the lung, such as I have attempted to represent in this sketch—then I am entirely in accord with him in regarding that appearance as diagnostic of tuberculosis. But, apart from this type of shadow, one often sees a definite accentuation of the normal streaky shadows which I am convinced is the result of non-tubercular bronchial troubles, such as I have pictured in this diagram. That, if I am right, is a very important point to be kept in mind when we attempt to interpret these root shadows. In any case, there is much to be said in favour of the view that adult, as well as juvenile, phthisis commences at the hilus of the lungs, for I cannot recall having seen a single case of a purely apical phthisis even in an adult. Most of the cases I have seen have been of this type, in which there is no doubt as to the root origin of the disease.

If time had permitted I should like to have dealt with the problem already raised by Sir Douglas Powell of how the disease spreads from the hilus throughout the lungs, at first sight against the direction of the lymph-stream. To put my views as concisely as I can, I believe (from my post-mortem experience) that the lung may become infected either by the direct spread of the tubercular foci from the breaking down of the caseous glands, or by the bacilli being carried backwards along the lymphatics by what is known as the *retro-impulsion* of the lymph, owing to the block that has taken place at the bronchial glands. Later, as the disease becomes more established, the apices may become infected either by this direct extension along the lymph-channels, or as independent foci by one of the other methods, such as autogenous blood infection, aspiration broncho-pneumonia, or by bronchial embolism of infective particles detached from an open lesion. In either event the practical point for us to remember is that the infection is derived from the hilus, and that it is in this region that we must look for the earliest evidence of tubercular infection.

X-RAYS IN THE EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS.

By Stanley Melville, M.D.

Up to quite recent times I think the attitude of the clinician towards radiography may be fairly summed up in the following sentence taken from a well-known text-book : "In the early diagnosis of phthisis we have found the method of but little use." These are plain words and from men entitled to respect, not only because of their attainments and profundity of knowledge, but because they are open-minded men and fair in their judgment. I do not wish to dwell unduly upon this criticism, but I make bold to say that we may look forward with some confidence to a revision of this sentence in the next edition.

Radiography of the chest, I have no hesitation in saying, must ever be the handmaid, and not the ruler. To divorce the radiographic and clinical examinations, or to make them independent of one another, will be a calamity. But I maintain with all the emphasis I can, that the clinician of the future should not feel satisfied until his findings have been either fortified, or modified, by X-ray examination. I suppose, as radiographers, one of our greatest dangers is that of reading more into a skiagram than is actually present—perhaps I ought to say in regarding radiography as diagnostic rather than confirmatory and critical. For example, as to whether a tubercular focus in the lung is active or inactive; the somewhat "woolly" appearance of the one, and the "harder" appearance of the other, are not in themselves sufficient and must be taken in conjunction with other evidence, both clinical and rational.

It is well, I think, to inquire as to what progress we have lately made in our knowledge of the X-ray appearances in the early stages of pulmonary tuberculosis. Chiefly, I imagine, in the great improvement in apparatus and technique (about which I should like to say a word presently), and resulting therefrom, our interpretation has, unquestionably, become more definite and exact. And yet, when looking up the literature on this subject, what a debt we owe to those earlier workers for the excellent foundation they laid. Who does not read with admiration Walsham and Orton's work on radiography of the chest? It is a small volume—but brimful of valuable work—with a bibliography long enough to cause mental vertigo. To this foundation we have lately been adding experience, and the first conundrum I venture to ask is this—viz., What are we to regard as a typically normal chest? Is it such a chest as the one I show you on the screen, one with the appearance of which you are all familiar? But, gentlemen, I examined a very great number of chests before I found this specimen, and I had begun to think it was a little joke played by Walsham and Orton on our guileless selves. Or, is the chest of everyday life, such as we meet with among town-dwellers at any rate, to be taken as a standard? The appearances of such a chest are very different, and opaque masses at the hilum with definite radiating striæ thence towards apex and base, are so common as to compel one either to regard them as within the limits of health or to consider them as arrested pulmonary tuberculosis, and accept the view that an overwhelming percentage of the population are, or have been, tubercular. I hope to get the answer to my conundrum to-night.

But, gentlemen, as the clinician recognizes a general type of chest which is prima facie suggestive of tuberculosis, so I think it possible for us to do the same. A chest, in which expansion is generally restricted; in which the cardiac shadow appears to be of small size and vertically placed; in which there may be restricted movement of the diaphragm; in which there may be, generally or locally, a suggestion of diminished translucency on deep inspiration, and in which are frequently seen opacities of varying density at the hilum on *both* sides—this is the picture I draw for your consideration. Let us examine such a type of chest a little more in detail—

First : Deficient movement of the chest wall and diaphragm. If any further evidence were required to convince me of the necessity for taking X-ray evidence in conjunction with the clinical history and physical signs—it would be at this part of the examination. Practically any and every inflammatory process, whether of lung or pleura, will limit the excursion of the ribs and diaphragm on the affected side. And what a pitfall, for example, is an apical pneumonia, for here we have not only restricted movement, but actual shadows (perhaps finely mottled) in the lung tissue itself. But, undoubtedly, unilateral limitation of the diaphragm is a sign of very great value. I need hardly mention that careful comparison should be made between the movements of the diaphragm on the two sides. A bilateral limitation is frequently observed in nervous patients ; in mouth-breathers, and in otherwise quite healthy persons, especially women, who normally make very little use of the diaphragm. Cog-wheel, jerky (or to use the most useful word, suggested I think by Dr. Bythell and Dr. Barclay—viz., "stammering") movements are evidences of definite value.

Secondly : The Heart. While not wishing to dogmatize, I am convinced that the frequently noted, small and vertically disposed heart is a factor of the greatest importance. For my part, I have not found this condition restricted to persons with long and narrow chests, nor do I think there is justification for regarding it as due to rotation. The apex of the heart is usually well within the mid-clavicular line, and the right border is not abnormally situated. I regard it as a true dystrophy, and I venture to maintain that this small and usually feebly acting heart (whatever its size in later stages of the disease may become) is a definite factor in regard to prognosis.

Thirdly : The Mediastinum. The lymphatics of the lung (which, in the more superficial parts, communicate freely with those of the visceral pleura), together with the deeper lymphatics of the lung and bronchi, finally drain into the glands of the mediastinum, the bronchial and posterior mediastinal glands respectively. Further, there is free communication between the lymphatics of the pleura and peritoneum. The mediastinal glands may thus become involved through the mesentery—possibly a point of importance in considering the incidence of tuberculosis of the lung, especially in children. How much more common it is, in children, to find enlarged glands at the hilum, and the first evidence of pulmonary tuberculosis in the more central part of the lung rather than at the apices ; indeed, how often does one ever see or hear of an apical tuberculosis in a child ? Are these enlarged glands

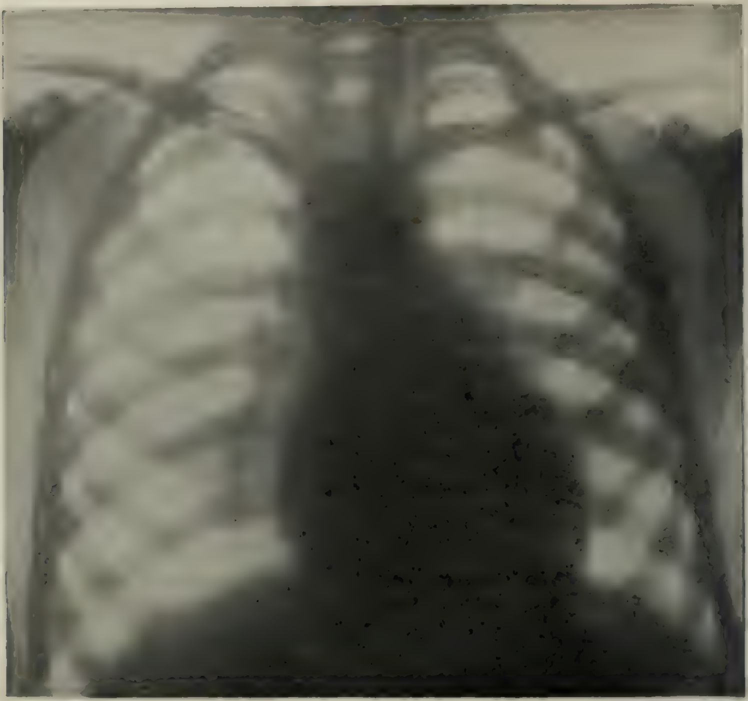

Fig. 1.

Skiagram of normal chest—ordinary method.

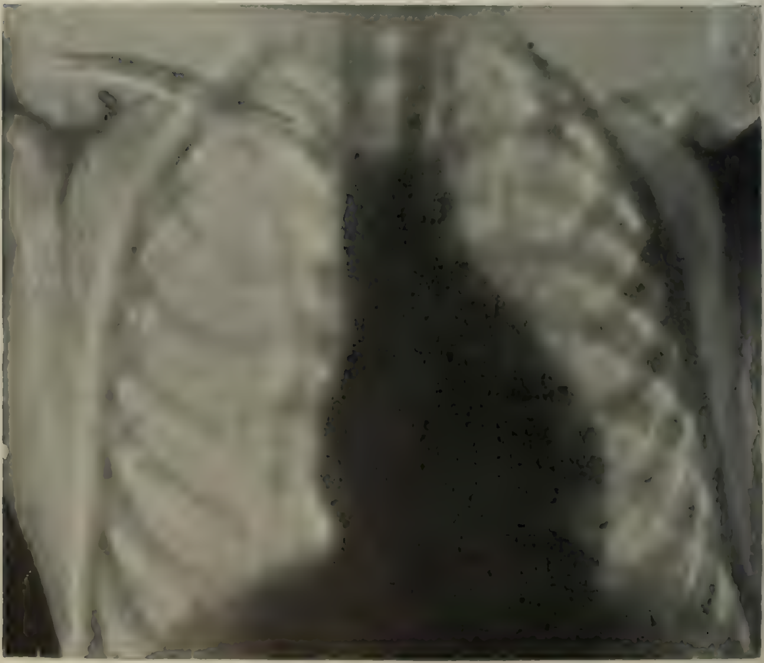

Fig. 2.

Skiagram of normal chest—plastic method.

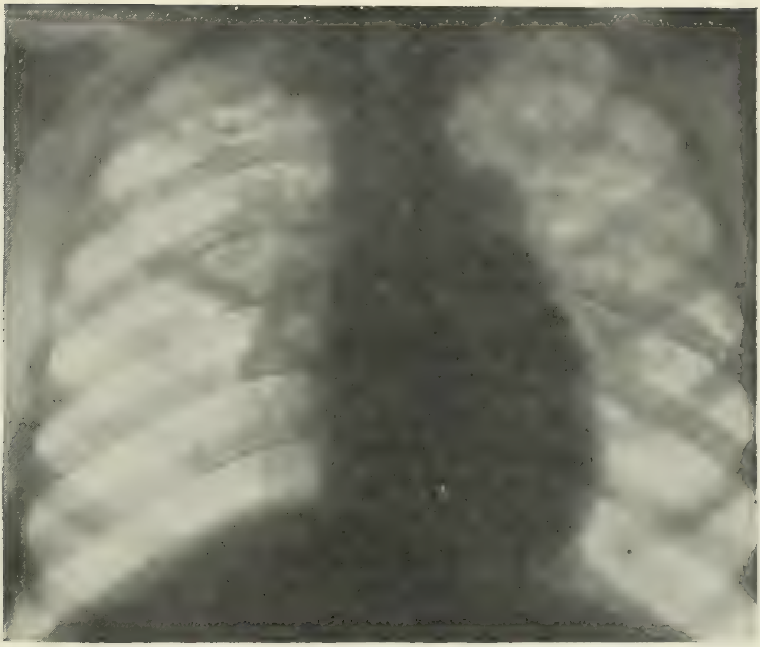

FIG. 3.

Skiagram showing characteristic mottling of centrally situated infiltration, from a case in which there were indefinite physical signs—ordinary method.

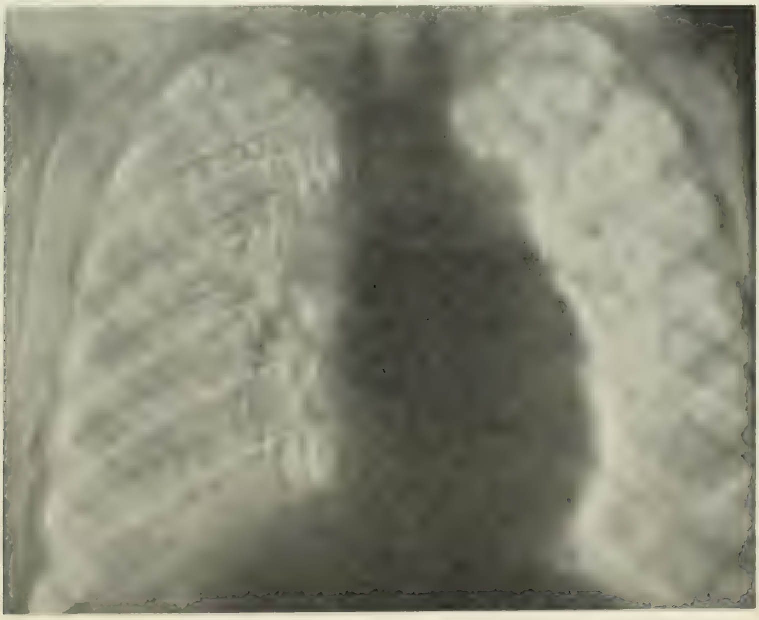

FIG. 4.

The same—plastic method.

at the root of the lung of mesenteric origin, and, if so, are they due to infection by bovine or human tubercle bacillus? If so, what is the definite relationship between them? Does the tubercle bacillus lie dormant in the glandular structures until aroused to activity by some exanthem or severe cold? Will further research show that these enlarged and possibly caseating glands at the roots of the lungs are the result of a primary pulmonary infection, and that in late life a secondary infection is superadded. These are questions of vital importance, and I venture to suggest require further investigation.

The question of the significance of the radiating linear shadows seen in a large proportion of chests examined has received much attention, and is still, in its relation to pulmonary tuberculosis, *sub judice*. These radiating lines can be seen whenever the expansion of the lung is restricted either by disease or on holding the breath, and, by contrast, show up very clearly in emphysematous conditions of the lung. They are, I think, undoubtedly shadows of the bronchial tubes, though they are still held by some to be shadows of pulmonary blood-vessels. I would suggest that they are coarser shadows than would be cast by blood-vessels, and I show you a beautifully injected sheep's lung which Dr. Cecil Clarke very kindly prepared for me. In this the one lung is injected through the bronchus, the other through the pulmonary artery, and the contrast in density of the shadows is well shown. Taken by themselves I must admit to feeling somewhat chary about regarding these radiating linear opacities as pathognomonic of tuberculosis—and only in conjunction with definite surrounding mottling should they, so far as my own experience teaches me, be so regarded.

Without being unduly critical, I must admit I do not like the term "peribronchial" phthisis. Am I wrong in suggesting that the term is not an absolutely sound one? Why not call it a "central" tuberculosis, in contradistinction to "apical" or "basal," any fibrosis around the bronchi being only the natural result of local irritation. The tubercle bacillus, either by aspiration, or more rarely by the blood-stream, finds its way to a fine bronchus or alveolus, and there develops, producing, by proliferation from the cells of the bronchi, alveoli and capillaries, a small mass of epithelioid cells. Extension then takes place by aspiration or by means of lymphatics. The fibrous stroma of the lung as the result of inflammatory irritation partakes of the nature of "growth under irritation" (I quote from Powell and Hartley), producing fibrosis of the lung. There is nothing unreasonable in the contention of radiographers that the tubercle bacillus will find a suitable locality in the more central portions of the lung—thence extending to the apex either by aspiration or by the lymphatics, or possibly by the blood-stream, and the slides which I show you illustrate this primary central invasion, at any rate, to my own satisfaction.

In the foregoing remarks I have in mind the recent work of Dr. Jordan—a work which I gladly join with other chest radiographers in welcoming. But, if Dr. Jordan will forgive my criticism, I am in some difficulty, if I have read him aright, in following his argument in its entirety. He appears to make several distinct propositions. Firstly, that opaque masses at the hilum, plus radiating linear opacities, are pathognomonic of a hilum invasion; secondly, that this condition, plus mottling, indicates activity. I am only in cordial agreement with him on this second proposition. Then I have another bone to pick with him—and this is when he becomes Napoleonic, and sweeps away difficulties as though they had no existence. I refer in particular to his dismissal of bronchiectasis. He writes: "It seems certain that bronchiectasis is merely a special form of fibroid phthisis in which the air-tubes have become dilated and putrefactive organisms have gained entrance." If Dr. Jordan is correct he will deserve our warmest thanks, and pathology will have to be re-written.

But, gentlemen, however suspicious we may be of the invasion of the lung by our examination, the one and absolute evidence is the presence in part or parts of the lung of the characteristic fine mottling. There are definite pitfalls for the unwary in the interpretation of this, but time will not admit of discussing them now.

It may be due to my own want of observation, but I have never been able to satisfy myself with making an examination with the fluorescent screen alone. I think it imperative to make this examination most thoroughly, and a vertical and horizontal diaphragm ought to be arranged on the screening apparatus to enable examination of a small area of lung, and the screen examination should, if possible, be made with the patient in the erect position. I, invariably, fortify my screen examination with a skiagram, and fine mottling of the lung, which can readily be overlooked with the screen, will be clearly demonstrated on the plate

With the modern apparatus at our disposal and the possibility of passing heavy currents through our tube, instantaneous, or, at any rate, nearly instantaneous, skiagrams should be the rule. For some time I have been in the habit of making plastic skiagrams, some of which I have brought for your opinion and criticism as to their possible value. They would appear to show the characteristic mottling well.

I have, personally, too slight an experience of stereoscopic radiography of the chest to speak with any assurance as to its value in pulmonary tuberculosis. It is certainly most beautiful and instructive, but whether of material use (having regard further to the expense and trouble involved) in the diagnosis of early pulmonary tuberculosis, I will leave others with greater experience to speak. My chief objection is that it cannot well be of daily routine use; somewhat complicated apparatus is required to do it efficiently, and the slightest movement of the patient during the exposure of the two plates is detrimental. These, however, are only working objections.

Gentlemen, I have to thank you for your kind patience with me. I should like, in conclusion, to make a confession of my own personal belief. I think—

(*a*) That we are behind other countries in the routine use of radiography as an aid to the early diagnosis of pulmonary tuberculosis.

(*b*) That radiography affords the earliest evidence of pulmonary tuberculosis, and, incidentally, demonstrates a more widespread extent of infiltration than is suspected by clinical examination alone.

(*c*) That a very high percentage of cases of pulmonary tuberculosis begin by invasion of the more central parts of the lung than at the apex—this especially noticeable in children.

(*d*) That clinical examination alone, even in the hands of the most experienced clinician, fails: (1) In the presence of emphysema, (2) in deep-seated invasions; and that these very factors, which hinder diagnosis by percussion and auscultation, are, for obvious reasons, the very factors which are an aid in X-ray examinations.

Dr. David Lees, in answer to the President's invitation, said he had had no intention of joining in the discussion; he came simply to learn, for he felt sure he would hear and see a great deal from which he would derive instruction; and he would have been glad to postpone any comments until he had heard the X-ray experts. He had felt much interest in the subject during the last few years; and Dr. Simmons would remember that seven years ago they made some observations together in the X-ray room at St. Mary's Hospital, to determine how far physical examination on the one hand and X-rays on the other would agree, and how far they would differ, and whether one was better than the other. At that time their conclusion was that the X-rays would not definitely show areas of apical dullness, which to his percussion were quite evident. Thus he felt at that time that the ordinary physical examination was more to be trusted than the X-ray examination. But even then he had a feeling that this conclusion would before long be altered, owing to the improvements in technique which were sure to come. And so it had proved. During the last few months, owing to the kindness of Dr. Orton, he had had the opportunity of testing the matter again, for Dr. Orton allowed him to send to his house a certain number of patients in whom he (Dr. Lees) detected what he considered definite evidence of incipient pulmonary tuberculosis, chiefly percussion evidence. He believed that the value of accurate percussion in the detection of incipient tuberculosis was much underestimated. In books and articles, stress was almost invariably laid on auscultatory signs, and but very little upon changes in percussion. Indeed, the statement was sometimes made that auscultatory signs might be present when there were no percussion signs at all. He believed that to be an absolute mistake, and until this was recognized it would not be appreciated how very important in the diagnosis of incipient tuberculosis was the practice of methodical percussion. He would even say that it would not be recognized until physicians gave up the habit of examining patients in the erect posture only. For a proper examination of the front of the chest, it was absolutely necessary for the patient to lie on a couch with relaxed muscles.

Having examined cases of what he regarded as early pulmonary tuberculosis, he sent them to Dr. Orton's house, and Dr. Orton kindly investigated them by means of the X-rays. The joint conclusion was, that in every case in which percussion gave reason to believe in the existence of incipient pulmonary tuberculosis, the rays showed definite and distinct signs. So the improvements in technique had brought about a great advance in the applicability of X-rays to the diagnosis of pulmonary tubercle. But a fact which could not be too carefully noted was that X-rays would show certain deep-lying structures (to which in this discussion attention had already been directed), old damaged glands, and thickened bronchial tubes, which, in many cases, were no doubt the result of former attacks of tubercle, and in some other cases may have been simply due to attacks of bronchitis and similar conditions. As Sir Douglas Powell had pointed out, the bronchial glands were the dust-bins of the bronchial tubes. The detection of such altered glands and bronchi did not prove that the patient was now suffering from active tubercle, and so it was easy to draw erroneous conclusions. He differed absolutely from the statement which had been made during the discussion, that in the majority of cases, in children at all events, the disease *started* in the bronchial glands. How did the disease-material get to those glands? It was known that a large number of children, examined post mortem, were found to have caseous bronchial glands, but no other sign of tubercle in their lungs. He believed the bacilli got there through the bronchial mucous membrane, sometimes without leaving any trace of their passage. Apparently the same thing might happen in the abdomen. Some patients were found post mortem to have very slightly affected mesenteric glands, yet the whole peritoneum was swarming with tubercles; the bacilli seemed to pass through the intestinal wall as easily as through the wall of the bronchial tubes. Even if a screen examination showed only disease of bronchial glands, that did not prove that the disease commenced there, but only that it was there the bacilli were arrested. In cases of caseous glands in children, the infection was often of quite a virulent nature, and the tubercles spread locally by direct contiguity into the lungs, and sometimes by rupture of a softened tuberculous gland into a bronchus. But both in adults and in children the *original sites of infection* in the lungs could be detected by careful percussion, though the auscultatory signs might be extremely slight. The early infiltrations detected by percussion are difficult to see by a mere screen examination, but they may be quite evident in the skiagram, which shows areas of fine mottling, chiefly in the apical regions, but often also elsewhere. The X-ray examination by Dr. Orton of the cases sent to him (1) confirmed in every case the existence of morbid changes in the lungs; (2) confirmed the statement, based on percussion, as to which lung was the more affected; (3) confirmed the localization in the upper intercostal spaces; (4) revealed opaque root-areas and thickened bronchial tubes. One case was of special interest, for by percussion it was determined that the left apex was more affected than the right, but with the fluorescent screen the right seemed the more involved. But when the skiagram was taken, it was found to agree with the percussion signs.

He thought X rays were likely to be of very great service in the detection of early pulmonary tuberculosis, and yet they were not indispensable, for the same facts could be determined by careful percussion. But if a practitioner was not accustomed to rely upon his percussion, he would do wisely to obtain an X-ray picture of the case. But it must be taken by an expert with the best possible technique, and must be carefully interpreted, for, as Sir Douglas Powell had pointed out, these pictures were liable to great misinterpretation.

He thought physicians were much indebted to X-rays for demonstrating the fact of inhibition of the diaphragm. That was not invariable, but it was very frequent and very important, and it clearly pointed to an inhibition through the nervous system, similar to that which existed in peritonitis (even localized). He thought Dr. Bythell said that he had noticed the same thing in regard to the ribs. This inhibition explained a phenomenon which every physician must have noticed in cases of phthisis—namely, the very great difficulty of getting the patient to take a deep breath; one might persuade and even command him to take a deep breath, but scarcely any air entered, and it seemed certain that the muscles of inspiration generally were inhibited, as the diaphragm could be seen to be in an X-ray examination.

Dr. A. C. JORDAN said Sir Richard Douglas Powell had been good enough to warn him that he would refer to his (Dr. Jordan's) work, and he wished to thank him for his kindly criticism. Sir Richard, and some others, doubted his statement as to the frequency with which peribronchial pulmonary tuberculosis occurred. He had with him a series of X-ray photographs showing the distribution of the pulmonary disease in thirteen consecutive cases of phthisis examined in one week at the Royal Chest Hospital. (Dr. Lees was kind enough to show this series recently after his lecture at the Royal College of Physicians.) The thirteen cases included examples of phthisis in all stages, and every one showed considerable peribronchial mottling. In some the mottling was exclusively of the peribronchial type; in others the apices, too, were involved. Nor was this an exceptional week. The peribronchial mottling would be found to be the most prominent feature of the phthisis cases at any visit to the hospital.

With regard to the "normal hilus shadows," Sir Richard Douglas Powell had insisted, very properly, that the real proof that these shadows were the result of tubercle would be the demonstration of tubercle bacilli in them. But one would not expect to find tubercle in healed lesions; nobody had ventured to doubt that the calcareous deposits found with great frequency in the bronchial glands of healthy persons were due to healed tubercle. Of course, these glands had other bacteria to deal with than tubercle bacilli; still tubercle was the chief cause of chronic affections involving these glands.

Sir Richard Douglas Powell and others had quoted him as believing that pulmonary tuberculosis began in the glands at the roots of the lungs. This is a belief which he never held. It would be as rational to argue that a patient with a tuberculous finger and tuberculous glands in the axilla had derived the disease in his finger from infection from the axillary glands. Of course the glandular involvement at the roots was a secondary change. The first stage was catarrh of the air-tubes; this gave no X-ray evidence of its existence. The next stage was catarrhal broncho-pneumonia; in this stage the lumen of the air-tubes was found to contain catarrhal cells (epithelioid plates), while the tissue surrounding the bronchial cartilages was found infiltrated with leucocytes. This peribronchial small cell infiltration gave rise to the X-ray mottling along the air-tubes which was the characteristic feature of peribronchial phthisis. The glandular enlargement at the roots then followed naturally, and was usually an early result.

In all other respects he was glad to find Dr. Bythell in agreement with himself, the only point of disagreement being in reality one of misunderstanding, for Dr. Bythell had quoted him as stating that the linear shadows always seen radiating from the roots were evidence of active disease, whereas he (the speaker) stated explicitly in his papers that the linear shadows might radiate even to periphery of the chest in healthy persons, but that mottling was the evidence of active disease.

Sir Richard Douglas Powell had objected to the statement, "The X-rays findings are facts"; he said, "No, they are shadows." True, but if the shadows can be proved to represent facts, they can be regarded as facts. In his second paper on the subject he had published an X-ray photograph in which he had marked five particular blotches of tuberculous broncho-pneumonia with the five letters, *a*, *b*, *c*, *d*, *e*, and below was a microscopical section through the entire lung of the same patient after death, with the same five blotches marked by the same letters, so that the identity of the five shadows with the five spots of tuberculous broncho-pneumonia was proved, at any rate, in this instance.

[SIR RICHARD DOUGLAS POWELL interposed to say he did not question the facts, but their interpretation.]

Dr. Jordan continued that the only way to settle the question was by making careful pathological and histological examinations of cases whenever the opportunity arose. In the case mentioned he had done so. Unfortunately, it was very rare for a chance to occur of making a pathological examination of a case of peribronchial phthisis while it was in a typical stage. As a rule, the disease had advanced far beyond that stage when the patient died, the greater part of both lungs being destroyed. In the case in which he had been able to compare the X-ray pictures with the pathological changes the girl had died of tuberculous ulceration of the intestines before the disease of the lungs had advanced beyond recognition.

The only other chance of obtaining a full examination of a typical case must offer itself by a sheer coincidence, for instance, if a patient were to be killed by an accident after the X-ray examination.

(Slides were shown illustrating the various points mentioned.)

Dr. PASTEUR said it had not been his intention to participate in the discussion, but views had been advanced which, to him, were somewhat bewildering. Physicians were being asked to reverse opinions they had long held as to the direction of spread of tubercular lesions in the lungs, and to believe that tuberculosis always began at the root of the lungs and spread outwards. One speaker mentioned the manner in which he considered the spread took place. The extension of tubercular lesions in the lungs, both on clinical and pathological grounds, was believed to follow a more or less definite course from the apex downwards, and he failed to see how that order of events could present itself so often if the commencement of the disease was usually at the root. He agreed that tuberculosis in children was often located near the root, and he recalled vividly a case of suddenly fatal hæmorrhage from hæmoptysis in a child aged 4; it was from a large branch of the pulmonary artery, which was in communication with a cavity that had formed at the root of the lung, by direct extension from a tuberculous gland. But such cases did not establish the general proposition which had been advanced. One must admit that much of this evidence was based on the interpretation of shadows which had been observed in the chests of living individuals, many of whom were in good health. Before opinions which were firmly established could be reversed, it was necessary that the views which were to supplant them should be supported by the very strongest evidence; and he agreed with Dr. Jordan that what was chiefly required was a closer correlation of radiographic evidence with post-mortem findings. Until that evidence was forthcoming he hesitated to change his faith.

Dr. BYTHELL, in reply, said that he had brought the subject forward not because he felt he had something very new to say, but because he considered it to be a subject which needed ventilating. He agreed that in some cases the first change was one of broncho-pneumonia, but in most cases the disease of lung was due to the breaking down of tubercular glands. As Osler and Murray Leslie said, in some cases one cut a section of these lungs, and it was very difficult to tell where the gland ended and the broncho-pneumonia began. He had looked up sections of his own, and found himself in agreement with that idea. It was well known that a large number of children had tubercle bacilli in their glands, though apparently the children were in the best of health. There was a danger-spot there, and some day they might caseate, as the result of measles, or whooping-cough, and the bacilli get the upper hand. Many cases originated at the root through this. Dr. Lapage and he now proposed to proceed in the following way: To screen a dead body and make a diagnosis, then make the post-mortem examination, and see if the two diagnoses agreed. He would like to answer Dr. Lees's remark that he did not believe in the origin of the disease in the bronchial glands. He agreed that these glands were usually inactive, but Dr. Lees said enlarged bronchial glands, as seen by the screen, did not indicate active tubercular disease. Of course, in most cases the glands were able to deal with the bacilli; probably nine-tenths of those present at the meeting had done so, and showed scars resulting from the conflict. In others the glands broke down and infected the lung; it did not matter where the bacilli came from, because the practical point was that the first morbid lesion was the enlarged bronchial glands, which in turn infected the root of the lung. He took mottling due to pulmonary consolidation as the sole reliable evidence of active pulmonary tuberculosis, and he was sure, if he could get Dr. Lees to attend the out-patients' department at the Manchester Children's Hospital for a month, he could convince him of two things, as he had other medical men at the hospital whose feeling at first was the contrary: namely, first, that in children, at any rate, the disease invariably, in over 300 consecutive cases, started from the roots; secondly, that the rays showed definite pulmonary disease at the roots which could not be diagnosed by percussion or auscultation, or other method of examination. The root was the dangerous part.

Dr. LEES explained that his point was that although caseous glands were extremely frequent in small children, yet that the disease had not commenced there, but had got to the glands from somewhere, and in the overwhelming majority of cases he did not doubt it entered through the bronchial mucous membrane, sometimes without leaving traces of the passage.

(The discussion was adjourned until March 19).

Electro-Therapeutical Section.

March 19, 1913.

Dr. Reginald Morton, President of the Section, in the Chair.

A Discussion on the Use of X-rays in the Diagnosis of Pulmonary Tuberculosis.[1]

Sir Richard Douglas Powell, in re-opening the discussion, said that in his opening remarks he endeavoured to define in what respects skilled X-ray examinations were most serviceable in the diagnosis of tuberculosis and laid some stress upon their value in recognizing the degree and extent of disease at different periods, thus serving to record advance or retrogression under treatment, and especially under institutional treatment. In the diagnosis of the disease as regards degree, activity and outlook he would appeal to X-ray examination as of value in certain cases provided its findings were carefully collated with those of other methods and taken in association with the general features of the case. In cases of differential diagnosis, too, some of which he had specified, the rays were valuable, and also as a help in defining the outlines of pneumothorax or empyematous or other abscesses or cavities with a view to surgical interference.

He was unable to follow all the conclusions arrived at by Röntgen-ray observers with regard to the morbid anatomy and distribution and spread of lesions of tuberculosis, but on this point he spoke at some disadvantage, as he was not aware how far the conclusions of Dr. Jordan and others were generally accepted by their colleagues in the X-ray department of clinical medicine. It seemed to him a rash conclusion to accept the central opacities found in the great majority of cases examined as evidence of tuberculous disease, commencing at the hilus and the shadow streaks extending therefrom along the bronchial tracts as paths of advance of the tuberculous lesions towards the periphery.

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[1] Adjourned from February 21.

One would expect all urban dwellers at least to have the shadows of their peribronchial glands deepened, and in this cigarette-smoking age the capacity of the glands as dust-sifters must be still more exercised. But the assumption of the X-ray specialists that these shadows were of tuberculous origin and that, as such, they preceded in most cases any peripheral lung lesion that could be recognized by other physical examination, was to his mind quite beyond what the facts warranted. Such an assumption received little support from pathological investigation, and excluding, perhaps, exceptional cases of mediastinal suppuration with inhalation of tuberculous pus into the bronchial tract, local infection extended along the lymphatics in the direction of, not against, the lymph currents. It also seemed to him that in their clinical aspects the cases brought forward by Dr. Jordan as typically illustrative of his contention as to the central commencement of lesions and their extension towards the periphery were by no means conclusive. In the case of the girl, aged 12, fig. 5 in the pamphlet, "Peribronchial Phthisis," the shadow over a large peripheral area P and the shadow over a corresponding peripheral area on the other side were nearly as deep as the central opacities and indicated the presence of physical signs of an obvious kind over portions of lung very commonly affected in an early stage of the disease. There were similar shadows in fig. 3, the case of a man, aged 35, significant of lesions which could scarcely have been overlooked on careful physical examination. It seemed to him impossible to regard these as lesions of extension from the central areas.

If X-ray examination revealed disease in all cases, where was the advantage of it? Like other clinicians, he had been accustomed these many years to give opinions for or against tuberculosis in cases which—occasional errors excepted—the views expressed had stood the test of time. But, to put it in another way, supposing an X-ray department attached to a life assurance office. It would be more easy for a camel to pass through the eye of a needle than for an insurance candidate to enter through that department to acceptance for first-class life assurance. And yet when statistics were examined it was found, for instance in Manchester, whence Dr. Bythell's statistics were derived, that the death-rate from all forms of tuberculosis was 9·8 of all causes and 0·16 on the population; from pulmonary tuberculosis, 6·6 of all causes and 0·11 on population; and in his own life assurance office, of 2,234 deaths in ten years 97 were from tuberculosis, or less than 5 per cent. of mortality. And of 150 cases in which policies had been issued in the last three years with a special clause that any person in whom tuberculosis had arisen could at once claim two-thirds of the amount insured, in no instance had any claim come in.

Sir Richard repeated that he was not questioning the value of X-ray examinations in recording physical phenomena and in divulging some lesions that might be too deep for detection by percussion and auscultation, but to be of clinical value, beyond merely recording physical signs, such examinations must be made with the greatest caution and interpreted with the most rigid care.

He showed, by means of the epidiascope, skiagrams of two cases taken by Mr. Lyster, which presented clinical features of some interest. The first case was that of a man who came from South Africa, where he had been working in the gold mines; he had had what he called a slight attack of bronchitis, which, he said, was unattended with fever. From his description Sir Richard thought it might have been an attack of influenza. He stated that he was examined by five physicians in the neighbourhood of the mines, who all declared that he had tuberculosis, and that he must leave the mines at once. He was allowed considerable compensation on his discharge. The prognosis expressed was very grave and the patient came to England, and consulted him (the speaker). The physical signs were those of some emphysema and some degree of fibrosis, more marked at the left upper lobe than elsewhere. He had some tenacious mucous sputum, dark-coloured, but not amounting to "black spit." This had been examined once or twice in South Africa, but no tubercle bacilli were found in it. A further very careful examination was made in London, but still no tubercle bacilli were discovered. Mr. Lyster made an X-ray examination of him, which showed a certain amount of general fibrosis and pigmentation of the lung (figs. 1 and 2). Sir Richard regarded the shadows as indicating a certain degree of peribronchial and interlobular fibrosis with some deposition of silicious dust in the lymphatic tissues, and he interpreted the deeper central shadow as due to the pigmentation seen through the greatest depth of the lung, and to some enlargement and pigmentation of the bronchial glands. The other half of the skiagram showed the same thing. The symptom which attracted attention was a peculiar air-hunger; the man would from time to time give a deep sighing respiration, and complained that he could not "get to the bottom" of his breathing. He also suffered from occasional attacks of heart failure. He had had several such attacks on board ship, some in South Africa, and one on his way down. Finding nothing wrong with his heart, Sir Richard attributed these to a neurosis for which, perhaps, the sudden shock from the announcement of the seriousness of his condition was responsible. He regarded the case as one of pneumo-koniosis in an early stage and had given a hopeful prognosis if the patient discontinued mining and lived a fresh-air life.

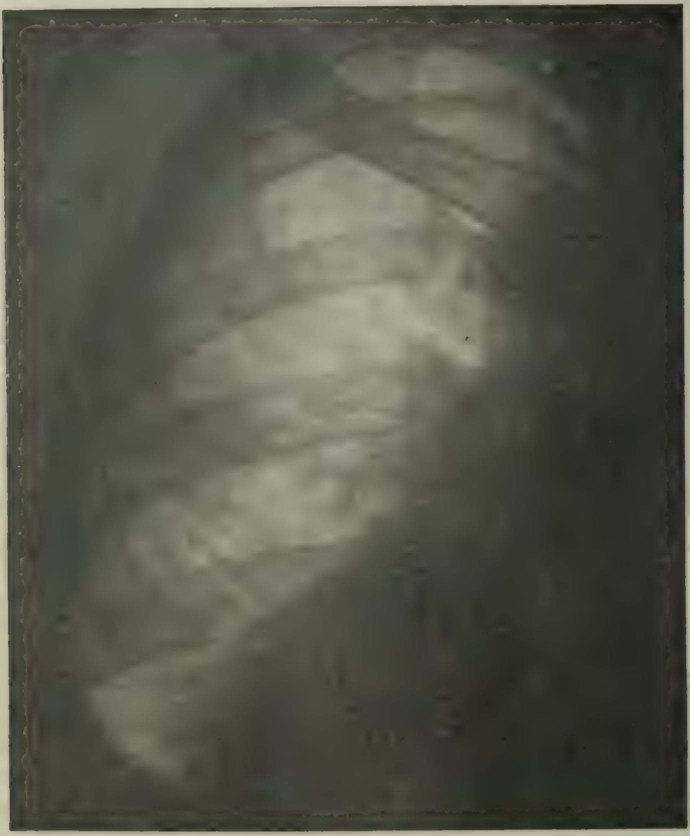

FIG. 1.

Case of pneumo-koniosis. Skiagram of left side, showing a general peri-infundibular and peri-lobular mottling of the lung with deeper central shadows partly interpreted as the general mottling seen through the thickness of the lung, with fibrous condensation and siliceous dust deposition in the bronchial glands.

The next case was to him a conundrum, and if any member could throw light upon it he would be pleased. The patient was an estate agent, aged 35, who had had a cough every winter for some years. In May last he had influenza, and the cough had persisted ever since.

and was now very troublesome, and often attended with a choking, flatulent regurgitation, and he occasionally brought up clumps of blood. His weight had been maintained. He had occasional acidity. Up to a month ago he had been a free smoker, but there was no history of alcohol. Twelve months before he had had syphilis. The physical signs were remarkable. There was dullness over the right front from the second space downwards, and inwards to within half an inch of the right margin of the sternum, the resonance of the left lung extending across the mediastinum to this line. The maximum impulse of the

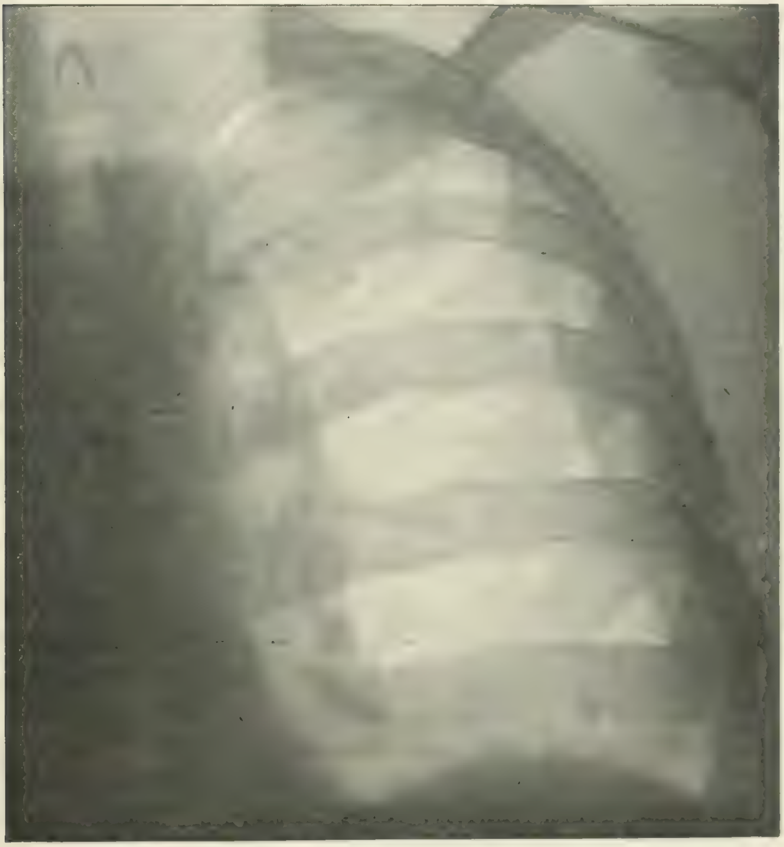

FIG. 2.

Skiagram of right side, from the same case, showing a less degree of the same condition.

heart was felt at the fourth intercostal space in the right anterior axillary line, but on careful palpation and auscultation the apex beat was made out to be at the fifth intercartilaginous space about half an inch from right margin of sternum. The liver dullness extended to within the margin of the cartilages and the liver could not be felt below. Posteriorly there was general dullness over the right side, the dullness being absolute from the midscapular level downwards, but the upper line of complete dullness was somewhat convex, the margin of lessened dullness extending a little downwards both in the interscapular and axillary regions. Over the region of complete dullness both respiratory and vocal fremitus were practically absent. The dimensions of the side were, however, not notably altered, but the movement was lessened. Above the complete dullness in the interscapular region below the spine of the scapula the breath-sound was imperfectly cavernous in quality, the voice pectoriloquous, and a few coarse crackles were heard with an occasional flatulent gurgle communicated from the œsophagus. In the supraspinous fossa and in front the respiratory sounds were somewhat blowing but weak. The resonance of the left side of the chest was full, and extended over the sternum to a little beyond its right margin. The respiratory sounds were coarsely vesicular. The stomach resonance was over the normal area. There was no abnormality notable about the heart except its position.

This man's mother and maternal uncle had died of phthisis. He had passed for life assurance after medical examination nine years ago, and up to eight years ago could bicycle 100 miles. There was no history of his having inhaled any foreign body nor of any pleuritic illness or attack suggestive of pneumothorax; and Sir Richard was at a loss to account for the extraordinary degree of displacement of the heart, and could only suggest a densely fibroid lung with possible excavation at the apex of the lower lobe and a cartilaginous caseo-tuberculous pleura. The history pointing to these was, however, quite deficient, and he had learned the further interesting fact that as recently as September last the patient had consulted his colleague Dr. Young, who then found only a slight impairment of resonance at the right base, the position and sounds of the heart being normal.

The skiagram bore out generally the physical signs and showed the enlargement of the left lung and the complete displacement of the mediastinal contents to the right of the spinal column, also a remarkable corresponding divergence of the lower trachea and main bronchi to the right. It showed also that the case was not one of pleuritic effusion and that no foreign body was impacted in the bronchus. Growth seemed to be excluded, and probably the case might ultimately prove to be one of tuberculous and, perhaps, loculated pleura with fibroid collapse of the lung. The history of syphilis was too recent for a gummatous explanation of the condition.

Mr. C. R. C. LYSTER said that, in regard to the case of miner's lung mentioned by Sir Richard Douglas Powell, it might be taken as a lesson from the radiographic standpoint. From what Sir Richard Powell said, he did not regard it as a case of tubercle, and yet the markings or mottling in the bronchi would be difficult to distinguish from those due to tubercle. He considered that in that case there was an actual deposit of earthy salts from the mine in the bronchi, causing an irritation fibrosis; and probably the deeper markings towards the hilum were due to enlarged glands, as the result of this chronic form of irritation.

He thought it right to give the result of his small experience on the question of diagnosing tuberculosis by X-ray plates and screens. He confessed he could not be certain that mottling in the bronchi was due to tubercular infection only. He had seen cases of small patchy bronchopneumonia in children suffering from whooping-cough, which he would read as identical with cases which had been shown and which he had been assured were tubercular. He hoped and believed a time would come when the X-ray plate would enable one to say which case was tubercular and which was not, but he did not think that time had yet arrived.

Dr. DAVID LEES desired to add a word or two to the remarks he made at the last meeting. He was grateful to Dr. Bythell for his invitation to visit his Manchester clinic, and regretted that he had not been able to accept it. But even if he had been able, he would have felt that a screen examination not reinforced or corrected by a skiagram was not absolutely trustworthy, and might lead to error of interpretation. By kind permission of Dr. Voelcker, Dr. Ironside Bruce and he had examined three cases at the Hospital for Sick Children, Great Ormond Street, in which he found percussion signs which seemed to him evidence of a present or past tuberculosis.

In the first case, on screen examination, there was some general increased opacity of the right lung, which was more definite and discrete at the root, and extended towards apex and base. (No note about the left side.) Diaphragm movements equal, right diaphragm flattened.

Skiagram: Increased opacity of both lungs, made up of fine striations, the right lung being particularly involved. Throughout both lungs *small rounded opacities arranged in groups*, more numerous though less opaque and less sharply defined at the right base and apex.

In the second case percussion signs again showed an excess on the right side; both sides were affected. Screen examination: Some general increased opacity of right lung, more marked towards the root; extends towards base and apex—most marked towards apex in second interspace. The left root showed some increased opacity. Diaphragm movements normal and equal.

Skiagram: Both lungs showed opacity, most markedly the right, and towards both roots. The increased opacity was made up of coarse and fine striations, but was also due to *rounded opacities arranged in groups*. These latter abnormalities were more numerous throughout the right lung, particularly the right base, but were present also on the left, the apex being principally affected.

In the third case the physical signs were nearly equal on the two sides, but rather more on the right. On screen examination there was increased opacity of right root, extending more markedly towards the base, but also to some extent towards the apex. Left lung showed nothing abnormal. Limited movement of right side of diaphragm.

Skiagram: Both lungs showed a considerable degree of increased opacity, which was coarsely striated, but there were also fine striations. The whole right lung was involved, there being present, in addition to the striæ, *small rounded areas arranged in groups and distributed throughout the whole lung*, but most numerous at the base and at the level of the second interspace. On the left the increased opacity was made up of coarse striations, which occurred at the root and towards the apex. In the latter situation a *few rounded opacities* were present.

The question requiring to be settled was whether these groups of opacities, which came out so distinctly on the X-ray plate (though possibly not always on the screen), definitely meant tubercle, or whether they might be the sequelæ of bronchitis or other conditions. In his own experience, they corresponded to a percussion dullness which he regarded as characteristic of tubercle. That was a proposition which he did not wish anybody to accept on his authority; he hoped they would test it for themselves; it required testing by careful and accurate observation of many cases.

Dr. SYDNEY A. OWEN said he felt some diffidence in addressing this Section, because he knew only too little of the technique of radiography. Through the kindness of the President he had been asked to express his opinion, from the standpoint of the clinician, as to the value of X-rays in the early detection of tuberculosis of the lung. Personally, he laid great stress on the value of radiography in this connexion and employed it extensively in his clinical practice. This he was enabled to do through the kindness of his several colleagues. He would like to take this opportunity of thanking Dr. Morton, Dr. Hugh Walsham, Dr. Stewart, Dr. Salmond, and Dr. Hampson, not only for permission to show the plates, but also for the numerous occasions upon which they had assisted him. He thought the best way to attempt to convince others of the value of radiography in this connexion was to try to correlate, by graphic methods, symptoms and physical signs, as elicited by routine physical examination, with the appearances found in negatives. To do this he had prepared, and would throw on the screen, a card giving a summary of the symptoms, signs, and radiographic reports, and at the same time illuminate the corresponding negative. He had arranged a series of cases under four groups. In this series there was a gradual gradation, clinically, from cases with tubercle bacilli in the sputum, definite symptoms and signs (Group I), to cases where the signs and symptoms were very slight (Group IV). In Groups I, II, III, the negatives always showed definite changes; in some cases the disease, though the physical signs were slight, was found to be more advanced than clinical examination indicated. In every case in Group I the radiologist had independently confirmed the tentative clinical diagnosis prior to the detection of tubercle bacilli; in one case as long as three months. Inasmuch as the appearances in all negatives in Groups I, II, III, seemed to him to be identical in kind, differing only in degree and distribution, was it not permissible to assume that in those cases where tubercle bacilli were absent (Groups II, III) the radiographic examination afforded, if not confirmatory, at any rate strong presumptive evidence in favour of tuberculosis? He was quite prepared to admit that great caution was necessary in the correct interpretation of shadows in the region of the hilus, but the densities he wished to draw attention to, and upon which he himself laid great stress, could be seen high up in first and second spaces, and, in some cases, at the extreme apex of the lung. In those cases where the radiographic appearances were unilateral the contrast was most striking. In his opinion, whilst it might be very difficult to define the appearances of a healthy hilus, the apical portions of the lung in health give a very definite picture. He would now throw upon the screen and illuminate the negatives referred to.

Group I comprised five cases (males—one, aged 16; females—four, aged 10, 27, 27, and 35). The evidence of tuberculosis was positive, tubercle bacilli having been found in the sputum. In addition there was a definite history of recent immediate contact from other members of the same family in three cases. All the patients had definite symptoms of lung disease—e.g., cough and expectoration. In three hæmoptysis occurred. In all the physical signs were slight but definite—e.g., slight percussion dullness, weak air entry, and in two cases a few fixed râles. In the face of evidence such as this he did not think these patients were mere tubercle carriers. In all the negatives showed very definite changes. In the case of the male patient, aged 16, a positive diagnosis of tuberculosis by the rays was made in April, but tubercle bacilli were first detected in the sputum in the following July. A second skiagram of this patient's chest, taken six months after the first, gave an almost identical picture. Surely this was good evidence that the densities seen on a plate, though merely shadows, were not due to mere accident. In all the other cases in this group, as already stated, a diagnosis of tuberculosis radiographically had been made by an independent person prior to the detection of bacilli in the expectoration.

Group II comprised three cases (all females, aged 11, 7, and 27). Tubercle bacilli had not, up to the present, been found in the sputum, but the speaker did not think there was any question as to the diagnosis of tuberculosis of the lungs being correct, because in the case of the child aged 11 (*a*) the mother had died recently of tuberculosis; (*b*) the symptoms were definite—e.g., cough and wasting, but no expectoration; (*c*) the physical signs in the lung were definite; (*d*) the temperature during six weeks ranged between normal and 100·6° F.; and (*e*) recently the child had developed tuberculous irido-cyclitis, a diagnosis which had been made independently by an ophthalmic surgeon. The negative showed very definite apical mischief, especially on the right side where the physical signs were chiefly in evidence. In the case of the child aged 7, in addition to cough, hæmoptysis, and definite physical signs in the lung, there was a large breaking-down, tuberculous gland in the lower neck on the right side. There was some question, too, as to whether the child might not have tuberculous disease of the spine as well. The negative showed a definite lesion. The contrast with the other side was very well marked. In the case of the patient aged 27, who had been under treatment for tuberculous irido-cyclitis since September, 1910, the physical signs in the lung were very slight. The sputum was negative for tubercle bacilli, and the von Pirquet's reaction was positive. A definite diagnosis of tuberculosis of the lungs was made radiographically by an independent person in April, 1912. The patient had her first hæmoptysis in March 1913.

Group III comprised nine cases (four males, aged 31, 28, 27, and 26; and five females, aged 27, 9, 11, 31, and 22). The proof of tuberculosis of the lungs, from the clinical side, rested upon (*a*) recent immediate exposure from one or several other members of the same family in six cases; (*b*) the presence of definite symptoms, such as cough and expectoration, in eight cases, hæmoptysis in three cases, pleurisy in one case, wasting in three cases, and continuous fever in one case; (*c*) slight, though definite, signs at one or other apex of the lung. In all, the negatives showed definite changes.

Group IV comprised four cases (two males, aged 17 and 33; and two females, aged 35 and 23). The evidence in favour of tuberculosis was very slight from the clinical side, so far as symptoms and signs were concerned, but though slight they were very suspicious. In two cases the negatives showed appearances which, in the speaker's opinion, were indicative of very early mischief. In two the changes, radiographically, were so slight that a tentative diagnosis had been made.

Dr. N. S. Finzi: During the two evenings of this discussion we have heard a great deal about the interpretation of the shadows at the roots of the lungs but nothing of the technique of obtaining the skiagrams and its bearing upon the results. It is upon these subjects that I propose to say a few words.

Dr. Lees mentioned during the discussion the improvement in technique during the past five years, but what is it that has caused this change? I answer, most unhesitatingly, instantaneous radiography. Yes, gentlemen, instantaneous or snapshot radiography has absolutely revolutionized our results by giving us detail in the negative that no human eye could recognize on the fluorescent screen. Further, with still shorter exposure than we at present can give we shall get even better results, for we are a very long way from perfection in this branch of radiography. Next to instantaneous radiography, shortening of the exposure so as to skiagraph a patient while holding his breath was the most important advance effected, but for most of us this was many years ago.

I now show you three slides of skiagrams taken from the same case one after the other with the same tube. The first is a time exposure with the patient breathing quietly. Notice the blurring of the hilum shadows and the absence of any detail in the structure of the lungs even of the apices, which move comparatively little. The second is a time exposure with the patient holding his breath. Each of these was taken in ten seconds. In this second skiagram the hilum shadows are fairly clear, but details of lung structure leave much to be desired. The third was taken in a fraction of a second, and we now begin to see a clear picture of the hilum and details of the structure of the lungs—not enough of this latter, as the exposure is not short enough, being about a quarter of a second, though nominally one-tenth. The great point in this method is that heart movements are eliminated as well as respiratory movements. It is essential, however, to give a full exposure, as under-exposure in itself results in loss of detail, and provided the exposure be sufficient, softer tubes give better results than hard.

Lately, exposures have been made in times from one-eightieth to one-two-hundredth of a second by what is known as one-flash apparatus, but it is impossible by this method at present to obtain a satisfactorily exposed plate of an adult chest without using an intensifying screen. Now the modern intensifying screen gives very little grain, but the grain is sufficient to detract from a picture of the lungs, and I look forward to the time when we can get a powerful enough flash to give a well-exposed negative of the chest without any intensifying screen. Then, indeed, will no physician be able to ignore the value of this method of examination.

I show you two other slides, one of a normal chest showing how marked the peribronchial shadows may be, and another of a case of chronic bronchitis showing only an increase in density of the normal lung structure.

I regret that I have not had the opportunity to look out some plates of phthisis.

Now as to the scope of X-ray examination in phthisis. First of all I must emphasize the fact that a skiagram is only a physical sign, and though I believe it is a very valuable one, in fact, the most valuable one, still it is nothing more than a physical sign. It is only a link in the chain of evidence for or against the disease. As to whether X-rays can detect more than other physical signs it is very difficult to express an opinion. Personally, I believe they can, for in undoubted cases of phthisis it is usual for the skiagrams to show not only more than can be detected by clinical methods, but more than can be inferred from them, especially in parts other than the apex. The demonstration of quiescent calcified nodules is of no value to the physician, and these must be carefully distinguished from active disease, and it is for us to train ourselves to do this. To show that 50 per cent. or more of healthy people are suffering or have suffered from phthisis is of no value in diagnosis. As to hilum shadows, when we consider what a quantity of dust and dirt the lymphatic glands in this region have to deal with, it is not surprising to find considerable shadows here, and we have no right to assume that they are tuberculous unless a definite tuberculous structure can be shown post mortem. Has it ever been proved that these deposits are calcareous and not silicious? Further, the demonstration of a shadow in one particular part neither proves that the disease began in this part or that this shadow represents the entire diseased area; more than this, it seems probable that in the early congestive stage of phthisis no shadows will be thrown, and it is only when caseation, fibrosis, or calcification begins that radiographic evidence is obtained. Fortunately, caseation is a very early change.

Another point I wish to deal with is the translucency so common in an apex undoubtedly affected, and several skiagrams were shown at the last meeting illustrating this. I consider that this is very frequently due to a cavity at the apex, and often a careful examination of the skiagram will reveal this, and, in fact, I thought I could detect such cavities in some of the slides shown. In skiagrams a cavity is not a very striking thing, and it needs carefully searching for—it does not hit you in the eye. Some years ago I had the good fortune to radiograph a case of secondary sarcoma in the lungs, and the patient succumbed the next day. Before I saw the plates I had seen the lungs themselves, and there was a large cavity at each apex. Though I should previously have missed them, these were quite easy to distinguish in the plates, but the only change presented was an increased translucency and greater detail over an area which was sharply limited by a sudden change to a denser part with less lung structure visible. I have subsequently frequently been struck by the small amount of change which an apical cavity produces.

It might be thought that the examination of chests post mortem would help considerably, but as the examination in the living should always be made during deep inspiration, it must be borne in mind that it is equally necessary to inflate the lungs of the corpse.

Dr. R. A. YOUNG wished only to make a few criticisms and ask questions. The subject was of great practical interest and importance, but he thought it must be approached from the clinical standpoint—i.e., as an applied method, rather than as an academic matter. He was somewhat disappointed at the last meeting, because he thought the results were too theoretically considered. The object of the meeting was the clinical evaluation of X-ray findings in intrathoracic tuberculous disease. The earliest possible diagnosis of such disease was the object for which physicians were striving in different ways. The outlook of many speakers at the last meeting seemed to him to be too academic. The determination of the sites of tuberculous deposit in the thorax was primarily a pathological question, and could only be settled by pathological research. That the earliest deposits, in most cases, were in the bronchial glands was not seriously disputed in the case of children. The relationship of these deposits to tubercle later in life was still a debated question. Was the apical deposit in adults a recrudescence of the old lesions, or was it a reinfection? He did not look to X-rays to decide this question, but to pathology. There was a danger that if too great stress were laid upon the X-ray appearances they might suffer the fate of some of the other clinical methods which had been applied to diagnosis and had showed too much; for example, the von Pirquet reaction, which was too delicate, except in young children, to be of practical value. It was necessary to know the exact significance of the chest shadows, especially those at the hilus of the lung. Post-mortem records, like those of Dr. Jordan and other observers, might show the frequency of old healed or arrested lesions in the bronchial glands, and even at the apex of the lung. There was nothing new in that; what was wanted was some further evidence that the disease spread from the hilus to the apex, in the way it had been said by Dr. Jordan to spread. It was probable that the spread took place by the lymphatics, and the name "peribronchial phthisis" did not appeal to him. He would certainly require further pathological evidence before accepting it. And he still felt he would distrust X-ray findings which were not definitely correlated with symptoms, or even with physical signs. He wished to ask whether X-rays could show a deposit in the bronchial glands of children before there was clinical evidence of such deposit? Secondly, could X-rays differentiate old healed, quiet lesions from recent, active, progressive ones, whether at apex, hilus, or base? Thirdly, was it clear yet how much of the X-ray appearances seen at the root of the lung were normal? And of those which were pathological, were they due to old quiet lesions or to active tuberculous processes in the bronchial glands, or at the hilus of the lung? Fourthly, accepting Dr. Jordan's statement as to the line of march, could the X-rays show progress from the hilum to the apex at an early stage, before there were recognizable physical signs? And, if so, what were those X-ray indications? Lastly, in basic conditions, which always seemed to cause most difficulty in diagnosis, would the X-rays give definite information in distinguishing between recent active infiltration, thickened pleura, bronchiectasis, and fibrosis?

He considered such meetings as the present one were of very great value, because they enabled those who were looking at the subject from different standpoints to gain further information, and to appreciate different aspects of the problems they were investigating.

Dr. GILBERT SCOTT said most of the points he had intended to bring forward had already been dealt with by the previous speakers. He quite agreed with what had been said on the subject by Dr. Finzi and Dr. Young. What made the radiographic diagnosis so difficult in chest cases was the fact that the normal radiographic appearance was still undecided. Skiagrams taken of what were regarded as normal lungs would show wide ranges of difference. In the case of people who lived in changeable climates, the amount of inflammatory change remaining in the lungs must be considerable, leaving behind fibrous tissue in a more or less degree, with enlarged and calcareous glands at the roots of the lungs; practically all those living in this climate had these conditions present. He did not think it justifiable that a person should be diagnosed as "tubercular" simply because this condition was present in his lung; a condition which might be caused by simple inflammatory changes other than that carried by the tubercle bacillus. It was as well to remember that even the typical mottling of phthisis, which might be seen at the apex or elsewhere in the lung, might be produced by other conditions. He mentioned a case of a man, aged 60, who showed at one apex the typical mottling of early phthisis. He died soon afterwards, and at the post-mortem the appearance was found to have been caused by collapse of that portion of the lung, due to a malignant gland having pressed on a bronchus. This portion of the lung was adherent to the chest wall and there was no air entry.

Dr. R. MURRAY LESLIE said he would like to dwell briefly on one or two points which had emerged from the debate, particularly one or two aspects of the question under discussion just brought out by Sir Richard Douglas Powell. One of these had reference to statements by recent writers, including the speaker, that the large majority of all persons were the victims of tuberculous infection in early life, and if that were so, why it was that the death-rate from phthisis was not much higher than it was known to be. It seemed to the speaker that this apparent contradiction was adequately explained by the observations of such investigators as Hamburger and Monti, of Vienna, who had found that although 94 per cent. of school children contracted the infection in some form before the age of 15, yet 80 per cent. of these never showed definite symptoms of disease. This was, no doubt, due to a permanent cure of the tuberculous deposit at the hilus of the lung and elsewhere having taken place, and to the establishment of a gradually acquired immunity, which rendered the individual less liable to subsequent infection. It would thus seem that the previous existence of old cured tuberculous foci at the hilus might be an actual advantage to the individual on account of the comparative immunity thus conferred. With regard to the spread of the disease in the reverse direction to the course of the lymph-stream due to the "retro-impulsion of lymph," and fully described in the speaker's recent publication on "Hilus Tuberculosis,"[1] Sir Almroth Wright and others had shown that this could happen in the case of plague and other infections, and that this backward spread could be readily observed in the case of tuberculous cervical glands in children. He agreed with Sir Richard Powell as to the difficulty of judging from an X-ray examination as to the direction in which the spread had occurred, whether from the hilus to the periphery or vice versa. This could only be ascertained by carefully analysing a series of successive skiagrams of the same patient taken at certain intervals. As Dr. Finzi pointed out, it was only during the last twelve months or so that instantaneous skiagrams had become possible; the use of the instantaneous method, in conjunction with the recent improved technique, promised valuable results in the future.

As regards the X-ray findings, he agreed with Dr. Ironside Bruce and other radiographers as to the importance of the patient holding his breath during examination, as otherwise the clusters of tiny opacities which frequently indicated foci of tuberculous broncho-pneumonia did not stand out clear and defined, and there was only a blurred result difficult to interpret. In the case of the shadows cast by hilus thickenings, he agreed it was difficult to state whether they *per se* were due to tuberculous or non-tuberculous fibrosis (i.e., silicosis and other conditions); he would point out, however, that even in the latter case it was difficult to exclude the co-existence of tubercle. When the appearances were limited to the root area the question could not be decided on X-ray evidence alone.

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[1] *Clin. Journ.*, January 22, 1913, xli.

Dr. E. L. Collis said that in his work as a factory inspector he had given much attention to tuberculosis, and he had endeavoured to get a series of X-ray photographs taken of persons much exposed to dust. The first attempt was carried out by Dr. Rayner, but the patients were tuberculous, and it was impossible to say that the extended area of shadows was not due to tubercle. He then got Dr. Hallam, of Sheffield, to radiograph for him some grinders who were not tuberculous, but had silicosis, due to the inhalation of dust of uncombined silica. Tubercle in such individuals runs a rapid course, the patients may be dead in six months. These men were alive and well twelve months after the photographs were taken, and the only apparent abnormal symptom was a shortness of breath. Yet the plates showed shadows which those accustomed to interpret them considered to be typical of disseminated tuberculosis.

Dr. A. E. Barclay said he was far from convinced as to the prevalence of peribronchial phthisis. Since Dr. Jordan read his paper some time ago he had set himself to look at all the normal chests he encountered, including a good number of stomach cases, in whom, presumably, the lungs were healthy. He had examined some 400 cases, and the root shadows which had been described were present in practically every case. Many of them showed thickened and enlarged glands. He was quite sure they had not *all* got peribronchial phthisis. He would diagnose the presence of peribronchial phthisis only when he was convinced that these shadows were of active pathological significance. The only way in which one could arrive at this conclusion was by a comparison of instantaneous skiagrams, taken at intervals of a month or two. If this method showed *extension* of the root shadows, then, and not till then, he would diagnose peribronchial phthisis.

Dr. W. J. S. Bythell, in reply, said the discussion had been exceedingly useful and instructive; but he had heard very little to throw doubt on his contention that in children—and his remarks were specially confined to them—the disease invariably started at the root of the lung. Sir Douglas Powell misunderstood his figures when he quoted that 94 to 97 per cent. had phthisis, and he was scornful as to the prevalence of bronchial tuberculosis at Manchester. At the Manchester Children's Hospital, in children dying from all causes the proportion was 55 per cent., which was below the rate given by most pathologists. Authorities varied, but from 60 to 90 per cent. of children dying from all causes were said to have tubercular bronchial glands. The majority of children no doubt got over the infection, leaving fibrous or calcareous glands. But that did not invalidate the argument that as long as bacilli were there the danger-spot was the infected bronchial glands. He agreed with Dr. Lees that in every case the screen examination should be confirmed by taking a plate, as the latter showed much more; also no doubt the skiagram should be an instantaneous one if possible. In criticizing his (the speaker's) contention as to pulmonary tubercle being a root infection, Dr. Lees said very often the tubercle bacilli entered the system through the bronchial mucous membrane; but he (Dr. Bythell) did not see that that made any difference. If a child swallowed a big dose of the bacilli, they passed through the alimentary mucous membrane and got into the lymphatics. If they secondarily infected the bronchial glands, in most cases these glands overcome the infection, but in others the glands broke down down and caseated. According to Dr. Lees, this should be regarded as a primary infection of the digestive tract, but his (the speaker's) contention was that the first pathological lesion in the lung was an infection of the bronchial glands, extending thence to the lung tissue in the root. If that were so, the recognition of this area of disease would result in the child being brought under treatment much earlier than otherwise.

It was necessary to insist at such a meeting as this, at which radiographers and clinicians were present, that it was not a question of the radiologist trying to upset the diagnosis of the clinician, or vice versa, or seeing which department could secure the earliest diagnosis. One had simply to regard the X-rays as a sixth sense which had been put into the hands of the profession, and which must be used in combination with clinical methods. In his hospital work he got the physicians to come down to the X-ray room to see the screen examination with him, and examine the plate, and the diagnosis was a result of mutual discussion. For instance, the presence of a shadow at the root, without other indication, he would hesitate to diagnose on; but if there were also wasting, a cough, a temperature, a positive von Pirquet, and so on, he would diagnose the presence of active tubercular infection of the root. He would like to ask radiologists present whether they had ever seen, even in the adult, a heavy apical shadow due to tubercular consolidation which was not accompanied by a much heavier shadow at the root. Had anyone seen a solitary focus in any part of the lung except at the root? If so, they had seen something which he never had.[1]

Dr. HORTON-SMITH HARTLEY said that the contention of Dr. Bythell that in children tuberculous disease of the lung originated in and extended from the bronchial glands might be true in not a few cases, but he thought it would be a pity for the meeting to dissolve with the belief that the same was necessarily true in the adult. He believed himself that this was not the case, for the conditions in the adult were not the same as in the child. In the adult patient suffering from phthisis, caseation of the bronchial glands was the exception. In 263 post-mortem examinations, which he had made at the Brompton Hospital, upon patients dying of chronic pulmonary tuberculosis, in only 9·1 per cent. were the bronchial glands found caseous. The antecedent condition, therefore, which made probable direct extension of disease from the bronchial glands into the lung, was of comparatively rare occurrence in the adult. He asked, further, whether the findings of morbid anatomy supported the doctrine that in the adult pulmonary tuberculosis started at the root of the lung, and he believed that they did not. When, for example, one made a post-mortem upon a case of phthisis, one lung was, as a rule, found to be extensively involved; in the opposite lung, on the other hand, the primary area of involvement was usually in the upper part, near the apex (whence the disease descended downwards through the organ), and not in the hilus or near the root of the lung. Again, when a person died and the lesions of arrested tubercle were found unexpectedly in the lung, in what portion of the organ were they discovered? Nearly always in the apical region, and very rarely near the root, whether such lesion consisted of a small calcareous nodule, an area of caseation surrounded by fibrosis, or a small cavity. He believed, further, that clinical observation agreed with such findings, and that very early in the disease altered physical signs could be discovered in the region of the apex, upon which, in conjunction with the symptoms, a diagnosis could be based. He agreed with Dr. Lees that in the majority of cases alteration of the percussion note was an early and most important sign. With regard to the X-ray appearances seen in the neighbourhood of the hilum in so many people, he believed that much further observation was required before we could be certain as to the interpretation of these shadows. He thought it very doubtful whether in most cases the radiating lines and striæ really represented areas of tuberculous infiltration, though the darker spots probably did reveal old calcareous foci in the bronchial glands. A valuable piece of research would be to examine a number of patients radiographically shortly before death, and then to compare the photographic appearances with the post-mortem findings. He ventured to suggest that in the cancer wards of the Middlesex Hospital such work might be done. At the present time even X-ray experts did not agree as to the meaning of the root shadows. There was, therefore, great need for a careful series of observations, so that the exact interpretation of these shadows might be definitely settled.

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[1] In the report of the meeting held on February 21, I regret that I failed to notice the omission of the reference to my quotation from Dr. Murray Leslie's paper on "Hilus Tuberculosis" in the *Clinical Journal* of January 22, 1913. The last ten lines of the report of my paper on p. 79 of the March Number of the *Proceedings* were a quotation of his views.—W. J. S. B.

Dr. HARRISON ORTON: Having attended practically every discussion on the subject which has been held in London since the first one introduced by Dr. Stanley Green at the Clinical Society in 1904, when, as a matter of fact, there was no discussion at all, I have been much struck by the enormous amount of interest the subject has aroused this evening. It is evident that at last the importance of this examination is beginning to be recognized, for although our technique has improved immensely, chiefly owing to the possibility of obtaining practically instantaneous skiagrams, nevertheless, the main points brought forward in its favour are much the same. For years we have been familiar with the shadows at the roots of the lungs, the radiating lines, and mottled shadowing; nine or ten years ago Dr. Hickey, of Michigan, showed that these lines were due to the pulmonary vessels. Later, they have been shown to be due to the bronchi. The distribution of these is so similar that it is difficult to say from an examination of injected specimens to which they may be due, and I am not yet convinced that they are entirely due to bronchi, and still less that they indicate peribronchial tubercle, which some speakers seem to imagine. In justice to Dr. Jordan, however, I think I ought to say that I do not think he ever intended it to be understood that the lines were the result of tubercle, but the mottled shadowing around them. Is there any reason why, in part at any rate, these lines should not be due to vessels? (Blood is an opaque material.) Has it ever been disproved that the mottled shadows may in part be produced by congestion around tubercular foci? Because we have glands at the root of the lung, is this any proof that the disease starts there? As one speaker has aptly put it, the glands are "dust-bins." A dust-bin must have refuse from somewhere to fill it. Where does the refuse in this case come from? At what stage is a tubercular lesion capable of casting a shadow? May not a lesion exist incapable of detection by any method at our disposal? Or perhaps attention is not called to it. A lesion which the dust-bins, in some cases, are able to deal with, the shadows in question being the result, then, of healed lesions. Whatever be the explanation, shadows at the roots of the lungs are almost always present in town-dwellers at any rate, radiating lines always, and therefore I think it must be admitted that within certain limits they are of no special importance in the diagnosis of pulmonary tuberculosis; we need some further evidence. However, one point seems clear—namely, that purely apical lesions are seldom seen on X-ray examination, the mottled shadowing is nearly always much more scattered; and I think this tends to show that the disease starts in a more scattered manner than is generally supposed, and is probably not recognized until a definite increase takes place in one particular spot. I agree with Dr. Barclay that a comparison of shadows after some considerable interval is of great value, and Dr. Walsham and I laid stress on this and gave the reasons for it in the little book we published in 1906. It is interesting to note that one negative, shown this evening, of a case in which there was no question of tubercle showed certainly quite as much, if not more, shadowing than some shown as definitely tubercular. The time is evidently not ripe, then, for us to say (to quote Sir Douglas Powell) "There the thing is, and it is no use arguing about it," but, rather, there the thing is, let us try and find out what it is, for our interpretation of "facts" is as yet by no means perfect.

I should like to take the opportunity of thanking Dr. Melville for his kindly reference to the work published by Dr. Walsham and myself some seven years ago.

Dr. WORRALL remarked that what had been said concerned chiefly early cases, but he would bring to notice a case of advanced disease which was sent to him at Westminster Hospital, by Dr. Carmalt Jones, for

X-ray examination. A young married woman, aged 26, was sent up as a case of pulmonary tuberculosis. On examining by the screen with the patient sitting—she could not lie down—he was struck with the great brilliance in the left upper half of the chest, chiefly the outer part. The area was limited below by a sharp horizontal line, and it remained horizontal when the patient inclined to either side. On the right side the lung appeared mottled all over, and near the apex was a large area almost as bright as the extra bright area on the other side. The report sent was, advanced pulmonary phthisis in right lung, the light area at apex indicating a considerable vomica. His interpretation on the left side was pneumo-hydrothorax. The shadow nearer the spine was cast by collapsed lung, and the horizontal line marked the limit of fluid in the chest. There was a rippling of that line synchronizing with the heart-beat. The mamma presented below a sharp outline, and at first suggested pericardium, but by displacing it for screen examination it became evident that it was breast. He would like to know what the explanation of these X-ray appearances was. He had shown these skiagrams to several X-ray experts and all agreed with the interpretation he had given. He would like to hear if anyone had a different interpretation to give, and the reasons for the conclusions.

No other interpretation of the X-ray appearances being suggested, Dr. Worrall proceeded to say that the patient had since died, and he showed post-mortem photographs of the two lungs. The right lung presented tuberculous disease throughout, with a large vomica in the apex, as suggested by the X-rays. The X-ray diagnosis as regards the left lung was quite wrong. There was no pneumo-hydrothorax. The lung presented complete unilocular cavitation, the fluid was contained in this cavity, and a catheter passed through a large bronchus communicating with the cavity showed where the atmospheric pressure was transmitted into the cavity. There was no fluid between the parietal and visceral pleuræ, which were adherent.

Dr. A. C. Jordan: The discussion has turned largely on my own work, and has been most instructive to myself. No real divergence from my views and results has been expressed, but the discussion has shown that much misunderstanding exists concerning my views. I am glad to have this opportunity to explain matters.

The lungs of all healthy persons show blotches at the roots, and linear shadows radiating from the roots: consequently, these appearances are *not* indicative of active tuberculous disease of the lungs. I have demonstrated their true nature, and shown that the blotches are due to enlarged glands at the roots, while the linear shadows are thrown by fibrous tissue surrounding the air tubes. Since calcareous nodules are present in these glandular blotches in most cases, it is clear that they represent healed tuberculous infection. I am quite prepared to admit, however, that other bacteria may contribute to their formation.

I should like to point out that I hold no theory regarding the mode of infection in pulmonary tuberculosis; in my papers I have merely described facts observed by myself, and stated my deductions from those facts. Given the facts, the deductions follow naturally.

Several speakers have referred to me as holding the view that the glands at the roots are the primary source of infection of pulmonary tuberculosis. As I explained at the last meeting, I have never held this view; I regard it as illogical, just as I should regard the parallel statement that a tuberculous finger was secondary to tuberculous glands in the axilla.

The ordinary course of events in the early stages of phthisis I find to be as follows:—

(1) Catarrh of the air tubes; their lumen is found to contain epithelioid plates and secretion. This stage gives no X-ray signs.

(2) Migration of bacteria (including tubercle bacilli) through the walls of the air tubes, and consequent accumulation of leucocytes around the bronchial cartilages. This peribronchial small-celled infiltration gives rise to the radiating mottling, which is the characteristic X-ray feature of peribronchial phthisis. Even in its earliest stage this mottling is not confined to the roots but may extend to the periphery. The important feature is that from the commencement the mottling is distributed in a radial manner from the roots, and is purely peribronchial in its pathology. Hence the name "peribronchial phthisis" seems to me appropriate, and free from objection, while such names as "hilus phthisis," "central phthisis," &c., are not in any sense distinctive, while they suggest the erroneous idea that the hilus glands are the starting point of the disease. It is true these glands are often found to be caseous, especially in children, and sometimes they break down and cause infection of the neighbouring lung by direct contact; but this is an accidental occurrence, and has no bearing on the ordinary mode of origin of pulmonary tuberculosis.

The "failure to clear up" on inspiration at either apex is usually given as an indication of commencing tuberculous disease at that apex.

I regard this indication with great suspicion: no doubt it does occasionally indicate commencing disease, but as a sign it is open to endless fallacies, and if noted conscientiously will lead to a diagnosis of phthisis in a large number of healthy persons. My advice would be to note this failure to light up, but to be careful not to commit oneself to a diagnosis of phthisis on account of it unless there is actually mottling. The presence of mottling affords a definite sign with a certain pathological significance—viz., points of peribronchial infiltration, and this mottling occurs very early. "Doubtful" mottling is of no value in diagnosis; fortunately, however, the "doubt" can always be cleared up by examining the suspected region through a small aperture. If the mottling is real each individual spot will now be visible and can be pointed out on the fluorescent screen. If the mottling is imaginary the region will appear clear through the small aperture. Hence the golden rule: Do not diagnose phthisis unless you can show mottling through a small aperture, and show it on a photographic plate.

I have endeavoured to avoid repeating the contents of my published papers on peribronchial phthisis, but I hope I have succeeded in making my position clear.

Electro-Therapeutical Section.

April 18, 1913.

Dr. REGINALD MORTON, President of the Section, in the Chair.

Gastric Ulcer.

By ALFRED C. JORDAN, M.D.

IT is my purpose to consider the use of the X-rays in the elucidation of the problems of gastric ulcer, discussing:—

(1) The diagnosis.
(2) The causes.
(3) Some of the terminations.

DIAGNOSIS.

I do not propose to dwell on the diagnosis of acute gastric ulcer; the main interest centres on the chronic ulcers. I would merely state that acute gastric ulcers give rise to a spasmodic constriction at the site of the ulcer, and that the spasm is very persistent. I desire to state very clearly, however, that the converse is not necessarily true. I have many times seen a most persistent constriction at a particular point of the stomach, present at several examinations on different days, and yet at operation there was no constriction and no organic lesion discoverable.

Chronic gastric ulcers occur typically in two situations: at the pylorus and at the middle of the lesser curvature. In the case of the pyloric ulcers the appearance is well known. There is pyloric stenosis, or else (in earlier cases) pyloric spasm, the effect of which is equivalent to that of stenosis, since the spasm is permanent. The stenosis leads to dilatation of the stomach, the appearances of which, on X-ray examination with bismuth, are too well known to require special description (figs. 1A and 1B). One sign, however, calls for special mention—viz., reversed peristalsis. I had to wait several years before I encountered this sign for the first time, but in the last two or three years I have seen it well in eleven or twelve patients (figs. 1A, 3, and 10A). In each case there has subsequently been found an organic lesion at the pylorus—usually a gastric or duodenal ulcer. The phenomenon is quite unmistakable, strong waves passing from the stenosed pylorus, back along the curvatures toward the cardiac end of the stomach. Sometimes the peristaltic waves suddenly change their direction, so that a series of reversed waves is followed by a series of normal waves, or vice versa.

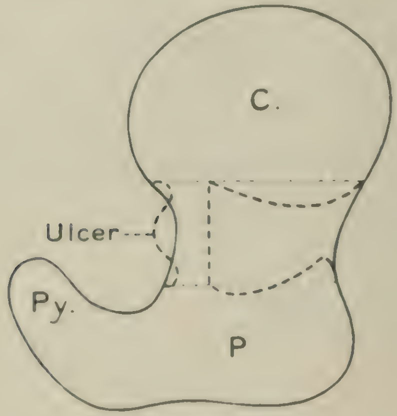

DIAGRAM A.

Stomach (firm outline) showing the production of the typical form of a chronic ulcer of the lesser curvature, with its depressed base and raised margins. The permanent contraction of the circular fibres over the ulcer (in the direction of the two fine dotted lines) gives rise to the hour-glass constriction. The cardiac portion (*C.*) becomes pouched from the weight of its contents. The pyloric portion (*P.*) acquires a nightcap-like projection upwards as the result of the pressure in this hypertrophied portion due to the spasm at the pylorus (*Py.*).

A most important point on which I desire to insist is the necessity of making a complete examination of the gastro-intestinal tract in all cases of gastric ulcer. On previous occasions I have explained the importance of a complete investigation in duodenal cases. When this is done it is found, regularly, that the same cause is at the root of both troubles, and that cause is intestinal stasis. The manner in which stasis leads to gastric ulcer will be clear from the instances I have to show to-night.

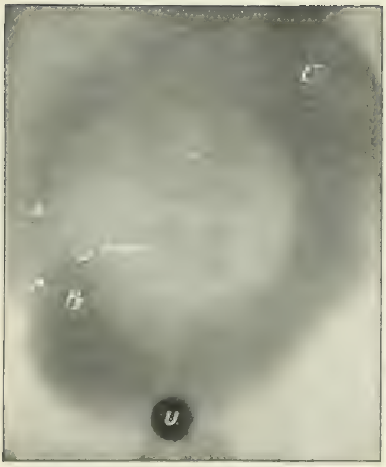

FIG. 1A.

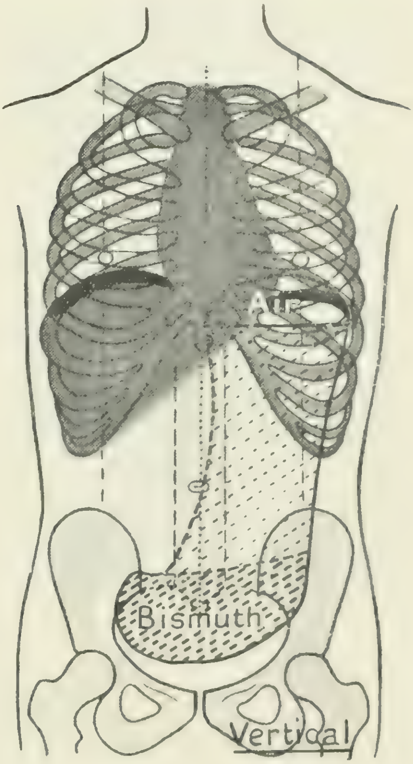

FIG. 1B.

Fig. 1A.—Stomach as shown on the couch after a bismuth meal in a case of pyloric stenosis due to a chronic ulcer in a man, aged 47. The stomach is greatly dilated ; there were powerful peristaltic waves, and on one examination definite reversed peristalsis was observed, strong waves passing, in regular sequence, from the pylorus back along the curvatures. At the end of fifty hours practically the whole of the bismuth was still contained in the stomach. *C.*, cardiac portion of the stomach ; *Py.*, pylorus ; *U.*, umbilicus. The arrows indicate the pyloric stenosis.

Fig. 1B.—Showing the appearance of a dilated stomach (due to pyloric stenosis) when examined vertically after a bismuth meal. The bismuth collects in a shallow pool at the lowest point (in the pelvis). Above the bismuth the liquid contents of the stomach are shown ; a collection of air under the diaphragm occupies the fundus of the stomach, and is separated from the gastric fluid by a horizontal line at which splashing is seen on shaking the patient.

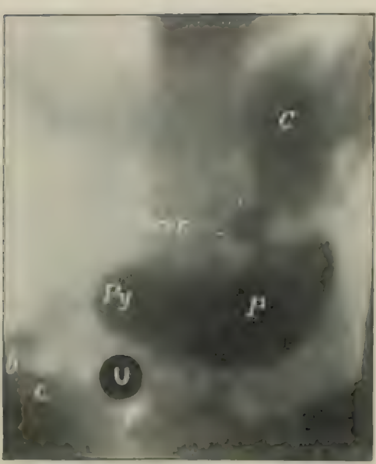

FIG. 2.

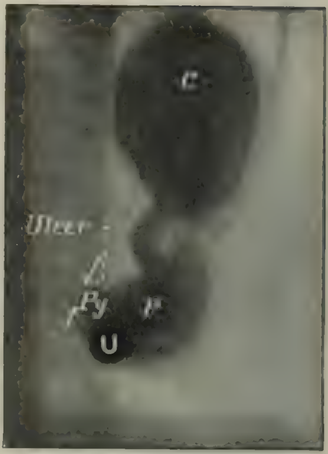

FIG. 3.

FIG. 2.—Taken on the couch after a bismuth meal, showing a typical chronic ulcer of the lesser curvature in a woman, aged 51 (thought to have been carcinoma prior to the X-ray examination). Showing the depressed base of the ulcer, its crater-like margins, and a tight, hour glass constriction due to spasm of the circular muscle-fibres of the stomach over the area of the ulcer, reducing the lumen of the stomach to a narrow passage. This constriction is permanent except under general anæsthesia. The large size of the pyloric portion of the stomach shows that there must have been great dilatation of the stomach before the ulcer existed. The pyloric spasm which caused this dilatation is still present. The duodenum showed well marked "writhing" contractions. After twenty-four hours there was still bismuth in the pyloric portion of the stomach; the terminal coil of the ileum was hypertrophied and tender. The aortic arch showed atheromatous elongation and dilatation—a change very apt to occur in the subjects of intestinal stasis. *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *a*, *b*, *c*, first, second and third parts of the duodenum; *U.*, umbilicus, marked by a penny.

FIG. 3.—Taken on the couch after a bismuth meal, showing a typical chronic ulcer of the lesser curvature in a man, aged 28. There was pain half an hour after food, and tenderness to pressure under the left costal margin. There had been two attacks of hæmatemesis, one four, the other two years ago. In addition to the typical appearances of a chronic ulcer of the lesser curvature (*see* description of fig. 2) there was tight organic stenosis at the pylorus, with strong anti-peristaltic contractions occurring in groups, alternating with groups of strong waves in the normal direction. At the operation two chronic gastric ulcers were found: one at the pylorus, the other on the lesser curvature. There was scarcely any hour glass constriction under the general anæsthetic. *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus. The arrows indicate the pyloric stenosis.

The constant occurrence of duodenal distension in intestinal stasis is now so well known that I shall take it (and its cause) for granted, referring those who have not yet had opportunities of familiarizing themselves with the subject to my recent writings,[1] and to the well-known publications of Mr. Arbuthnot Lane.[2] The effect of duodenal distension upon the stomach is great. The high pressure in the

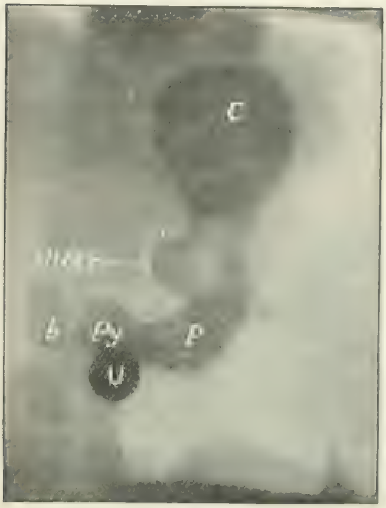

FIG. 4.

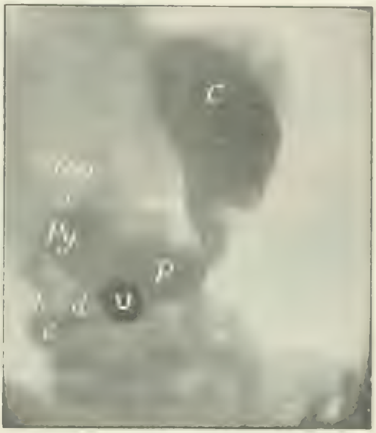

FIG. 5.

Fig. 4.—Taken on the couch after a bismuth meal, showing a typical chronic ulcer of the lesser curvature in a woman, aged 28. She had been constipated and bilious since a girl. Pain, vomiting and wasting for ten months. The typical characters, as shown on the couch, are well seen. The nightcap-like projection upwards from the pyloric portion is very well shown. There was moderate spasm at the pylorus. The duodenum was best seen after one hour; it was dilated, especially in its first part (confirmed by operation). The aortic arch showed slight general dilatation. *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *b*, second part of the duodenum; *U.*, umbilicus.

Fig. 5.—Large chronic ulcer of the lesser curvature in a woman, aged 43. Taken on the couch, showing a modification of the typical form. Abdominal pain and vomiting for a year. Some pyloric spasm; duodenum elongated and tortuous (confirmed by operation). *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *a*, *b*, *c*, *d*, first, second, third and fourth parts of the duodenum; *U*, umbilicus.

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[1] *Proc. Roy. Soc. Med.*, 1911, v (Electro-Therap. Sect.), pp. 9-37; or *Lancet*, 1911, ii, p. 1824; *Brit. Med. Journ.*, 1912, i, p. 1225; *Practitioner*, 1913, xc, pp. 441-54.

[2] *Proc. Roy. Soc. Med.*, Discussion on Alimentary Toxæmia, pp. 49-117.

duodenum causes the stomach to work abnormally hard to get its contents out through the pylorus. As soon as a portion of the gastric contents has made its way into the duodenum the pylorus closes behind it to prevent regurgitation. Physiological experiments show that spasmodic closure of the pylorus always occurs (apart from duodenal distension) when acid materials pass into the duodenum; in gastric ulcer the contents of the stomach are often very acid. The spasm set up in these two ways has a great tendency to become persistent, and to produce a state of affairs closely simulating organic stenosis (figs. 2, 6, 9, and 14). The difficulty of demonstrating radiographically the duodenal distension in stasis is due, in great measure, to this pyloric spasm; it must be overcome, and the best way known to me is to give a large dose of carbonate of bismuth in an emulsion, an

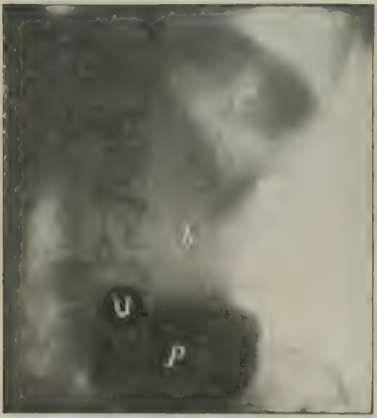

FIG. 6A.

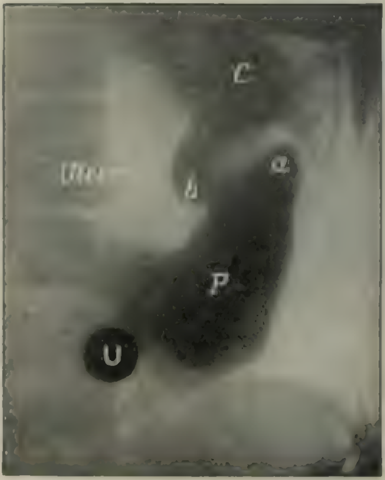

FIG. 6B.

Typical chronic ulcer of the lesser curvature in a woman, aged 48, contrasting the appearance as shown when the patient is examined upright (fig. 6A) and on the couch (fig. 6B). There had been pain and vomiting for seven or eight years. In the vertical posture the depressed base of the ulcer is represented merely by a small collection of bismuth at *b*, while the nightcap-like projection upward from the pyloric portion is shown filled with air (*a*). Note the large size and low position of the pyloric portion of the stomach, showing that there had been long-standing pyloric spasm, which had caused dilatation of the stomach before the appearance of the ulcer on the lesser curvature. After one hundred hours there was still bismuth in the large intestine. *C.*, *P.*, *a*, *b*, are corresponding points on the two pictures.

hour or so after a meal (usually breakfast), and to place the patient on the right side (on the couch) for a minute or two before examining him on his back. Occasionally a little bicarbonate of soda must be added before the pyloric spasm will yield, and if there is an ulcer the spasm will not yield even then, or even with the further addition of morphine and atropine. By this method an accurate report on the condition of the duodenum can be given in nearly every case. On turning the patient from the right lateral to the supine posture the duodenum is seen well filled with bismuth emulsion. In a normal case it is narrow and short, and the bismuth is seen to pass through the duodenum, and on into the jejunum so rapidly that no photograph of it can be obtained with an ordinary apparatus. In stasis, however, the behaviour of the duodenum is quite different; in well-marked cases it is much too long and much too wide, especially in its first part. It shows strong peristaltic contractions, but these are unable to forward the contents

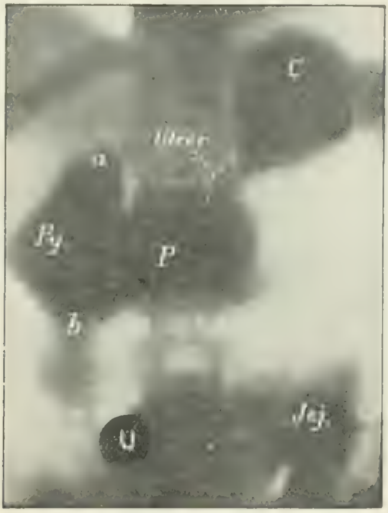

Fig. 7.

Taken on the couch after a bismuth meal, showing a chronic ulcer of the lesser curvature in a woman, aged 37. She had pain twenty minutes after food. Vomiting five weeks. Two weeks ago she vomited 3 pints of blood. The pyloric portion of the stomach is large; the duodenum is elongated, and greatly dilated, especially in its first part (confirmed by operation). *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *a*, *b*, *c*, first, second and third parts of the duodenum; *Jej.*, jejunum; *U.*, umbilicus.

of the duodenum into the jejunum: there is repeated regurgitation of the bismuth from the third and fourth to the second part of the duodenum, and in severe cases the duodenal contractions amount to strong writhing movements. In such cases the bismuth remains in the duodenum many minutes, and it is an easy matter to obtain a skiagram of it by inserting a photographic plate beneath the fluorescent screen, and leaving it there while observing the behaviour of the duodenum. All intermediate stages are found, according to the degree of the stasis. I have described this before, and should not repeat the description were it not that I find there is still a tendency to attribute my results to my method, inferring that a normal duodenum would behave in the way I describe. I cannot repeat too clearly that this is not the case; all my patients take a large dose of bismuth, and are examined in the same way, and the "static" duodenums can be picked out from among the

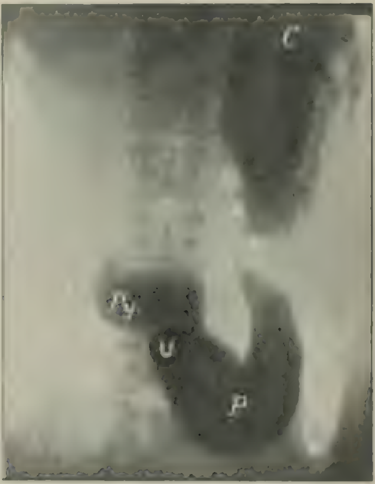

FIG. 8A

Stomach of a woman, aged 45, taken on the couch an hour after a bismuth meal. A very thin streak may be seen joining the cardiac to the pyloric portion of the stomach. More than half the bismuth is still contained in the cardiac portion. The pyloric portion is enlarged and dropped. At the operation, performed a few days later, the stomach appeared as a large sac, showing no sign of an hour-glass constriction, and no ulcer could be felt although it was thought to be there. A gastro-enterostomy was performed. Two days later a fatal hæmatemesis occurred, and the post-mortem examination showed a chronic ulcer of the lesser curvature. *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *a*, *b*, first and second parts of the duodenum; *U*, umbilicus. The arrows point to the hour-glass constriction.

normals with the greatest certainty. My results, both normal and abnormal, have been tested over and over again at operations, and have been found accurate.

EFFECTS OF PYLORIC SPASM.

Returning to the stomach after this necessary diversion, let us inquire what is the result of the continued pyloric spasm; it is similar to that of pyloric stenosis; the stomach, being unable to evacuate its contents in the normal time, becomes overloaded and begins to dilate and to be dragged down by its own excessive weight. The mechanics of this has been fully explained by Mr. Arbuthnot Lane. The stomach is suspended by its two "ligaments," at the pylorus and the œsophagus. The weight of the overloaded transverse colon is added to that of the overloaded stomach, and eventually an ulcer is formed at the point of greatest strain—the pylorus. The pyloric spasm then becomes more complete, and the overloading of the stomach becomes greater still, so that there is little chance for the ulcer to heal. If there is general dropping of the viscera (and the dilated stomach pulls the other viscera down, and if

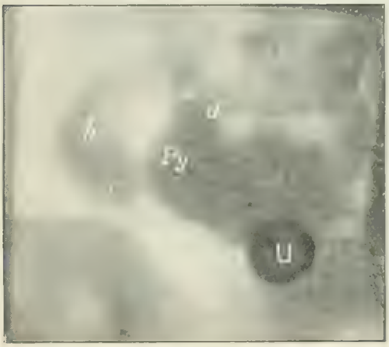

FIG. 8B.

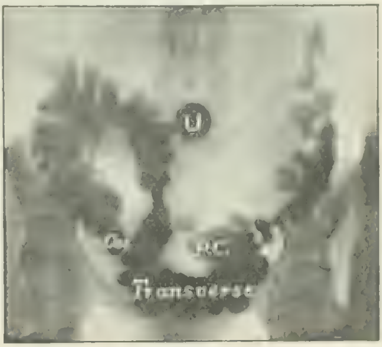

FIG. 8C.

Fig. 8B. Taken on the couch immediately after fig. 8A, showing the [illegible] duodenum. Strong "writhing" contractions were seen, bismuth appearing repeatedly in the fourth part of the duodenum at *d*, and returning again and again to *c* and *b*. *Py.*, pylorus; *a*, *b*, *c*, *d*, the four parts of the duodenum; *U.*, umbilicus.

Fig. 8C.—Taken seventy-two hours after the same bismuth meal, showing great stasis in the dropped large intestine, very little bismuth having passed beyond the middle of the transverse colon after this time. *Cæ.*, cæcum; *P.C.*, pelvic colon; *U.*, umbilicus.

the liver drops, carrying the pylorus down with it, the point of greatest strain is shifted from the pylorus to the lesser curvature, and an ulcer may then appear at this point. It will be understood that the mechanical factor furnishes the predisposing cause; the actual determining cause is microbic, and perhaps in part chemical; the colon bacillus has been found in pure culture in the depressed base of chronic ulcers of the stomach.

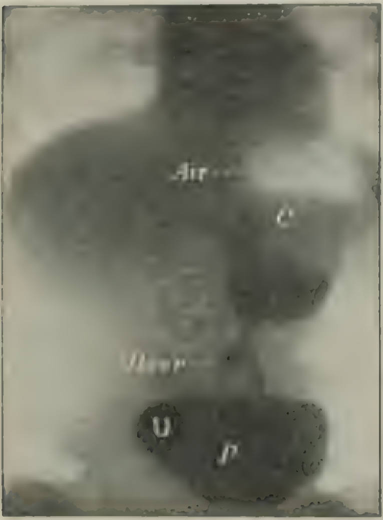

FIG. 9A.

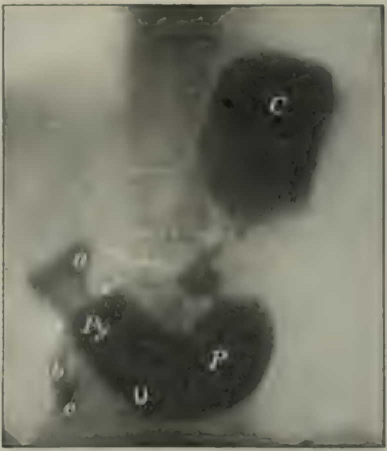

FIG. 9B.

Typical chronic ulcer of the lesser curvature in a woman, aged 58, taken vertically (9A) and on the couch (9B), showing the depressed base of the ulcer and the tight spasmodic constriction of the circular fibres of the stomach over the ulcer. As usual, there was persistent spasm at the pylorus, with dilatation and dropping of the pyloric portion of the stomach. The duodenum was elongated and dilated, especially in its first part; it showed marked "writhing" contractions, with repeated regurgitation, only small amounts entering the jejunum in spite of the strong duodenal contractions. After twenty-four hours fully one-third of the bismuth was still contained in the pyloric portion of the stomach. (*See* also fig. 9c.) *C.*, *P.*, cardiac and pyloric portions of the stomach; *U.*, umbilicus; *Py.*, pylorus; *a*, *b*, *c*, first, second and third parts of the duodenum. The arrows indicate the persistent pyloric spasm, simulating organic stenosis.

CHRONIC ULCER OF THE LESSER CURVATURE.

These chronic ulcers of the lesser curvature form a most fascinating study; as shown on the couch the picture is very characteristic (figs. 2, 3, 4, 5, 6, 7, 8, and 9B); the depressed base of the ulcer occupies some point of the lesser curvature; it is usually adherent to the tail of the pancreas—sometimes to the left lobe of the liver. The raised, crater-like margins of the ulcer are well seen, but the most striking feature is the firm spasmodic contraction of the circular fibres of the stomach over the area of the ulcer, giving rise to a tight hour-glass constriction, with a narrow isthmus joining the cardiac to the pyloric portion of the stomach. This constriction is permanent, except under an anæsthetic. This is a fact of the greatest importance to surgeons, for at operations they see the stomach fully open, with little or no hour-glass appearance,

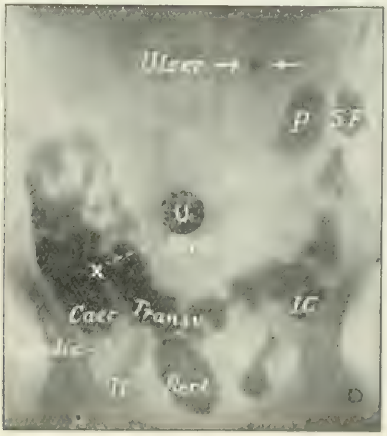

FIG. 9C.

Taken on the couch forty-seven hours after the same bismuth meal, showing that there is still bismuth in the pyloric portion of the stomach and in the depressed base of the ulcer. There was great stasis in the large intestine, and at the end of seventy-two hours most of the bismuth was still contained in the cæcum, ascending colon, and dropped transverse colon. The appendix was shown to be normal. At the operation there was found to be a chronic ulcer of the lesser curvature, adherent, and causing only slight organic constriction. The pylorus was tied by bands to the under surface of the liver, but there was no pyloric stenosis. The duodenum was prominent. There was a well-marked ileal kink, holding down the terminal coil of the ileum (*Il.*) in the right iliac fossa. Mr. Lane "short-circuited" the ileum into the upper part of the rectum, leaving the stomach untouched. The yellow skin and the cold, unpleasant sweat cleared up within six weeks of the operation, and the hands became warm and dry. The chronic mastitis subsided, and she felt much better. Six months later she was well and was working as a maid. She had suffered from severe dyspepsia and constipation for many years. For six months she had great pain, especially after food, and vomiting. Never hæmatemesis. She had lost weight for one year. *P.*, pyloric portion of the stomach; *S.F.*, splenic flexure; *Cæc.*, cæcum; *I.C.*, iliac colon; *U.*, umbilicus; *Transv.*, transverse colon; the X marks the ileo-cæcal entrance.

and they are very apt to overlook the fact that the constriction will return as soon as the effect of the anaesthetic has passed off. The surgeon may even select the seat of the ulcer for his gastro-enterostomy opening—with disastrous results. The form of the stomach in a typical case is remarkably constant; one would never have guessed that the pyloric portion of the stomach would present a sharp nightcap-like projection upwards at the seat of the ulcer (figs. 2, 3, 4 and 6), yet one has only to draw a diagram of the stomach with the ulcer, and represent on the drawing the effect of the abrupt shortening of the circular fibres

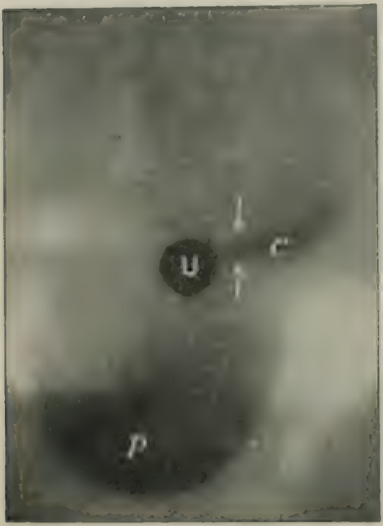

FIG. 10A.

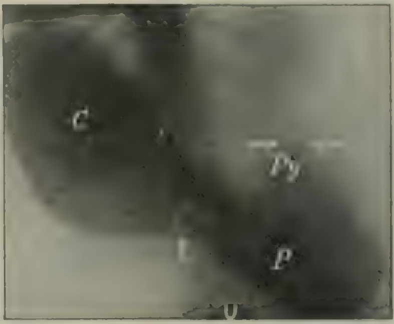

FIG. 10B.

FIG. 10A.—Taken vertically, showing the stomach of a woman, aged 30; there had been attacks of epigastric pain since the age of 14, coming on every few months and lasting four or five days. At the age of 20 the pain became continuous, and vomiting began. She was apt to vomit food eaten the previous day; never blood. The vertical examination revealed an hour-glass constriction; the pyloric portion of the stomach was greatly dilated and dropped. On the couch, reversed peristalsis was observed, strong waves passing from the pylorus back along the curvatures. After nine hours no bismuth had left the stomach. *C.*, *P.*, cardiac and pyloric portions of the stomach; *U.*, umbilicus. The arrows indicate the hour-glass constriction. (*See* also fig. 10C.)

FIG. 10B.—Taken prone immediately after fig. 10A, showing the hour-glass constriction, marked by an arrow, and the organic stenosis at the pylorus (*Py.*), marked by two arrows. *C.*, *P.*, cardiac and pyloric portions of the stomach; *U.*, umbilicus.

over the ulcer and nowhere else, to see how the particular form of the stomach arises (diagram A).

In most cases of chronic ulcer of the lesser curvature it is obvious, from the large size of the pyloric portion of the stomach, and from the low position of this portion when examined vertically, that gastric dilatation preceded the formation of the ulcer. This fits in with the explanation already given, for the pyloric spasm was there long before the ulcer appeared on the lesser curvature, and in addition the pylorus is often slung up to the under surface of the liver by bands which catch up the gall-bladder as well (fig. 9c). In quite a good proportion of cases there is also an ulcer at the pylorus (fig. 3), a very chronic ulcer, perhaps cicatrizing. The explanation of this has already been given. I shall now show typical cases illustrating the X-ray appearances of gastric ulcer, and proving its origin from intestinal stasis (*see* descriptions of figs. 2 to 12).

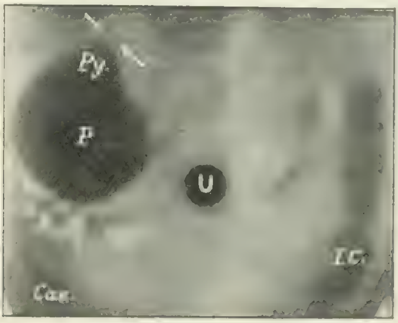

FIG. 10C.

Taken on the couch forty-eight hours after the same bismuth meal, showing the greater part of the bismuth still in the pyloric portion of the stomach. The terminal coil of the ileum contained traces of bismuth, and was hypertrophied, for it could be felt as a cord. After seventy-two hours there was still a large amount of bismuth in the stomach. At the operation Mr. James Sherren found a cicatrized ulcer of the lesser curvature, and a duodenal ulcer, just beyond the pylorus, with hard, thick edges, producing great stenosis. The terminal coil of the ileum was thick; the appendix was free, its tip obliterated by fibrous tissue. Mr. Sherren performed two gastro-jejunostomies, the loop of jejunum being united in turn with the pyloric and the cardiac sac of the stomach. The patient made an excellent recovery, and four months later was apparently cured.

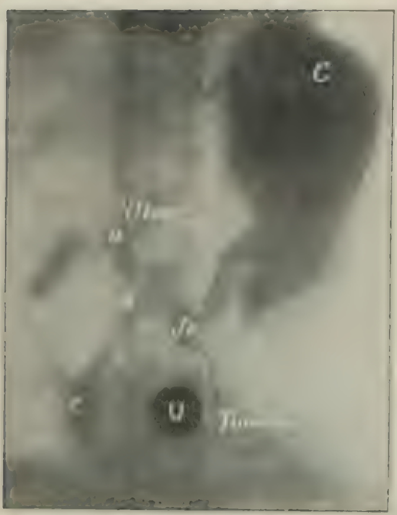

FIG. 11A.

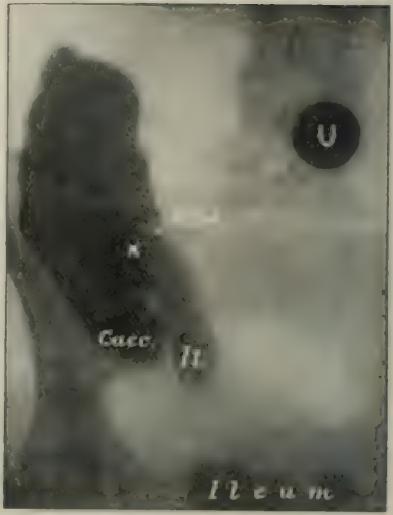

FIG. 11B.

FIG. 11A.—The stomach and duodenum of a woman, aged 45, taken on the couch after a bismuth meal (administered vertically as usual). There was a tumour felt in the left side of the abdomen; it was smooth and tender; the wire marks its lower border. For five years there had been pain coming on two hours after meals, and relieved by food and by alkalies. Pain came on also at 2 a.m. Six months ago she used to vomit; she was constipated and depressed. The X-ray examination showed, in a modified form, the characteristic appearance of a chronic ulcer of the lesser curvature, causing a well-marked hour-glass constriction. The tumour was connected with this part of the stomach. The pylorus was normal, the duodenum elongated. *C.*, cardiac portion of the stomach; *a*, *b*, *c*, *d*, the four parts of the duodenum; *Je.*, jejunum; *U.*, umbilicus. (See also fig. 11B.)

Fig. 11B.—Taken on the couch nine hours after the same bismuth meal, showing an ileal kink near the ileo-cæcal entrance, and the terminal coil of the ileum somewhat dilated. After twenty-six hours most of the bismuth was contained in the cæcum, ascending and transverse colon, the latter being much dropped. The appendix appeared normal. *Cæc.*, cæcum; *Il.*, terminal coil of the ileum; *U.*, umbilicus. The X marks the ileo-cæcal entrance. At the subsequent operation Mr. D. J. Armour found a chronic ulcer of the lesser curvature surrounded by much hard inflammatory tissue. He performed a posterior gastro-jejunostomy above the mass, with a good result. An enlarged gland was removed for examination, and proved to show inflammatory changes only, but five months later it became evident that there was malignant involvement.

TERMINATIONS.

What is the fate of these chronic ulcers of the lesser curvature? Many of them persist for years, often unrecognized, though the X-ray examination will "change all that" in the near future. A few cicatrize completely, leaving a scar with a varying amount of hour-glass constriction (figs. 10 and 12). This may be so tight that no bismuth can be

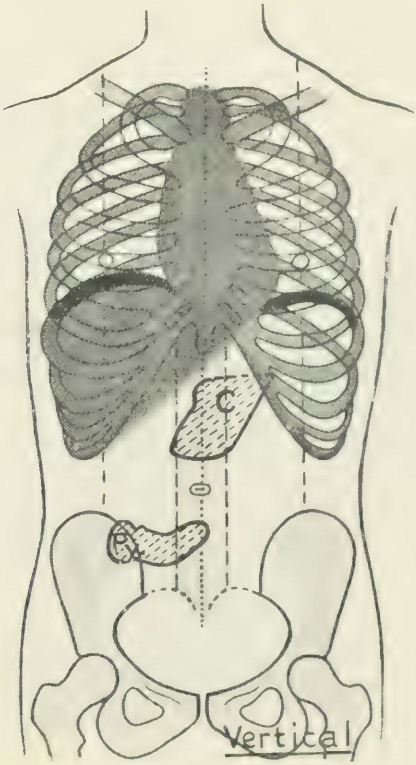

FIG. 12.

Taken vertically ten minutes after a bismuth meal (a smooth emulsion which the patient drank) in a woman, aged 39, showing the main bulk of the bismuth in the cardiac portion of the stomach (*C.*), and a small collection in the pyloric portion, which is dropped, but shows normal peristalsis and a normal pylorus (*Py.*), except for spasm. When first examined in the upright posture no bismuth entered the pyloric portion, and on subsequent examinations the whole of the bismuth remained in the cardiac portion of the stomach, no bismuth being ever again seen in the pyloric portion. At the operation a very tight fibrous hour-glass constriction was found.

seen to pass through the constriction. In the case of a woman, aged 39, who was sent to me in the first instance as an out-patient, I suspected, from the appearance of the stomach shadow with a bismuth emulsion, that I was observing only the cardiac portion of an hour-glass stomach, although not a trace could be seen to pass out even with the smallest diaphragm. Accordingly I kept her sitting in a chair for ten minutes and examined her again. This time I was rewarded by finding a small pool of bismuth below the level of the iliac crests, showing normal gastric peristalsis and a normal pylorus, though there was some spasm there (fig. 12). The bulk of the bismuth was still contained in the cardiac pouch. This patient was admitted to the hospital next week, and sent down to me again for confirmation of the diagnosis, but although I examined her several times, at different intervals after taking the bismuth (half an hour, two hours, several hours), I was

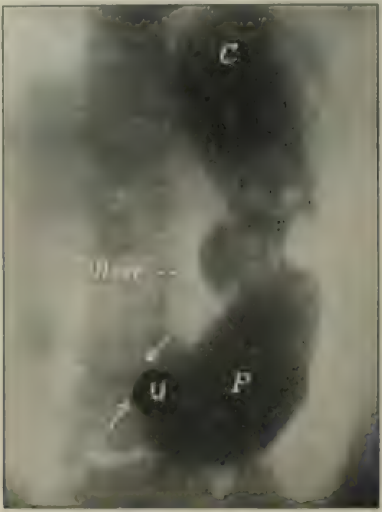

FIG. 13.

Stomach of a woman, aged 46, taken on the couch after a bismuth meal, showing the typical characters of a chronic ulcer of the lesser curvature: the depressed base of the ulcer, the raised margins, the dilated pyloric portion, with spasm at the pylorus. The sole distinction is in the lighter areas in the neighbourhood of the ulcer, found subsequently to be due to nodules of cancer. In this case the actual transition from a simple chronic ulcer to a cancer is illustrated. (See also figs. 14A and 14B.) *C.*, *P.*, cardiac and pyloric portions of the stomach; *U.*, umbilicus. The arrows indicate the pyloric spasm.

never again able to show any bismuth in the pyloric portion of the stomach. The first examination was so definite, however, that I adhered to the diagnosis then made, although the clinical examination did not seem to fit in with it. Mr. Charters Symonds operated, and found a very tight cicatricial hour-glass constriction, for which he performed the operation of gastro-gastrostomy with good result.

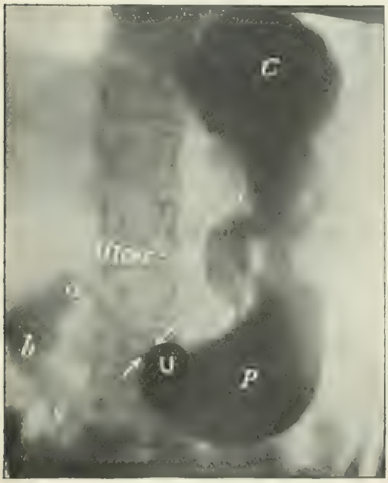

FIG. 14A.

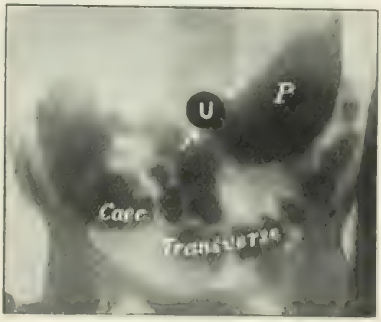

FIG. 14B.

Fig. 14A.—Another view of the same stomach taken a week later, showing the same characters, and indicating, in addition, the dilated duodenum. *C.*, *P.*, cardiac and pyloric portions of the stomach ; *U.*, umbilicus ; *a*, *b*, *c*, first, second and third parts of the duodenum. The arrows indicate the pyloric spasm. There was constipation, and abdominal pain an hour after meals. (*See* also fig. 14B.)

Fig. 14B.—Taken twenty-four hours after the same bismuth meal, showing the greater part of the bismuth still in the pyloric portion of the stomach, and no bismuth beyond the splenic flexure. The transverse colon is greatly dropped. After seventy-two hours there was still a large amount of bismuth in the pyloric portion of the stomach (*P.*). The arrows indicate the spasm at the pylorus. At the operation a malignant growth of the stomach was found, infiltrating the omentum, with secondary glands in the portal fissure. There was no pyloric stenosis, the permanent spasm having passed off under the general anæsthetic. *Cæc.*, cæcum ; *U.*, umbilicus.

CANCER.

A good many chronic gastric ulcers meet with a different fate; they become cancerous. This is a fact which has often been denied.

but radiography affords positive proof of its accuracy (figs. 11 and 13 to 18). Early cases of cancer often show the typical appearance of a chronic gastric ulcer, with only slight alteration due to the malignant involvement (figs. 11, 13 and 14). Even fairly advanced cases, where there is an obvious tumour to be felt, sometimes show the typical appearance quite clearly, though with a varying amount of modification

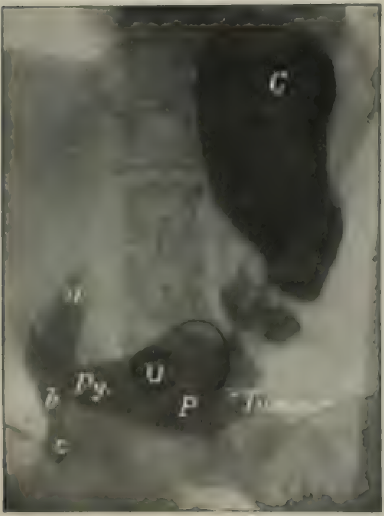

FIG. 15.

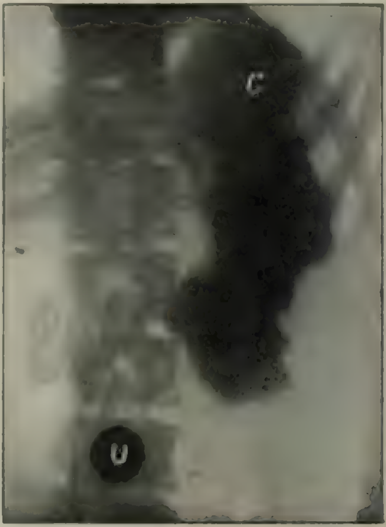

FIG. 16.

Fig. 15.—The stomach and duodenum of a woman, aged 60, taken on the couch after a bismuth meal. Epigastric pain after food for two years; worse the last six weeks. Constipation; great wasting; no vomiting. The X ray appearances are those of chronic ulcer in caricature. The wire marks the position in which a tumour was felt. The duodenum was dilated, and showed well-marked "writhing" contractions. The origin of the cancer in a chronic ulcer of the lesser curvature is still evident, though the deviation from the typical form is greater than in the case of figs. 13 and 14. The duodenum supplies evidence of intestinal stasis. The aortic arch showed considerable atheromatous elongation, and slight fusiform dilatation. *C.*, *P.*, cardiac and pyloric portions of the stomach; *Py.*, pylorus; *a*, *b*, *c*, first, second and third parts of the duodenum; *U.*, umbilicus.

Fig. 16.—The stomach of a man, aged 37, taken on the couch after a bismuth meal. For two years there had been pain half an hour after food; blood was vomited two years ago. Vomiting for the last five months; wasting. The picture shows great irregularity of outline of the stomach with stenosis of the pyloric portion, only traces of bismuth trickling through. Even here the appearance of the lesser curvature suggests dimly the origin of the cancer in a chronic ulcer, and the history adds probability to this supposition. *C.*, cardiac portion of the stomach; *a*, *b*, traces of bismuth in the duodenum; *U.*, umbilicus.

(figs. 15 to 18). In such cases (with a palpable tumour) the origin from a chronic ulcer is not nearly so obvious on direct inspection (at an operation or at a post-mortem) as it is in the X-ray picture. Sometimes the skiagram shows a veritable caricature of the picture of a chronic ulcer (fig. 15). Another point—a very important one: all the patients exhibit the characters of intestinal stasis, and the distended duodenum can usually be shown on the same picture as the stomach (figs. 11A and 11B, 14A and 14B, 15 and 17).

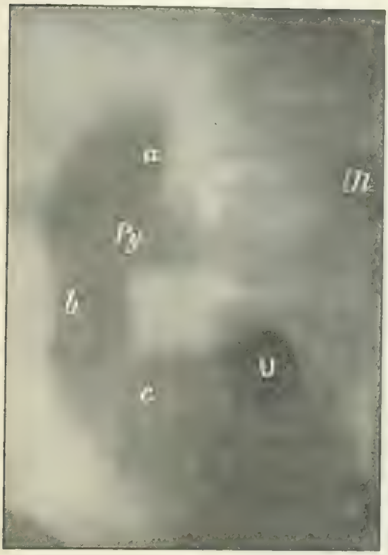

FIG. 17A.

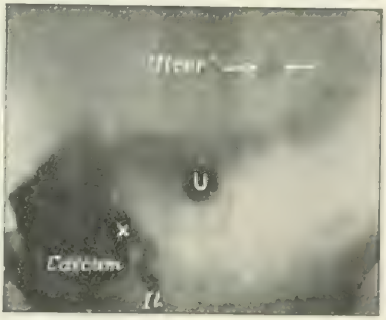

FIG. 17B.

Fig. 17A.—Gastric "ulcer," pylorus and duodenum in a man, aged 45, taken on the couch after a bismuth meal. He had pain coming on after food, and relieved by eating; he had lost weight during the last six months; constipation, which had become extreme the last four months. The X-ray examination showed an hour-glass constriction, with a pouch in the position of the lesser curvature (*Ul.*). The pylorus was normal; the duodenum was greatly distended; its first part much dilated. *Py.*, pylorus; *a*, *b*, *c*, first, second and third parts of the duodenum; *U.*, umbilicus. (*See* also figs. 17B and 17C.)

Fig. 17B.—Taken on the couch eight hours after the same bismuth meal. The base of the "ulcer" still contained bismuth, and was very tender to pressure. There was a trace of bismuth in the terminal coil of the ileum; the remainder occupied the large intestine. The appendix was shown to be normal. *Il.*, terminal coil of the ileum; *U.*, umbilicus. The **X** marks the ileo-cæcal entrance. (*See* also figs. 17A and 17C.)

Thus we have a further illustration of the importance of intestinal stasis in the ætiology of malignant disease. When stasis is recognized by medical practitioners in its early stages, and rectified, there will be far less cancer, not only of the stomach and large intestines but also of the bile-ducts and pancreas, and of the breasts. All must agree, therefore, as to the immense importance of recognizing the primary disease early, and of subjecting the patients to effective treatment. Few cases would call for operation if this were done.

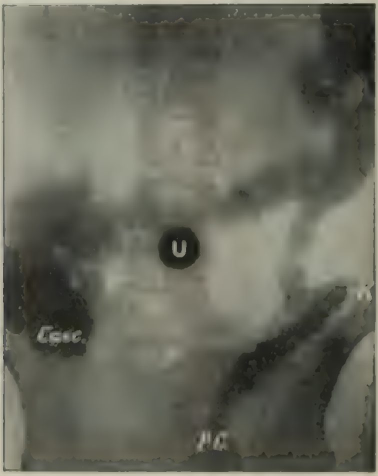

FIG. 17C.

Taken on the couch twenty-five hours after the same bismuth meal, showing all parts of the large intestine, and a sharp kink of the iliac colon just below the left iliac crest, the bowel being firmly fixed to the iliac fossa at this point (*K.*). At the end of thirty-six hours the pelvic colon was shown to form a long loop, rising almost to the level of the umbilicus. All parts of the large intestine still contained bismuth at the end of forty-eight hours. *Cæc.*, cæcum; *P.C.*, pelvic colon; *U.*, umbilicus. At the operation there was found to be a malignant growth of the lesser curvature with an extensive mass behind the stomach, extending down to, and below the great curvature, which was involved. There was much growth in the transverse meso-colon, and nodules throughout the great omentum and elsewhere. The duodenum was distended; its first part much dilated. The kink of the iliac colon was very tight.

EARLY DIAGNOSIS.

In the early diagnosis of the changes occurring in intestinal stasis the radiologist takes a most important share; he must work out his method with a view to demonstrating these changes in their earliest stages, before any definite symptoms have appeared that can be put down directly to their influence. I am frequently able to demonstrate an ileal kink in its very beginning, when it consists of nothing more than a slight local thickening in the mesentery of the last inch or two of the ileum. It is far easier to recognize an extreme kink with permanent obstruction. In its slight degrees there is no obstruction; only moderate difficulty in the upright posture, accentuated when the cæcum is overloaded and the patient fatigued from prolonged exertion. Even the early stages of ileal kink produce their inevitable reaction on the duodenum, which becomes distended, and secondarily on the stomach in the manner I have explained already.

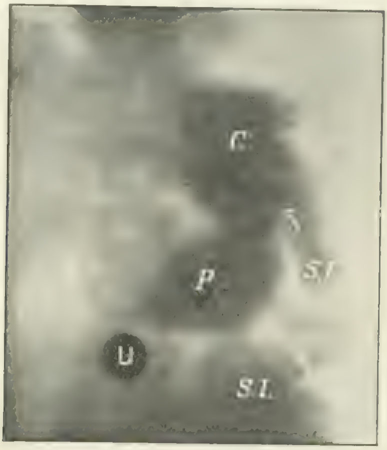

FIG. 18.

Taken on the couch after a bismuth meal in a man, aged 58, suffering from pain and vomiting for three months, the vomit containing fæcal matters and no free hydrochloric acid. There is an hour-glass constriction, the form of which suggests its origin in a chronic ulcer of the lesser curvature. Above the constriction is a spontaneous communication with a coil of small intestine, and secondarily with the large intestine in the region of the splenic flexure. At the post-mortem examination an extensive cancerous growth was found, the stomach being evidently the seat of the primary lesion. *C.*, *P.*, cardiac and pyloric portions of the stomach; *S.I.*, small intestine; *U.*, umbilicus. The arrow points to the spontaneous communication between the small intestine and the stomach.

It is a significant fact that many of the patients who prove to have a chronic ulcer of the lesser curvature give, clinically, the classical symptoms of duodenal ulcer; pain coming on two or three hours after food and relieved by eating; in some cases pain coming on at 2 a.m.

also *see* fig. 11A). No doubt the duodenal distension with congestion causes these symptoms. Eating gives rise to pyloric spasm, and so relieves the tension in the duodenum. As the pyloric spasm subsides, allowing the gastric contents to pass through, the distension of the duodenum again becomes greater, and the pain returns.

IMPORTANCE OF A COMPLETE INVESTIGATION.

Lastly, I would repeat that it is of the first moment that the radiologist should make a complete investigation of the gastro-intestinal tract in all cases of gastric ulcer or of pyloric stenosis. It is no longer sufficient for him to diagnose the gastric disorder, which is, after all, merely an end result; he must bring all the resources of his art to the elucidation of the primary cause of the gastric disorder, and to a large extent he will be successful.

The Ætiology of Gastric Ulcer.

By A. E. BARCLAY, M.D.

PERHAPS the title of my paper should have been "The Radiographic Indications of the Ætiology of Gastric Ulcer," for although one also attempts to study the clinical, pathological and surgical sides of each case, that aspect with which we are most in touch will necessarily assume an importance which may not be shared by those who see the same conditions from a different point of view. I am fully conscious of the fact that radiology does not show microscopic details, but on the other hand, I claim that it does show gross changes of great functional significance that cannot be detected by any other means. These appear to me of such paramount importance that I have ventured to give to my paper a title that is perhaps too ambitious, especially when one considers the extensive literature that there is on the subject.

I would like to clear the ground on one point. The radiologist examines cases of chronic indigestion; he is seldom, if ever, called upon to examine those cases of ulceration that arise in the course of acute infective processes and other conditions, and it is only when these lead to chronic indigestion that he has the opportunity of examining the patient. Although these cases eventually lead to chronic gastric ulcer under certain conditions, they are not, I believe, primarily of the same nature as the ordinary gastric ulcer of which I wish to speak to-night, that, so to speak, just comes by itself, of gradual onset following attacks of indigestion in most cases, giving rise to pain at varying times after food, usually to vomiting and sometimes to hæmatemesis. The ætiology of gastric ulcer is obscure and the number of theories is ample proof that there is as yet no completely satisfactory explanation.

The X-ray method of examination is the only way in which we can obtain information as to the action of the stomach while it performs its functions, for, as we now know, the stomach, as seen in the dissecting room and on the operating table, may be a gross caricature of the organ as it fulfils its functions. After death the vital attribute of tonic action has disappeared, while on the operating table the tone, and therefore the shape of the stomach, are profoundly altered by the anæsthetic. The factors of fear of the anæsthetic, nausea, and vomiting cause such extraordinary changes that those who have had experience will seldom commit themselves as to the size of the stomach that the surgeon will reveal. Those who watch the continuous movement of the stomach during life will hardly recognize the occasional halting and uncertain contractions sometimes seen on the operating table as peristaltic waves.

The X-ray method is the only feasible means of studying the pathology of the living, functioning alimentary tract, and the following observations seem to give very definite guidance towards the solution of the problem of the cause or causes of gastric ulcer.

It frequently happens that an investigation is undertaken with the object of proving or disproving some theory. In this instance I had no theory and no thought of setting myself the task of solving a problem that has foiled pathologists. The theory evolved itself, it forced itself out from the mass of accumulated evidence and called to be set down in black and white. It is impossible to put down the stages by which one arrived at one's conclusions, for they came almost unconsciously, and not step by step, as usually occurs in such investigations.

To give point to the reading of the observations, it is necessary to give an outline of the conclusions arrived at, and briefly stated, the hypothesis is that septic conditions in the mouth or other sources of swallowed septic matter, constipation, mucous colitis, and a variety of other conditions, are capable of producing spasmodic contractions of various parts of the stomach. The spasm may be so severe that it produces a narrowing of the lumen which from time to time is of great functional importance, causing a definite obstruction to the passage of food. By some indiscretion of diet or want of mastication, something too large to pass easily through the channel has to be forced through by powerful peristalsis. This produces bruising or possibly an abrasion at the point where the lumen is narrowed, and where there is the constant irritation of food passing over it. It is probable also that the spasm interferes with the blood supply. Moreover, a surface is exposed that is not structurally fitted to withstand the action of the gastric juice. In this way an ulcer is formed which perpetuates the original spasm that determined the site of the ulcer. The ulcer actually becomes the cause of the spasm and prevents healing, so that even if the original cause of the spasm is removed there may be little if any improvement in the local condition. The chronic ulcer is yet one more example of the effects of a vicious circle.

The theory that spasm is associated with the ætiology of gastric ulcer is not new; for instance, in 1853 Virchow,[1] followed by Key,[2] initiated the idea that spasm or portal stasis might cause hæmorrhagic infiltrations. Gerhardt[3] held that erosions might be due to crampy contractions, but that these very seldom gave rise to chronic gastric ulcer. My time is limited, but I cannot pass on without acknowledging the splendid work that Dr. Charles Bolton has done on this subject.[4]

The multiplicity and varied nature of the observations, original and otherwise, that I have to record makes it imperative that I should cut these down to the narrowest limits in order to keep within my allotted time, and I crave your indulgence if by so doing I fail to make my points as clear as I would wish, and also for the apparent lack of continuity.

I may say that this paper arose out of a thesis in which I tabulated nearly 300 cases with the radiographic appearances and operative findings.[5]

(1) The stomach is very sensitive, and many cases of marked

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[1] Virchow's *Arch. f. Path. Anat. u. Phys.*, Berl., 1853, v, p. 363.

[2] *Hygeia*, Stockholm, 1870, p. 261.

[3] Virchow's *Arch. f. Path. Anat. u. Phys.*, Berl., 1892, cxxiv, p. 85.

[4] Bolton, *Brit. Med. Journ.*, 1910, i, pp. 321, 1221, 1382, 1444; *Proc. Roy. Soc. Lond.*, 1904-05, lxxiv, pp. 135-47; *Quart. Journ. Med.*, Oxf., 1912, v, pp. 429-62.

[5] "The Stomach and Œsophagus—a Radiographic Study," Sherratt and Hughes, Lond. and Manch., 1913.

spasmodic contraction have been met with. Quite a large number of spasmodic hour-glass stomachs have been examined and found at operation to show no trace of ulceration or other abnormality that, by local action, would cause the spasm, and yet, radiographically, they gave all the appearances seen in organic hour-glass contractions. These spasmodic contractions have been met with in the body of the stomach and also at the pylorus. On several occasions I have seen that the vomiting took place from the upper sac of a spasmodic hour-glass contraction, and as soon as the food passed down the tendency to vomit and the pain ceased. The contractions are often present one day, absent the next; they are usually relaxed by massage or manipulation. There is no pain over the site of the spasm on deep palpation. Hertz and I both believed at one time that peristalsis does not occur in the upper sac of a pure spasmodic hour-glass, but this is not so, as my partner, Dr. Bythell, and I now have complete proof to the contrary.

(2) Identical contractions have been met with, in fact, are almost invariably met with, in association with even quite small ulcers; and I have always believed that the severity of the spasm resulting from an ulcer was dependent on its irritability and not on the size of the ulcer, since small ulcers gave just as marked contractions as the large florid ulcerations, indeed in some cases where only a small ulcer was found the food failed to get into the lower sac for hours.[1]

(3) I have seen pyloric obstruction, presumably functional—as proved by seeing the greater part of the food still in the stomach after twenty-four hours—permanently *cured* by removing bad teeth. In another case, an out-patient with many decayed stumps, practically the whole of the food was still present in the stomach after twenty-four hours. On admission he was made to use a tooth-brush, and when I examined him a few days later practically all the food had passed out of the stomach in five hours. He was a fairly intelligent man and had tried careful dieting before he was admitted to the hospital, with no effect.

(4) In two cases I have been so much impressed by the irritable appearance of the stomach that, in spite of fairly well marked clinical evidence, I reported that I thought the symptoms were secondary, the condition of the teeth being, in my opinion, sufficient to cause the

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[1] A slide was shown of the bismuth food still in the upper sac of an hour-glass stomach after twenty-four hours. This was a rather acute case, and at the operation there was complete absence of cicatrization, only an active ulcer of the greater curvature; not a trace of the food appeared to have passed or.

radiographic appearances noted: this in spite of the fact that some retention of food was noted on one occasion. Clinically, the cases were quite definite, and the surgeon who operated found no evidence of gastric or other intra-abdominal lesion of any kind, not even an adhesion of the appendix.

An irritable condition of the stomach is also frequently noted when there is severe constipation. This is evidenced by the hypertonicity and the active peristalsis, which is of a rather unusual type, as many as four or five waves being noted on the greater curvature at the same time.

(5) Spasmodic contractions, forming hour-glass stomachs, have been noted as being not so marked or entirely absent after the bowels have been moved in cases of severe constipation. The following case is perhaps instructive: There was a very marked hour-glass condition which I thought was due to an ulcer of the body of the stomach, in association with pyloric obstruction. Nothing was found to account for the hour-glass contraction, but the pylorus was thickened and therefore a gastro-enterostomy was performed. I expected that this patient would not be cured, but a year later he writes: "I have put on 2 st. in weight and can eat everything. I was always very costive, in fact, I never had a motion without opening medicine, but have never had a dose since the operation." In the light of other cases it looks as if the operation had not only relieved the pyloric obstruction but also the constipation, and that this latter had been the cause of the spasmodic hour-glass contraction.

(6) In taking brief clinical histories of approximately 1,400 cases I have been impressed with the frequency of a history of constipation, and also by the presence of bad teeth or a history of having had teeth removed after the onset of the symptoms.

(7) The following observations indicate the transition from an apparently spasmodic condition to a definite organic lesion.

In fully a half of the cases of ulceration of the body of the stomach that I tabulated there was also evidence of retention of food behind the pylorus—i.e., pyloric obstruction—and in the large majority of these latter there was actual thickening of the pylorus. In two cases in which no thickening of the pylorus could be detected at the operation, a gastro-jejunostomy to the upper sac failed to cure, and a subsequent examination showed that a portion of the food had passed into the lower sac and was retained there for twenty-four hours or more. A further operation showed well-marked thickening of the pylorus, and excision of the lower sac cured the patient.

(8) I have just said that in fully a half of the cases of ulceration of the body of the stomach there was also evidence of pyloric obstruction. It is a fact also that in a large percentage of the pyloric obstruction cases there was also noted at the radiographic examination a more or less marked spasm of the body of the stomach. Indeed, the relation between spasm or even ulceration of the body of the stomach and pyloric obstruction is so marked that it can hardly be accidental. It appeared as if lesions in these two sites were dependent on some common cause rather than on one another.

(9) Moynihan put into words a belief that had gradually taken shape in my own mind. "The diseases of the stomach, duodenum, and gall-bladder, with which the surgeon deals are not primary, but secondary."[1] I had been much impressed by what I called the interdependence of abdominal conditions—e.g., when one found an irritative condition of the large bowel, e.g., mucous colitis—one usually found the appearances, and often also the symptoms, of duodenal ulcer, and in one case there was a gastric ulcer with hour-glass contraction. The duodenal appearances and symptoms were also noted in several cases of lesions of the small intestine (carcinoma, tubercular ulcer, and adhesions) in which the actual site of the disease was indicated to the surgeon solely by the X-ray examination. In one of these lesions of the small intestine a typical ulcer of the duodenum was also found, while in another patient, who died a few days after the operation, the mucous membrane was injected and inflamed, although there was no evidence of this on the peritoneal surface (*see* figs. 1 and 2).

(10) The excessive and rapid formation of gastric secretion has been noted in all the later cases in which a pyloric ulcer has been found, except those in which marked retention of food was also present, this feature making it impossible to detect the hypersecretion. Well-marked hypersecretion has been noted in cases where there was slight delay in emptying (six hours), and under medical treatment not only has the delay in emptying disappeared, but also no hypersecretion could afterwards be detected. The hypersecretion seems to be the accompaniment of pyloric and possibly duodenal lesions, and disappears with the pyloric "irritation." I have not yet seen hypersecretion in association with an ulcer of the body of the stomach unless there was an actual lesion of the pylorus also present. In one instance I found what was evidently the upper sac of an hour-glass contraction of cicatricial type

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[1] *Lancet*, 1912, i, p. 10.

(fig. 3), and during the half-hour I had the patient under observation (the surgeon was waiting to operate) only a very small quantity of food found its way through into the lower sac, but there was profuse secretion into the upper sac (fig. 4), and, as suspected, this indicated an active ulcer of the pylorus. At the operation there was only a very narrow channel connecting the upper and lower sacs, so that the secretion must have been poured out from the cardiac end, strongly suggesting that the hypersecretion of pyloric ulcer is a general secretory activity, probably reflex.

(11) When there is appendix dyspepsia the radiographic appearances are usually those of duodenal irritation, more rarely there is an hour-glass contraction; and it is a striking fact that in operations for duodenal and gastric ulcer there is so very frequently evidence of old appendix trouble.

(12) Gastric symptoms—the old typical symptom-complex of gastric ulcer—have been cured by short-circuiting the large intestine in a case of severe constipation.[1]

(13) My personal experience of oral sepsis is of interest. Occasionally when I am below par I have acid risings into my mouth, probably indicating an excessive formation of gastric juice, such as I have frequently noted in marked cases, in association with pyloric ulcer, with "pyloric irritation" in the less marked cases. On two occasions when I have been conscious of retention of food for a prolonged period—and once I actually brought up a mouthful that contained fragments taken on the previous day—a more thorough and persistent use of an antiseptic mouthwash relieved this unpleasant symptom within the course of a couple of days. Occasionally I am also troubled with hunger pain. I am certain I do not suffer from any gastric lesion, for I have perfect health in the ordinary course of events, and do not know what indigestion is. This experience is by no means uncommon I find; slight loss of appetite, sense of weight in the stomach for hours after food, burning sensations, acid risings and waterbrash, that are quickly relieved as a rule by sodium bicarbonate. In nearly all these cases it is the teeth that are at fault. A doctor was telling me of this trouble a few days ago, and I inquired as to his teeth and found that he had had false teeth for rather more than a year and, when he came to think of it, since his teeth were removed he had not needed a single dose of his sodium bicarbonate bottle, which he always kept on his dressing-table, and used to resort to frequently.

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[1] *Lancet*, 1913, i, p. 418.

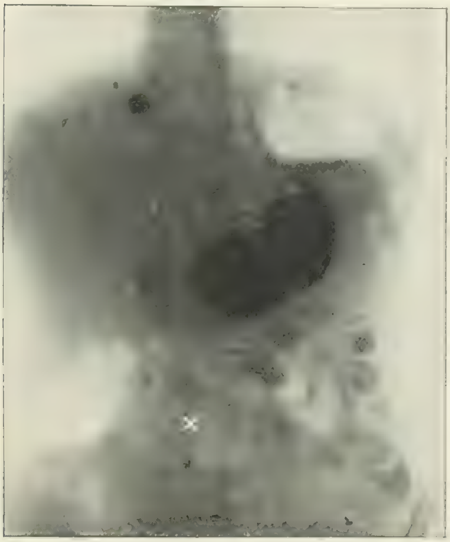

FIG. 1.

Small and very hypertonic stomach, with active peristalsis, emptying rapidly, and food shadow seen in duodenum. (Typical marked duodenal irritation picture). Note the food shadows seen beginning to collect in the small intestine. Plate taken ten minutes after the food was given. X is the umbilicus.

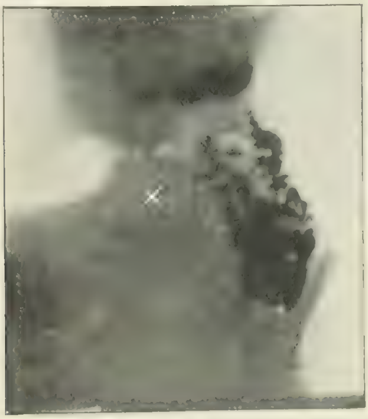

FIG. 2.

Same case as fig. 1, but twenty minutes later. Stomach nearly empty and all the food collected in the jejunum. This turned out to be an early carcinoma of the jejunum, and the post-mortem showed an infected and inflamed duodenum but no actual ulcer. X, umbilicus.

(14) The experimental production of gastric ulcer in animals shows that these always tend to heal very rapidly, and a typical chronic ulcer has not yet, I believe, been produced, except by causing stasis or local thrombosis.

(15) I have seen no case in which ulceration has taken place at the site of a gastro-enterostomy opening, and yet at this point the mucous membrane is comparatively roughly joined, and one would certainly expect that if ulceration was caused by the action of the gastric juice this would be a very common sequence to the operation, especially when we remember that in most of the operations a clamp is used that must produce a certain amount of bruising, also that in a certain proportion of cases the operation fails because of the formation of adhesions beyond the stoma which cause obstruction. This is certainly not the case in the hospital with which I am connected, and it is inconceivable that the mucous membrane is brought into perfect apposition in every case.[1]

(16) In the duodenum the conditions do not appear to be exactly the same as in the stomach. Dr. Hertz's observations and my own show that in every case of duodenal ulcer, also where there is duodenal irritation, the stomach begins to empty itself very rapidly unless there is obstruction, and one can easily see quite large shadows pass through the duodenum. In some cases there is a separate bolus persistently present in some part, as if a pocket was formed; but in spite of large quantities passing through I have very seldom observed obstruction, and whenever this has been seen there has been more or less cicatrization or some external cause found at the operation to account for the retention. It seems, therefore, as if spasm, as seen in the stomach, is not associated with duodenal ulceration in the same way, and I think it likely that some other influence is at work in these cases. The conditions that obtain in the stomach and duodenum during life are as yet only partly understood, and the various factors that control the passage of the food through the pylorus are by no means clear. Before the Anatomical Society I gave my reasons for believing that the control of the pylorus was influenced by the duodenal condition. In all cases of duodenal ulcer and of duodenal irritation one sees very abnormal pyloric relaxation resulting in the passage of large masses of food, whereas in

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[1] Neither the pathologist nor any of the surgeons at the Manchester Royal Infirmary have seen a single case of ulcer occurring at the site of the stoma (gastro-jejunal ulcer), nor have they seen a jejunal ulcer. The only case of ulceration occurring in this region was an acute ulcerative process that almost separated the jejunum from the stomach, but this was four years after the operation had been performed.

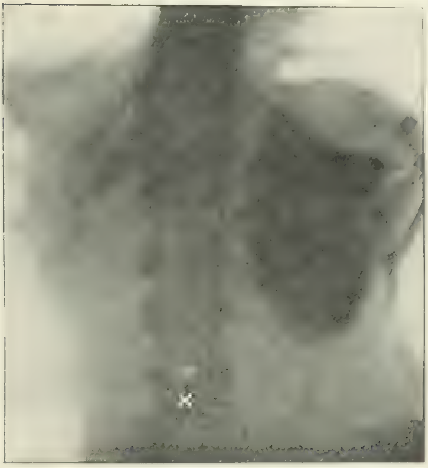

FIG. 3.

Upper sac of hour-glass stomach. Plate taken five minutes after the bismuth food had been given. Note the thin clear fluid between the air-space and the bismuth food—this was all secretion poured out on top of the bismuth food, the upper sac having been quite empty before the food was given. **X**, umbilicus.

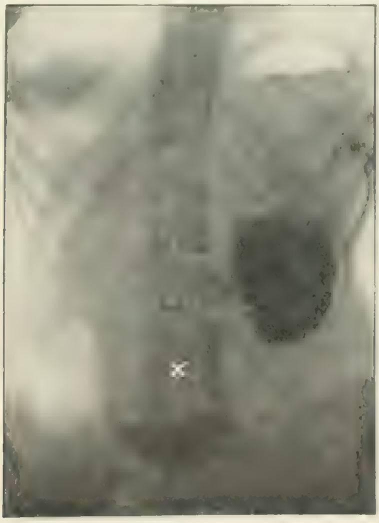

FIG. 4.

Same case as fig. 3, but twenty minutes later. Note the very large increase in the quantity of secretion that lies between the air-space and the bismuth. Below the umbilicus (**X**) some food can be seen in the lower sac.

the normal subject it is practically impossible to see the food passing through the duodenum, so fine is the stream that passes through the pylorus, and so rapid the segmentation and shredding of the food. The appearances suggest that the segmentation contractions that normally shred the food in the small intestine are absent in the duodenum when there is ulceration or irritation, otherwise the large shadows would not be seen in the duodenum. We know that the pylorus is abnormally relaxed in these cases, and one suspects that the relaxation extends to the duodenum and that the relaxation is sufficient to counterbalance whatever tendency towards spasmodic contraction the ulceration might give rise to. The absence of a spasmodic contraction in these cases would account for the fact that they so frequently heal spontaneously. The causation of these ulcers, however, does not appear to be the same as in gastric ulcer.

Taking all these observations together, I think that they become intelligible on the following hypothesis: The stomach and duodenum are, as it were, the storm centre for the alimentary tract to which peripheral stimuli are referred, the result being either a general irritable condition of the stomach and duodenum, or more frequently spasmodic contractions of one or more parts of the stomach. Presumably some of these stimuli are referred to the stomach and duodenum directly from swallowed septic matter. Others are referred indirectly from lower parts of the tract, as in the case of lesions of the small intestine, where we have the radiographic indications and also the operative proof of the occurrence of trouble in the duodenum in association with lesions situated in the jejunum or ileum. In fact, whenever I see marked duodenal irritation I am always on the look out for indications of a lesion in the small intestine. Again, in the mucous colitis, constipation and appendicitis cases we have both clinical and radiographic evidences of functional disturbances in the stomach or duodenum, and the subsequent history of the cases has shown that the patient could be cured and that these radiographic indications would disappear by treating a lesion that was apparently far removed.

Now these spasmodic contractions may give rise to all the radiographic appearances of actual lesions—on several occasions a surgeon has reopened the abdomen at my suggestion, because of the persistent hour-glass contraction that has been noted above the stoma of a gastro-enterostomy, which was functioning perfectly but had not relieved the symptoms. Three of these patients were actually observed vomiting from the upper sac of the hour-glass contraction, the pain and tendency to vomit disappearing when once the food had passed down into the lower sac, and yet no cause could be found at the operation to account for the contraction. In several cases where I have reported definite pyloric obstruction the surgeon could find nothing to indicate gastro-enterostomy, and in two of them a subsequent operation showed thickening of the pylorus. There are many cases of various types all pointing in no uncertain manner to the obvious conclusion that the spasmodic contraction is of as great importance, functionally, as the organic; and that these apparently purely spasmodic contractions are replaced by actual organic lesions has been noted in two cases in which a second operation has been undertaken, the pylorus being found quite normal at the first operation while subsequently well-marked thickening was noted.

That ulcerations of the alimentary tract are irritable and give rise to spasmodic contractions has been proved time and again—e.g., an ulcer of the œsophagus that only caused symptoms (and obstruction) after the patient took dry breadcrumbs. (The ulcer was actually seen through the œsophagoscope afterwards.) The same has been noted in the stomach on many occasions. Hour-glass contractions with retention of food in the upper sac have often been found, at operation, to be due to quite small ulcers with practically no cicatrization.

My thesis is that the original contraction causes undue obstruction to the passage of the food and that perhaps indiscretions of diet lead to bruising or even abrasions of the mucous membrane. The persistence of the spasm, aided by the action of the gastric juice and the mechanical irritation of the food, leads to the formation of an ulcer—the symptoms being probably more severe than usual during the attack. The raw ulcer is irritable and capable of reproducing or accentuating the spasm that originally caused the lesion. It follows that when once an ulcer is formed it will perpetuate the spasmodic contraction even if the primary cause of the spasm is removed. Hence it is clear that removing the primary cause will not cure the patient, for the ulcer when once formed continues to induce a spasm that tends to the perpetuation of the ulcer. If, on the other hand, an ulcer has not formed, the removal of the primary cause, whether it be teeth, nasal discharge, or swallowed pulmonary discharge, stagnation of fæces, appendix inflammation, or some other source of infection, will probably bring about a rapid and lasting cure of the patient.

To what extent the gastric juice is responsible I cannot say, but it seems reasonable to suppose that Dr. Bolton's excellent work is correct.

and that when once an abrasion is formed a surface is exposed that is not adapted to withstand the action of the gastric juice and will therefore tend towards the extension of the ulceration.

Since this conception of gastric ulcer came to me I have applied it as far as possible in radiographic practice, and in nearly every case found one of the various factors at work. Speaking very roughly, it is usually constipation in females in association with ulceration of the body of the stomach, while in males one more often found bad teeth in association with pyloric obstruction.

It is very highly probable that this theory does not cover the whole ground, and that there are many other causes than those I have mentioned e.g., infective ulcers that are said to be multiple. I mention this lest I should give the impression that I believe this theory covers the whole subject. I stated in my opening remarks it is the radiographic aspect on which this paper is based, and a theory such as this can only be proved by clinicians.

If we accept this theory, has it a practical bearing? Most emphatically, yes, and it applies even more to general practice than to consultation work, for the general practitioner sees the case in the early stages. In every case of indigestion a thorough search must be made for any possible source of irritation or infection both in the alimentary tract and in the other systems of the body. The teeth must be carefully examined and no decaying stumps left to pour their discharge direct into the stomach. Fixed bridges, that nearly always harbour putrefying foodstuffs, should be cleared out and replaced by a more sanitary system of artificial teeth. Disease of the accessory sinuses, constipation, colitis, and all possible sources of septic absorption, must be attended to. It is the *fons et origo* that we must endeavour to locate. The treatment of indigestion must be attacked at its source; and because we happen to relieve the patient by a dose of medicine that gets rid of the symptoms we must not be content, for unless the cause is removed there will be recurrence after recurrence, and we are all well acquainted with the result—the sad and depressed individual, of gloomy outlook, whose dietary is a problem and whose life is a burden.

My acknowledgments are due to the members of the staff of the Royal Infirmary of Manchester, to my partner, Dr. Bythell, and to many friends who, directly or indirectly, have given me assistance and encouragement.

DISCUSSION.

Mr. THURSTAN HOLLAND showed a number of slides bearing on the subject. He said it would be interesting to know whether intestinal stasis preceded the stomach symptoms, or vice versa; because he was sure that in some of the cases of which he had shown the skiagrams there was no stasis whatever. If stasis was the cause of the symptoms of gastric ulcer and the hour-glass contractions, why was it that simple gastro-enterostomy into the upper segment would frequently cure the patient? Every surgeon would admit having operated upon cases of hour-glass contraction—doing a posterior gastro-enterostomy into the upper segment and the patient had been cured. He felt sure that in many cases the pylorus was quite patent and normal. In some cases gastro-gastrostomy was done, and the patient gained in weight and got well, and nothing more was heard of him, though probably more would be heard if he did not remain well. Dr. Jordan seemed to think that cancer often occurred on top of old chronic gastric ulcers, but in a series of twenty-five cases when the latter condition had gone on to hour-glass contraction there had not been malignant disease in any one.

Dr. JORDAN said he was pleased to find there was not, after all, much essential difference between Dr. Barclay's results and his own. Such differences as existed were, as Dr. Barclay had suggested, due to differences in technique. He considered that radiologists should be able to report on the duodenum in a routine manner in all cases, describing it as normal when it was so, and reporting on all degrees and kinds of abnormality. There was now a great demand rapidly growing among physicians and surgeons, for trustworthy information concerning the duodenum in their patients, and it was for radiologists to supply that information. As already stated in his paper, he was able to distinguish with the greatest certainty between the normal duodenum and the distended duodenum as it occurred in the subjects of intestinal stasis. Sir Berkeley Moynihan, in the latest edition of his book, gave as his opinion that too much had been made of "supposed" dilatations of the duodenum. He regarded the duodenum as being normally "a large inert mixing chamber."[1] This was a significant admission, showing that Moynihan has come to regard the dilated duodenum as the normal, in consequence of the very large proportion of cases of duodenal disease included in his operative practice. As to Moynihan's opinion that the duodenum is inert, he would soon modify that view if he were to observe the duodenum, well filled with bismuth, as it appears on the fluorescent screen. Mr. Thurstan Holland's series of lantern slides was very interesting; they showed gastric ulcers as they appear in the vertical examination, and this accounted for the differences between Mr. Thurstan Holland's pictures and his own. The night-cap-like projection which he (the speaker) had pointed out was shown beautifully in Mr. Thurstan Holland's pictures, but filled with air instead of bismuth. As he had stated in his paper, the hour-glass constriction due to a chronic ulcer of the lesser curvature passed off completely under a general anæsthetic; if any constriction remained it was due to cicatrization (partial or complete) of the ulcer. Mr. Thurstan Holland had asked why patients with organic stenosis at the pylorus were cured by the operation of gastro-enterostomy. He replied that these patients had been suffering under a great disability, perhaps for years, for they had been deprived of intestinal digestion, since the food was unable to get out of the stomach. If a free opening were then made from the stomach into the small intestine the patient would begin to gain in nutrition at once, and he was the last to deny that the results of gastro-enterostomy were very good in cases of organic stenosis. His experience made him equally sure, however, that the cure was not complete; scarcely a week passed without some patient being sent to him for investigation on whom this operation had been performed with success. The patients returned because they presented all the signs and symptoms of intestinal stasis, and he was always able to give positive proof of the existence of stasis in these subjects. The fact was becoming recognized more and more widely that gastric and duodenal ulcers were end-results. Surely, then, the most important work for the future consisted, not in curing these ulcers by operation, but in preventing their occurrence by recognizing, sufficiently early, the primary condition to which they were due. The radiologist should detect the presence of intestinal stasis in its earliest stages, so that early measures might be taken to combat it. The duodenum was the most sensitive part of the alimentary tract, and its distension was one of the earliest and most constant signs of intestinal stasis.

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[1] "I believe that too much is made of these supposed (duodenal) dilatations. The duodenum is a mixing chamber wherein the chyme ejected through the pylorus is made to mingle with the bile and the pancreatic juice. The contractile power of the duodenum is, I believe, small, for though eager waves of contraction can be seen in the pyloric part of the stomach and in all parts of the jejunum and ileum, they are extremely rare in the duodenum. The duodenum is normally, in my experience, both large and inert." Moynihan, "Duodenal Ulcer," 2nd ed., 1912, p. 136.

Dr. BARCLAY said that he did not agree with Dr. Jordan's views on several vital points and he did not think it was likely they would come nearer agreement by arguing from the platform. He believed that Dr. Jordan did not have the patient's bowels cleared out and that they had an ordinary meal which was followed by a huge dose of bismuth. Then if the ordinary food was in the small intestine the bismuth food would be held back and there would be artificial distension of the duodenum. The same would occur at the ileo-cæcal valve if the patient's large intestine was not empty in front of the bismuth meal, for it was highly probable that the cæcum was filled simply by propulsion of food from the ileum. He confessed that he was rather tired of hearing of intestinal stasis due to kinks which he could not himself demonstrate except by omitting to clear out the bowels before the examination. In every case where he had seen the described appearance of kinking it was due to old appendix trouble or other adhesions. He thought that if in order to show these kinks it was necessary to have the large intestine filled by stagnant fæces (as must be the case in constipation when the patients were not prepared) the common sense deduction was that the cause of the trouble was beyond the alleged kink through which the stagnant food must have passed days before. In the hour-glass stomachs, although the contraction was always towards the lesser curvature, it was not by any means always that the base of the ulcer was on the lesser curvature as Dr. Jordan had suggested and demonstrated. The ulcer gave rise to a spasmodic contraction, a ring of contraction, and no matter where the ulcer was situated the organ always contracted towards the lesser curvature simply because this was the thicker and more fixed part. Of course, when adhesions occurred and the base of the ulcer became adherent this became the most fixed point, and he had seen a considerable number of cases in which the channel had been from the posterior wall and even from the greater curvature. He did not think that carcinoma of the body of the stomach was a frequent sequence to chronic gastric ulcer, as Dr. Jordan had suggested. He had recently tabulated a large number of cases[1] and found under ulceration of the body of the stomach that out of forty-seven cases thirty-nine were females, while in the cases of carcinoma of the body of the stomach only eight out of twenty-four were females, figures which he thought indicated that only a very small number of these cases took on malignant growth; otherwise his figures must surely show that carcinoma of the body of the stomach was far commoner in the female than in the male, whereas the direct opposite was the case. At the pylorus, however, carcinoma seemed to be of about equal frequency in the two sexes. He would like to talk over with Dr. Jordan the question of duodenal ulcer. He knew duodenal irritation when he saw it, also duodenal ulcer. Four years ago he started to diagnose the latter, and he had done so successfully in many instances, but he could not be certain of the diagnosis between irritation and ulceration. In one case where he had said the duodenum was perfectly normal, the operation had revealed grey patches on the duodenum, and in the very next case when a similar report was given the surgeon again said there was a duodenal ulcer present, but the patient died from hæmorrhage three days after the operation, and the hæmorrhage was apparently that of duodenal ulcer. No duodenal ulcer or remains of an ulcer were present and no cause could be found for the hæmorrhage, although there were plenty of patches on the duodenum. Even surgeons were not infallible, and radiographers had to check their findings by those of the surgeon. He certainly held no brief for the infallibility of the radiologist.

He agreed with Dr. Jordan that the stomach and duodenum were the most sensitive parts of the alimentary tract, but he did not think that intestinal stasis played such an all-important rôle in the ætiology of gastric ulcer. Until he knew more of the normal stagnation of fæces in the large intestine, of the pathology or more likely the physiology of various peritoneal folds and adhesions, he preferred to speak of constipation; for the contents of the large intestine were always in a state of "intestinal stasis," except for some few sudden on-rushes occurring a few times during the day.

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[1] "The Stomach and Œsophagus," Sherratt and Hughes, 1913.

Mr. THURSTAN HOLLAND, in further remarks, said, in reference to ileal kinks, and their demonstration, the technique was as follows: One must not open the bowels, by medicine or enemata, for at least two days before giving the bismuth meal, which meant that there was two days' food inside the person before the examination, and presumably it was in the ascending colon, transverse colon, and descending colon, because they were cases of chronic stasis. Dr. Jordan said that after this two days' filling up one must give them their breakfast, and then their bismuth food, and then the ileal kink could be demonstrated. Did not that mean that the food was prevented from getting into the ascending colon because it was full, and the bismuth food was dammed back in the last coils of the ileum? If the ileal kink was the cause of the symptoms, and the cause of the stasis, surely it should be demonstrable, whether the colon was or was not full.

Dr. JORDAN, in reply, said that all his patients were examined in the same way; if his results were due to his method, and did not represent pathological conditions, then all patients, normal as well as abnormal, would present these appearances, and his results would not be substantiated (as they were) at operations. His results were perfectly consistent, and he was able to distinguish, with the greatest certainty, between the normal duodenum and that seen in stasis cases, and between the normal end of the ileum and the same part as it occurred in stasis. An ileal kink was an anatomical condition, and could not be demonstrated with any certainty in patients whose bowels had been previously cleared by purges and enemata.

Electro-Therapeutical Section.

May 16, 1913.

Dr. REGINALD MORTON, President of the Section, in the Chair.

Case of Recurrent Carcinoma.

By S. GILBERT SCOTT.

THE patient, a woman, aged about 40, had carcinoma of the mammæ, and had since developed recurrent subcutaneous nodules all over the body, some of those on the abdomen being the size of a walnut. At the meeting of this Section, recently held at the Middlesex Hospital, Mr. Scott had said that, in these cases, although the nodules disappeared under X-ray treatment, the patient, as a rule, did not live more than two months after the nodules began to extend, for it was found impossible to keep pace with them. But this case illustrated an exception.

History of the Case.—September, 1909: Left breast removed for carcinoma. December, 1911: Lumps in right breast noticed; shortness of breath first noticed. February, 1912: X-ray treatment begun. November, 1912: X-ray treatment stopped; patient very ill; great dyspnœa; nodules rapidly increasing. December, 1912: Many nodules on head, abdomen, neck, and chest. January, 1913: Vomiting; very little nourishment could be taken. February, 1913: Nodules getting smaller; patient better. March, 1913: Some of the nodules disappeared; eating heartily; walked with assistance; no dyspnœa; gaining 1¼ lb. per week. April, 1913: The only nodule remaining was behind the right ear, this getting smaller; lumps in right breast smaller; still gaining weight.

As the nodules did not begin to disappear till four months after the last X-ray treatment he could scarcely arrive at the conclusion that it was a cure by the rays, and it was therefore probably one of the cases of spontaneous cure which were heard of but seldom seen.

DISCUSSION.

Mr. **Deane Butcher** did not think Mr. Scott should decline to attribute the cure to X-rays; he would himself claim it as a cure by X-rays. If ill-effects which ensued months after the use of X-rays were to be attributed to the effects of the rays, it was reasonable to invoke their influence when the effect was good and the period long. Such cases as the present one were continually being reported, especially from abroad.

Dr. **Frank Fowler** said he thought the case illustrated the great importance of prophylactic treatment in every case of cancer of the breast. It was not justifiable to withhold any possible benefit which the rays might confer in preventing local recurrences; in many cases such recurrences could be prevented, the hopeless cases being, of course, those in which recurrence was in bone or liver, or other deep viscus.

Dr. **Harrison Orton** asked whether a microscopical examination was made of a portion of the original tumour which was removed.

Dr. **Feld. Bailey** remarked that the skin of this patient appeared very little affected considering the amount of X-ray treatment given. In similar cases of recurrent nodules which he had treated at intervals during three or four years, the X-rays left the skin in a very different condition from the skin of this patient, there being finally considerable atrophy, pigmentation and dryness, although no definite erythema had ever been produced.

Mr. **Gilbert Scott** replied that the growth was proved to be carcinoma. He gave 1½ Sabouraud, measured, on the tube side of the filter, at the ordinary standard distance, and filtered through ¼ mm. of aluminium, using a high tube. He used that dose when trying to treat both subcutaneous and deeper structures. That thickness of aluminium sufficed to prevent dermatitis. He gave this dose twice a week for six weeks on the scar, after which she had a month's rest. The subcutaneous recurrent nodules could usually be made to disappear with one dose of the rays. The last time he treated her when her case seemed so hopeless, he felt certain, from the difficulty in breathing, that enlarged glands at the root of the lung were pressing on the bronchus and causing the noisy respiration. The prognosis seemed as bad as it could be, yet she recovered.

Case showing Favourable Results of Treatment of Suppurating Tuberculous Glands by X-rays.

By E. P. CUMBERBATCH, M.B.

THE patient was a girl, aged 16. Five years ago the glands in the neck first appeared, and a year later they were much larger, and had commenced to suppurate, and sinuses formed. They were first treated with fomentations, unguents, and other local applications. X-ray treatment was commenced in May, 1911. The mother said that improvement set in at once and no more glands became enlarged, and no fresh sinuses formed. The discharge also lessened, and the swellings became smaller. Dr. Cumberbatch saw her first in October, 1911, and the improvement was then steadily progressing, the neck getting smaller in circumference, and the glands were less palpable, and the sinuses had closed. She received the last application in July, 1912; her weight had increased, and she now looked very healthy. The neck was not swollen, no glands were palpable, the sinuses had closed, and there were left only some scars on the skin marking the site of the former abscesses. The dose which she had averaged $\frac{1}{4}$ pastille (Sabouraud B) per week on each side of the neck. Dr. Cumberbatch felt justified in saying that this great improvement was the result of the irradiation; he noted the diminution in size of the neck dose by dose.

Dr. NORMAN ALDRIDGE thought the good results which followed X-ray treatment of tuberculous glands ought to be better known. During the last five years he had treated a large number of cases by this means, and he did not think there was one which was not greatly improved—he had seen them again after considerable intervals. One such case he had in mind in which only one side of the neck was affected. With treatment the glands went down, all except a small nodule. This patient and another of the same age (16) came some time afterwards with enlarged glands on the other side of the neck, and the side originally treated was found to have remained quite sound. Exophthalmic goitre and tuberculous glands were the two conditions in which X-ray treatment was most favourable.

Cases of Exophthalmic Goitre treated by X-rays.

By W. M. Kingsbury.

Five patients who had been treated by means of X-rays for exophthalmic goitre were shown. The dosage varied from a full pastille to half that quantity. The first was that of a policeman who had the disease severely when first seen—exophthalmos, tremor, and considerable palpitation. His illness started three and a half years ago, and he was laid by nine months, and later had five months' absence from duty. He came to hospital in April, 1912, and was kept in bed three or four months. He had a dozen pastille doses at three-weekly intervals. Last September he went back to duty, and had remained well ever since. His pulse was now 68.

The next patient, a man, had seven pastille doses. He had all the signs, and his pulse was 100; it was now 60. There was no tremor, the thyroid was still somewhat enlarged, but that was the only remains of the disease.

The remaining three patients were females; one of them was now practically well, and the others were much improved. The first of these, when she came to hospital, had considerable difficulty in breathing, marked tremor and palpitation, pulse 132, and exophthalmos. She now had practically no tremor, the thyroid was smaller, and the pulse 68.

The next patient had had nine pastille doses, and had been in hospital twelve weeks. She was now so well that she helped her mother daily. Her pulse had been 168; it was now 120. Her eyes were now not so prominent, and her neck girth had diminished 1 in.

The last patient had had eight pastille doses. She had not had exophthalmos, but was very excitable, and at times became depressed. There was a symmetrically enlarged thyroid, and a rapid pulse. The pulse was now 88, and there was but little tremor remaining.

Treatment of Exophthalmic Goitre by X-rays.

By W. Ironside Bruce, M.D.

Dr. Ironside Bruce gave an analysis of eighteen cases of exophthalmic goitre which had been treated at Charing Cross Hospital since 1905, and he acknowledged his indebtedness to Dr. Lindsay Lock. Six of the eighteen did not appear for re-examination, and one had died, in which case only three exposures had been given when diphtheria supervened. The remaining twelve were examined three months ago, and they had been divided into three categories: (1) cured; (2) greatly improved; (3) improved. Four were cured; the exophthalmos and goitre had gone, and no symptoms remained. Five—of which one had been operated upon—were greatly improved. Three had improved, and one of these also had been operated upon, two still remaining under treatment. The exophthalmos disappeared in four cases, was slight in three, and marked in five, two being still under treatment. The skin was normal in seven cases, there was slight telangiectasis in four, marked telangiectasis in one. These latter were his earliest cases, and the number of exposures given was much greater than had been since found to suffice. All the patients expressed themselves as much better, and they stated that their weight had increased. In none of the patients was there any sign of myxœdema; and that was of interest, since in a case he recorded some years ago, in which he thought myxœdema had developed, the patient got very fat at the latter end of the treatment, which lasted two years, and the hair over her forehead began to fall. When Dr. Lock saw that patient three months ago her appearance was normal, no sign of myxœdema being noted. All his cases were treated with the X-ray tube at a distance of 18 to 12 in. from the skin, the surrounding part being screened off by a 4-in. thickness of felt. They had attended either two or three times a week for periods varying up to two years. The effect of the treatment was wonderfully good. But he had always tried to have in mind the fact that cases of exophthalmic goitre had been known to recover without treatment, so that one must somewhat discount the value of the X-ray treatment on that account.

DISCUSSION.

Dr. HUMPHRIS said that when this subject came up for discussion at the last Electro-Therapeutical Congress in America, much attention was devoted to the question of electrical stimulation of the region of the sixth and seventh cervical nerves in exophthalmic goitre. By such stimulation of these centres he had obtained good results in reduction of symptoms, but not reduction of the goitre. Dr. Arnold Snow said, in her last book, that she brought about a large reduction of pulse-rate by this means, and by abdominal vibration in a large number of cases. He (the speaker) many years ago used an interrupted faradic current, with a plate on each side of the thyroid, and got good results. He believed this disease was peculiarly suitable to electro-therapeutical treatment, even apart from X-rays. In one case, however, there was no reduction of the gland even after two years' treatment. But her palpitation was practically gone, although at one time it was so bad that she was likely to lose her situation, that of lady's maid. And she had had as large a dosage of X-rays as it was thought prudent to give her.

Dr. FRANK FOWLER said the association of myxœdema and Graves's disease was interesting, and he was encouraged by Dr. Bruce's experience that the œdema passed off. He thought the thymus should be included in the irradiation, and he was not afraid to ray the whole of the thyroid.

Dr. G. B. BATTEN said he treated a case of Graves's disease by means of X-rays in 1908. He applied the rays to two-thirds of the thyroid seventy-five times, using a dose of two-thirds of a pastille through four thicknesses of carpet felt. There was no burning; the thyroid gradually became smaller. The rays were applied through a hole in opaque rubber. He left one-third of the gland untreated to prevent the chance of myxœdema supervening. The patient was not absolutely cured of all her symptoms.

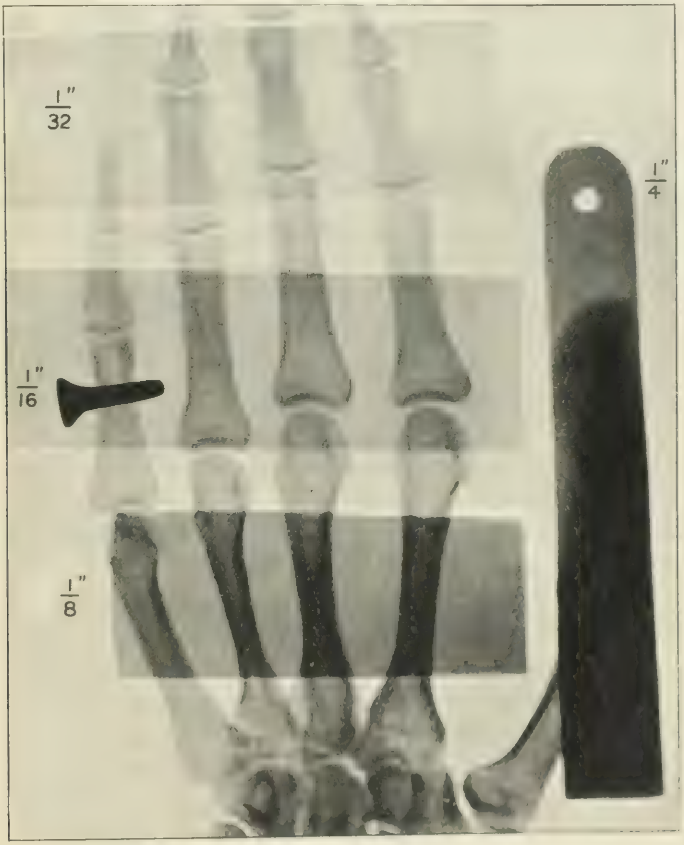
Dr. DONALD BAYNES agreed with Dr. Humphris that very good results could be procured in this disease without using X-rays; he had never used X-rays for Graves's disease, but other modalities, which gave very good results. concussion applied to the seventh cervical spinous process gave excellent results.

The PRESIDENT (Dr. Reginald Morton) said that exophthalmic goitre could be placed alongside tuberculous glands as offering excellent results from irradiation. He had never had any trouble in the matter of overdoing the dosage or frequency, perhaps because he believed in moderate doses, and was very cautious in that regard. That also diminished the likelihood of telangiectasis.

Demonstration of "Duralumin."

By Stanley Melville, M.D.

Dr. Stanley Melville exhibited a number of radiographic prints, and splints designed especially for orthopædic work, made of the new alloy of aluminium and magnesium, termed "Duralumin," invented by Messrs. Vickers and used in the construction of their airship. It permitted the transmission of X-rays, and hence the wearing of a splint made with the metal did not prevent a skiagram being taken of the part. Its strength was equal to that of mild steel, but it was only



Skiagram taken through varying thicknesses of the metal.

one-third the weight. Heating would not temper it, but hammering would. Mr. Muirhead Little, of the Royal National Orthopædic Hospital, made some interesting experiments with the metal from the point of view of surgical appliances. After immersion in strong salt water for three months the metal was as good as ever. Strong acids, and therefore acid perspiration, did not affect the metal, but caustic alkalies quickly produced disintegration. The pelvic band of steel which it had been the custom to use to strengthen the plaster of Paris after reduction of a congenital dislocation proved a great nuisance to the radiographer, but duralumin would obviate that difficulty while in no wise detracting from the strength of the support. Moreover, there was now no need to take a fracture down to see how it was progressing, as a skiagram could be taken with the splint in position. An ordinary valgus plate of the metal showed no shadow at all. The skiagram reproduced (*see* figure) would suggest that an interposed sheet of duralumin, $\frac{1}{8}$ in. or $\frac{1}{16}$ in. thickness, actually intensifies the image.

A New Apparatus.

By Reginald Morton, M.D.

It is very generally admitted that the ordinary induction coil as used in medical treatment and diagnosis is a very unscientific instrument notwithstanding its usefulness. This arises from the fact that we seldom know the exact character of the current given off by any particular coil, and that it is impossible to reproduce any given set of conditions when and where desired. To meet this difficulty I made, as far back as 1903, a revolving commutator with an adjustable brush, as suggested by Leduc, but so modified that the current was reversed as well as interrupted. This practically got rid of any possible accumulation of electrolytic products, and also increased the tetanizing effect. By means of the shifting brush the length of the alternating impulses could be varied from 0 to 0·5, so that at the maximum each impulse was followed by an equal period of rest before reversal took place. Long impulses are painful without having any corresponding advantage. A very useful proportion of period of impulse to period of rest is about 1 : 10. The current from the battery or other source of supply passes through the milliamperemeter before reaching the commutator; this gives us the mean value of the current passing. I have satisfied myself that this current is as useful as that from a coil for most purposes, and with similar instruments any given set of conditions can be reproduced by anyone and at any time. With the introduction of the condenser method of testing, so beautifully demonstrated to us in January by Dr. Lewis Jones, there would seem to be less reason than ever for the retention of the induction coil in scientific work of any kind.

There is another device used in the application of electricity that has become very popular during recent years, and that is the rhythmic interrupter. The use of this instrument has very greatly enhanced the value of electrical treatment, and the more I use it the more I like it. Most of the bodily functions are performed in a rhythmical manner, and it seems but natural that electrical applications should be made in a similar way.

Being somewhat dissatisfied with the progress of a case of arthritis I was treating by ionization, it occurred to me that if I passed the current through the rhythmic interrupter it might do better. While in the laboratory it is true that the number of ions transferred depends on the current density and the time, there was the possibility that a high current density might give rise to a sort of tetanic condition in a living membrane such as the skin, that would not be favourable to the best results. However this may be, the effect on the case in question was quite marked, improvement synchronizing with the change in the method of application. I have tried it on a few other cases with equally encouraging results, and I cannot help feeling that this modification may be of some value in ionization. I believe it to be worth experimenting with, and if some others will join me in this we may be able to arrive at some decision regarding it.

The rhythmical variation should be a very slow one—a complete cycle in not less than about six seconds—and the dipping needle so set that the current is just not cut off. I have not yet decided whether the needle should be bare or insulated to near the point; the tissues will get more rest in the latter case but the ionization may not be so efficient.

These two valuable adjuncts to electrical treatment with constant or variable currents may easily form a permanent part of the apparatus, and I have here a rough model which answers its purpose admirably. The current after passing through the meter is led through terminals to the common points of a double pole two-way switch. This latter enables us to pass the current through the commutator or not, as desired. The wires then lead to terminals on the other side of the board to which the patient is connected, one of the wires taking in the rhythmic interrupter on the way; a short-circuiting switch places the latter device out of action when not wanted. It thus follows that these two instruments may be used in combination, singly or not at all, when the constant current only is required. It is also arranged that though driven by the same shaft the speed of either may be varied independently of the other. For very exact work a speedometer may be attached, so as to indicate the periodicity of the alternating impulses. This commutator gives two complete cycles per revolution and a scale and pointer indicates the length of the impulses relatively to the periods of rest. With a variable resistance in the motor circuit the periodicity may be varied within wide limits; the rhythmic interrupter is driven from the opposite end of the same shaft by means of a friction wheel acting on the face of a copper disk, the speed varying with the point it engages between the centre and periphery of the latter. It is not necessary for me to dilate on the advantages of such a combination as this. I think I have provided for all practical requirements, but I shall highly value any criticisms that may be made.

Dr. BATTEN expressed his interest in the President's exhibit because Dr. Morton invented his rectifier at about the same date as he (Dr. Batten) invented another, and adapted his rectifier for this kind of purpose. It made contacts with silver studs on each side, and the current passed through lamps. It picked out a little of the sine curve, either on one side or on both sides. One could use either the sinusoidal or unidirectional interrupted current, and the voltage could be altered. But at that date it was not proceeded with.

Case for Diagnosis.

By G. B. BATTEN, M.D.

DR. BATTEN showed a skiagram of a case on which he asked for opinions. The patient was a gardener, aged 64. Most of his life had been spent in the open air, but he stoked furnaces for greenhouses. Last July he examined the man, and found the right side quite dull, but there was a small area of resonance in the right apex, close to the middle line. The sputum did not show either tubercle bacilli or pneumococci, though there were some staphylococci. He had had a little jaundice, and his heart was weak. The skiagram showed a clear space high up on the affected side, all the rest of the right lung was very dense. Dr. Batten did not believe it was fluid. The heart was not displaced. The patient was still in the same condition at the present time.

DISCUSSION.

The PRESIDENT believed it was a case of new growth, though not carcinoma, because of the well-defined edge. The triangular area he considered to be normal lung. When he was at the London Hospital there was a case which appeared exactly like this. Treatment by X-rays was suggested, but after one or two applications the patient kept away, for family reasons.

Dr. FRANK FOWLER thought it likely the case might be one of new growth and fluid as well; such a combination was not uncommon.

Dr. BAILEY said he had lately seen a woman, among his out-patients, who bore a large depressed scar over the liver, the result of an operation for hydatids when a young girl. She had never been out of England. There was a history of a favourite dog who played with them in childhood in the nursery, and it was suggested that the dog was the host of a *Tænia ecchinococcus*, and that the nursery floor became infected with ova.

Dr. IRONSIDE BRUCE did not think malignant disease of the lung would show an even distribution such as this skiagram revealed. It must be, in his view, either a collection of fluid, or an endothelioma growing from the pleura, which had invaginated itself into the pleural cavity.

Mr. GILBERT SCOTT said he remembered the case of a woman who came to hospital with complete opacity on one side, except in a triangular patch such as seen in the present skiagram. It was homogeneous and dense. She was in hospital several weeks, and then a large aneurysm was revealed. It had pressed on the root of the lung, there was no air entry, and she also had pleural effusion. She was between 40 and 50 years of age, and before she left the hospital the lung trouble had disappeared. In the early stage the whole condition had been masked by the fluid. The upper border was formed by the aneurysm, which gave the impression that it was a solid growth.

Case of Arborescent Burning by Lightning.

By C. WARD, F.R.C.S.I.[1]

ABOUT Christmas two years ago, the patient, "Gaudud," a Zulu lad aged about 18, was leading the first two of a team of ten mules drawing a farm wagon near Vryheid (he was a voerlooper, a leader of mules or oxen). A dry storm was threatening; there was no water nor trees near. The wagon contained a load of firewood, no iron rails or metal He was wearing a felt hat with a metal buckle in front, a khaki tunic with black metal buttons, a belt with a metal buckle, black cloth trousers, no boots; nothing in his purse; nothing in his pockets. Suddenly the first flash of the storm came; there was no rain. He did not see the flash nor hear the thunder. The upper part of his trousers was burnt, and they dropped down, but his legs escaped injury. The two mules he was leading were killed, and three others behind them. The driver on the box-seat escaped, and took the patient on to Vryheid Hospital, where he remained three months. He recovered with a few superficial scars on the left arm, and the condition shown very well in the photograph.

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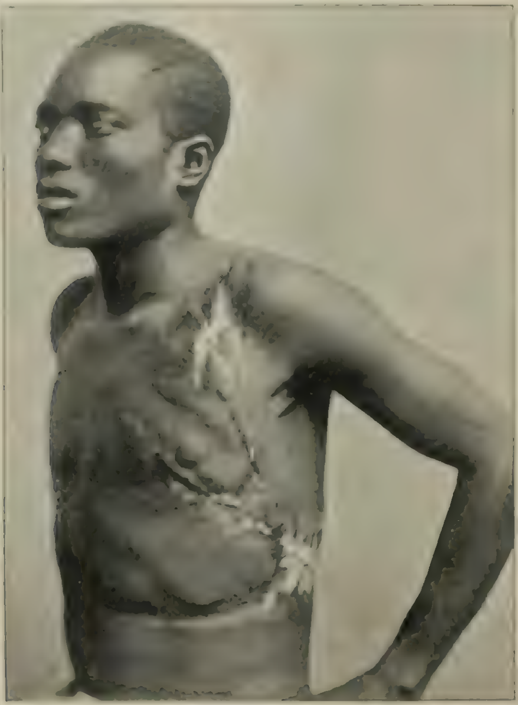
[1] District Surgeon, Pietermaritzburg, Natal.



Case of arborescent burning by lightning.

Dr. Ironside Bruce showed a series of skiagrams of chest conditions.

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